UNIVERSITY OF KHARTOUM
POSTGRADUATE MEDICAL STUDIES BOARD
FACULTY OF MEDICINE

CLINICAL PATTERN OF INTRACRANIAL SPACE OCCUPYING LESIONS IN ADULT SUDANESE PATIENTS

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A thesis submitted in partial fulfillment of the requirements for the Clinical MD Degree in Clinical Medicine of the University of Khartoum

1999
PLEASE BE AWARE THAT
ALL OF THE MISSING PAGES IN THIS DOCUMENT
WERE ORIGINALY BLANK
DEDICATION

To the soul of my Mother
ACKNOWLEDGEMENT

It is with great pleasure, that I record my gratitude to my supervisor Dr. Mohammed Najeeb Abdulla. I wish to thank him for his meticulous supervision, encouragement and patience. My debt to him is so great. Also I wish to thank Dr. M.A. Arbab and Dr. Abu Salih for their advises, help and support.

To all those and to many others not individually mentioned here, my gratitude goes out.
<table>
<thead>
<tr>
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<tr>
<td>AD</td>
<td>Autosomal dominant</td>
</tr>
<tr>
<td>AIDS</td>
<td>Acquired immunodeficiency syndrome</td>
</tr>
<tr>
<td>AR</td>
<td>Autosomal recessive</td>
</tr>
<tr>
<td>BCNU</td>
<td>Bischloroethylnitrosurea</td>
</tr>
<tr>
<td>CNS</td>
<td>Central Nervous System</td>
</tr>
<tr>
<td>CCNU</td>
<td>Cyclohexylchloroethylnitrosourea</td>
</tr>
<tr>
<td>CSF</td>
<td>Cerebrospinal fluid</td>
</tr>
<tr>
<td>CPA</td>
<td>Cerebellopontine angle</td>
</tr>
<tr>
<td>CT</td>
<td>Computed tomography</td>
</tr>
<tr>
<td>DIG</td>
<td>Desmoplastic infantile ganglioma</td>
</tr>
<tr>
<td>ESR</td>
<td>Erythrocyte sedimentation rate</td>
</tr>
<tr>
<td>FSH</td>
<td>Follicle stimulating hormone</td>
</tr>
<tr>
<td>HCMV</td>
<td>Human sytomegalovirus</td>
</tr>
<tr>
<td>HSV</td>
<td>Herpes simplex virus</td>
</tr>
<tr>
<td>ICP</td>
<td>Intracranial pressure</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imagine</td>
</tr>
<tr>
<td>NF₂</td>
<td>Neurofibromatosis (type two)</td>
</tr>
<tr>
<td>LH</td>
<td>Luteinising hormone</td>
</tr>
<tr>
<td>PCA</td>
<td>Posterior communicating artery</td>
</tr>
<tr>
<td>PXA</td>
<td>Pleomorphic xanthoastrocytoma</td>
</tr>
<tr>
<td>SOL</td>
<td>Space occupying lesion</td>
</tr>
<tr>
<td>TWBC</td>
<td>Total white blood cell count</td>
</tr>
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</table>
ABSTRACT

This study was done in the period between August, 1997 and October, 1998, with the aim to determine the types of SOLs in adult Sudanese patients, also clinical presentation and CT Scan changes were studied. The number of patients included in this study was 118, all were seen in Khartoum city hospitals, Khartoum Teaching Hospital, El Shaab Teaching Hospital and Ibn Khaldoon Hospital.

Intracranial SOLs were found to be more common in males (56.8%), and were more common in the age group > 30-40 years (32.2%). The commonest intracranial SOL was meningioma (28.8%), followed by glioma in (26.3%), then abscess in (10.2%), followed by tuberculoma in (8.5%), pituitary adenoma in (6.8%) and other SOLs were found to account for (19.5%).
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CHAPTER ONE
INTRODUCTION

The increased availability of modern imaging techniques (CT and MRI) has improved the diagnosis of intracranial space occupying lesions. Earlier diagnosis, in regard to brain tumours, has posed new problems and ethical dilemmas about treatment, which may not improve the outcome. But which may now to be considered in asymptomatic patients. Nevertheless, improved imaging has led to new therapeutic strategies, which may well result in better quality and length of survival for many patients.

It may be of some importance to mention why this study is conducted? Although clinical pattern of neurological disease was studied by Daoud et al who found that intracranial SOLs account for 4.95% of all neurological cases\(^1\) and brain tumours were also studied in Sudan many years ago by Abu Salih and Abdul-Rahman\(^2\) it is fair enough to mention that no similar study about intracranial SOLs has been done in Sudan.

A series of 3010 cases of SOLs was described in literature\(^3\). These were as follows: - Glioma 41.5%, Pituitary adenoma 3.4%, Meningioma, 11.6%, Acoustic neuroma 2.5%, Congenital tumours 3.5%, Metastases23.7%, Granulomatous tumour 2.5%, Blood vessels tumour 2.5%, Sarcoma 0.3%, Parasitic cyst 0.7%, Miscellaneous 2.2%

Irfan et al mentioned that SOLs were more common in males, in the age group 11-20. Gliomas comprised 32% of the total cases followed by meningiomas 13.7%, abscess 13.2% and pituitary tumours 13.2% and tuberculoma constituted 5.5%\(^4\). A rare type of space occupying lesion is cranial mycetoma. This type of SOL was recently studied in Sudan by Arbab et al\(^5\).
**Definition:**

The term intracranial SOL is generally used to identify any lesion, whether vascular or neoplastic or inflammatory in origin, which increases the volume of the intracranial contents and thus lead to a rise in the intracranial pressure. In the strict sense the term 'intracranial tumour' should be reserved for neoplasms, whether benign or malignant, primary or secondary, but conventionally this inclusive term is often used to embrace lesions such as vascular malformations and granulomas of inflammatory origin 'tuberculoma' as well as parasitic cysts, which are not neoplastic in the strict pathological sense\(^{(3)}\).
LITERATURE REVIEW

BRAIN TUMOURS:
Incidence and Distribution:

Most primary brain tumours in adults occur above tentorium cerebelli and must be considered generally to be malignant\(^6\). The intracranial tumours of childhood differs considerably from the adult forms in term of distribution within the brain, histological characteristics and prognosis\(^6\). The annual incidence in U.K. is ranging between 4-6 per 100,000\(^6,7,8\). Intracranial tumour is the sixth most common cause of neoplasms in adults, of which 70% occur above the tentorium\(^6\). Autopsy findings suggest that intracranial tumours comprise approximately 8% of all primary neoplasms\(^6\).

In adults they account for about 2% of primary malignant tumours\(^7\). Cerebral gliomas rarely occur in the first two decades of life, but the incidence then steadily increases\(^6\). They are twice as common in males as in females\(^6\). Meningiomas and schwannomas occur mainly in women and are rare in childhood\(^6\). According to another source primary brain tumours arising within brain meninges or parenchyma of the brain are common at all ages of the life\(^3\). There appears to be an increased incidence of craniopharyngioma and pineal germinoma in Japan, ependymoma in India and medulloblastoma in Europe and North America\(^6\). Metastatic tumours account for about 25% of all intracranial tumours found at autopsy\(^6\).

Approximately 17,000 of primary brain tumours are treated each year in USA. The incidence in 1986-1990 for males is 7.3 per 100,000 year and for females is 5.3 per 100,000 per year\(^10\). According to some estimates it causes the death of 90,000 patients in USA each year\(^10\). Some authors claimed that females showed an increase of 68% and males
an increase of 36% in incidence rates of brain tumours in USA in the period between 1985-1986\(^{(11)}\), while others claimed that there is no any significant increase in the incidence rates of brain tumours in the period between 1980-1990, except for lymphoma in men\(^{(12)}\). The incidence of primary brain tumours in elderly Floridians has increased. This increase is confirmed to be histologically specific and is independent of increase case ascertainment associated with introduction of CT scan and independent of general increase of all cancers\(^{(13)}\). Further investigation of this increase is warranted\(^{(13)}\).

Of all primary brain tumours 60% are gliomas, 20% are meningiomas and other tumours comprise 20%\(^{(7)}\). Metastatic tumours are as frequent as gliomas, so that they comprise 25-30% of all intracranial tumours\(^{(7)}\). Other authors mentioned that gliomas may comprise 48.8% of all primary intracranial tumours, meningiomas about 24%, pituitary adenoma 6.8%, sarcomas 6.2%, haemangiomas 6.5%, haemangio-blastomas 3%, craniopharyngiomas 2.5% and neuroma 2.4%\(^{(14)}\). In the study conducted by Abu Salih and Abdul Rahman the incidence of brain tumours in the Sudan in the period between 1971-1981 was 7% of all neurosurgically admitted patients. They concluded that their series showed a high incidence of meningioma and very low incidence of acoustic neuroma compared to regional and international series\(^{(2)}\).

Incidence of brain tumours in the Sudan as reported by Abu Salih and Abdul Rahman was as follows\(^{(2)}\):
Meningiomas 45.5%
Gliomas 39.8%
Pituitary adenomas 10.6%
Craniopharyngiomas 5.7%
Acoustic neuromas 0.8%
Metastatic tumours 6.5%

Brain tumours were found to be more common in males (63.4%) and most commonly affect those in the third decade, followed by those in the fourth decade\(^{(2)}\).

**Etiology:**

The activation of oncogenes and inactivation of tumour suppressor genes within neoplastic cells lead to transformation and loss of growth control. The cause of intracranial tumours remains unknown but in some predisposing factors are recognized\(^{(8)}\). Heritable syndromes and ionising radiation are the only established two causes of primary CNS tumours\(^{(13)}\). These heritable syndromes include:-

- Von Recklinghausen’s disease (neurofibromatosis) which is associated with optic and hypothalamic glioma, intracranial meningiomas and schwannomas of various cranial nerves, most commonly the eights\(^{(6)}\).
- Tuberose sclerosis which is associated with periventricular glioma\(^{(6)}\).
- Von Hippel Lindau disease which is associated with cerebellar haemangioblastomas and similar lesions in the retina and cystic lesions in the pancreas and kidneys\(^{(8)}\).

Apart from these syndromes findings are inconclusive for other suggested risk factors including head trauma, prior infection and pesticides\(^{(13)}\). Early beliefs that head injury or glial scarring increased the risk of glioma have not been substantiated. But meningiomas have
occasionally been reported as occurring directly below the site of the skull injury(6). In regard to pesticides, Bohner-NI et al mentioned that existing data are insufficient to conclude that exposure to pesticides is a clear risk factor for brain tumours. It seems more plausible that exposure to multiple agents and/or other factors such as genetic predisposition are most relevant with respect to brain tumour pathogenesis(16).

Other investigators from USA found statistically significant excess of brain tumours among grain farmers, rubber workers and workers in transportation equipment manufacture and repair(17). Risk factors among textile spinners and winders were of border line significance. Elevated but not significant risk of 2.0 or greater was seen among nurses which is considered as a new finding (17). Attempts to find a link between brain tumours and occupational exposure to magnetic field were proved unconvincing (18).

It has been estimated that the risk of developing an intracranial tumour of any type is marginally greater in close relatives of patients with known cerebral gliomas(6). Also there have been occasional reports of families having a high incidence of various intracranial tumours but no clear cut pattern of inheritance of the development of an isolated intracranial neoplasms has been demonstrated (6). Small clusters of tumours (malignant astrocytoma) have appeared in certain occupational settings notably in petroleum processing industry (9).

**Classification:**

The classification of brain tumours is a rather confusing subject. Using a simple approach, brain tumours can be classified into metastatic, primary extraaxial and primary intraaxial. This classification include all of the primary brain tumours listed in the World Health
Organization classification, adds pituitary and metastatic tumours and is obviously simple, more over it follows practical clinical thinking\textsuperscript{(10)}. Common brain tumours in adults with percentage incidence by category\textsuperscript{(10)}.

<table>
<thead>
<tr>
<th>Metastatic</th>
<th>Primary extraaxial</th>
<th>Primary intraaxial</th>
</tr>
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<tbody>
<tr>
<td>Lung 37%</td>
<td>Meningioma 80%</td>
<td>Glioblastoma 47%</td>
</tr>
<tr>
<td>Breast 19%</td>
<td>Acoustic neuroma 10%</td>
<td>Anaplastic astrocytoma 14%</td>
</tr>
<tr>
<td>Melanoma 16%</td>
<td>Pituitary adenoma 7%</td>
<td>Astrocytoma 15%</td>
</tr>
<tr>
<td>Colorectal 9%</td>
<td>Others 3%</td>
<td>Oligodendroglioma 5%</td>
</tr>
<tr>
<td>Kidney 9%</td>
<td></td>
<td>Lymphoma 2%</td>
</tr>
<tr>
<td>Others 11%</td>
<td></td>
<td>Others 7%</td>
</tr>
</tbody>
</table>

Another important classification and WHO classification of brain tumours. It is one of several formal schemes that are based on neuropathologic criteria, metastases are not considered and one can get no sense of a given tumour as a clinical problem as suggested by the previous classification\textsuperscript{(10)}.

A) Astrocytic tumours--:

1. Astrocytoma
   a. Fibrillary
   b. Protoplasmic
   c. Gemistocytic

2. Pilocytic astrocytoma

3. Subependymal giant cell astrocytoma (ventricular tumour or tuberous sclerosis).

4. Astroblastoma.

5. Anaplastic malignant astrocytoma.

B) Oligodendroglial tumours--:

1. Oligodendroglioma.
3. Anaplastic oligodendroglioma.

C) Ependymal and choroid plexus tumours:-
1. Ependymoma
   Variants
   a. Myxopapillary ependymoma.
   b. Papillary ependymoma.
   c. Subependymoma.
2. Anaplastic ependymoma.
3. Choroid plexus papilloma.
4. Anaplastic choroid plexus papilloma.

D) Pineal cell tumours:-
1. Pineocytoma (pineal cytoma).
2. Pineoblastoma (pineal blastoma).

E) Neuronal tumours:-
1. Gangliocytoma.
2. Ganglioglioma.
4. Anablastic (malignant) gangliocytoma and ganglioglioma.
5. Neuroblastoma.

F) Poorly differentiated and embryonal tumours:-
1. Glioblastoma
   Variants
   a. Ganglioblastoma with sarcomatous component (mixed glioblastoma and sarcoma)
   b. Giant cell glioblastoma.
2. Medulloblastoma
   Variants
a. Desmoplastic medulloblastoma.

b. Medullomyoblastoma.

3. Medulloepithelioma.

4. Primitive polar spongioblastoma.

5. Gliomatosis cerebri.

Primary intracerebral tumours are also classified by their degree of malignancy. They vary in incidence by age and localization. Malignant intracranial tumours include, glioma, oligodendroglioma, medulloblastoma, ependymoma and microglioma. Benign tumours include, meningioma, neurofibroma, craniopharyngioma, pituitary adenoma and colloid cyst\(^\text{19}\).

It is important to mention that the concept of malignancy in the central nervous system has a different meaning from that which applies to systemic cancers. The term ‘malignant’ has nothing to do with metastasis out of the CNS which is extraordinary rare. Unless a tumour can be completely excised to the last cell, all intracranial neoplasms are potentially malignant in that they may recur, and often do\(^\text{10}\). The table below details the incidence of intracranial tumours examined by the Neuropathology Department, Institute of Neurological Sciences Glasgow (population 2.7 million) over a 5 year period\(^\text{8}\).
<table>
<thead>
<tr>
<th>Supratentorial</th>
<th>Adults</th>
<th>Children (&lt;15 yrs)</th>
</tr>
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<tbody>
<tr>
<td>Anaplastic astrocytoma (including ganglioblastoma multiforme)</td>
<td>347</td>
<td>5</td>
</tr>
<tr>
<td>Meningioma</td>
<td>137</td>
<td>-</td>
</tr>
<tr>
<td>Metastasis</td>
<td>105</td>
<td>-</td>
</tr>
<tr>
<td>Astrocytoma</td>
<td>73</td>
<td>5</td>
</tr>
<tr>
<td>Pituitary adenoma</td>
<td>31</td>
<td>-</td>
</tr>
<tr>
<td>Craniopharyngioma</td>
<td>13</td>
<td>9</td>
</tr>
<tr>
<td>Oligodendroglioma</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>Colloid cyst</td>
<td>4</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>2</td>
<td>&lt;1%</td>
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<tr>
<td>Others</td>
<td>11</td>
<td>6</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Infratentorial</th>
<th>Adults</th>
<th>Children (&lt;15 yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurilemmoma</td>
<td>50</td>
<td>-</td>
</tr>
<tr>
<td>Metastasis</td>
<td>39</td>
<td>-</td>
</tr>
<tr>
<td>Haemangioblastoma</td>
<td>17</td>
<td>-</td>
</tr>
<tr>
<td>Astrocytoma</td>
<td>12</td>
<td>19</td>
</tr>
<tr>
<td>Meningioma</td>
<td>12</td>
<td>27%</td>
</tr>
<tr>
<td>Medulloblastoma</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>Dermoid, epidermoid</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Ependymoma</td>
<td>4</td>
<td>6%</td>
</tr>
<tr>
<td>Others</td>
<td>8</td>
<td>4%</td>
</tr>
</tbody>
</table>

**Classification by location of the tumours:-**

1) Cerebral hemisphere
   
   a. Extrinsic: Meningioma
      - Cysts (dermoid, epidermoid, arachnoid)
   
   b. Intrinsic:
      - Astrocytoma.
      - Glioblastoma.
      - Oligodendroglioma.
      - Ganglioma.
2) Hypothalamus
   Astrocytoma.

3) Sellar/Suprasellar region
   - Pituitary adenoma.
   - Craniopharyngioma.
   - Meningioma.
   - Optic nerve glioma.
   - Epidermoid cyst and dermoid cyst.

4) Ventricular system
   - Colloid cyst.
   - Choroid plexus papilloma.
   - Ependymoma.
   - Germinoma.
   - Teratoma.
   - Meningioma.

5) Pineal region
   - Pineal cytoma/blastoma.
   - Astrocytoma.
   - Meningioma.
   - Germinoma.
   - Teratoma.
   - Ependymoma.

6) Posterior Fossa
   a. Extrinsic
      - Neurilemoma (VIII, V).
      - Meningioma.
      - Epidermoid, dermoid cyst.
- Arachnoid cyst.
- Metastasis.

b. Intrinsic
- Metastasis.
- Haemangioblastoma.
- Medulloblastoma.
- Astrocytoma.

7) Skull base and sinuses
- Carcinoma (nasopharyngeal, sinuses, carcinomatous meningitis).
- Chordoma.
- Glomus jugulare tumour.
- Osteoma (Mucocele).

Pathological classification:-

Neuroepithelial:-

• Tumours derived from Astrocytes (Astrocytoma):-

Is the most common primary brain tumour. Histological features permit separation into 4 grades depending on the degree of malignancy. Grading is of limited accuracy and only reflects the features of the biopsy specimen and not necessarily, those of the whole tumour. The most malignant type, anaplastic astrocytoma (grade iv) occurs most frequently and widely infiltrates surrounding tissue. The less common low grade astrocytomas include the pilocytic (juvenile) type, fibrillary protoplasmic and gemistocytic types.

• Tumour derived from Oligodendrocytes (Oligodendroglioma):-

Usually a slowly growing, sharply defined tumour. Variants include an anaplastic (malignant) form and mixed astrocytoma oligodendroglioma.
- Tumours derived from Ependymal cells and choroid plexus.

- Ependymoma:
  Occurs anywhere throughout the ventricular system or spinal canal, but particularly common in the fourth ventricle and cauda equina. It infiltrates surrounding tissue and may spread throughout the CSF pathways. Variants include an anaplastic type and subependymoma arising from subependymal astrocytes.

- Choroid plexus papilloma:
  Rare tumours and an uncommon cause of hydrocephalus due to excessive CSF production. They are usually benign but occasionally occur in a malignant form.

- Ganglioma/gangliocytoma/neuro-blastoma:
  Rare tumours containing ganglion cells and abnormal neurons. Occur in varying degrees of malignancy.

- Pineocytoma/Pineoblastoma:
  Extremely rare tumours. The latter are less well differentiated and show more malignant features.

- Poorly differentiated→Glioblastoma multiforme:
  A highly malignant tumour with no cell differentiation preventing identification of its tissue origin

- Embryonic tumours (Medulloblastoma):
  A malignant tumour of childhood arising from cerebellar vermis. Small closely packed cells are often arranged in rosettes surrounding abortive axons. Many seed through CSF pathways.
Tumours of the Meninges:

Meningioma arise from the arachnoid granulation, usually closely related to the venous sinuses but also found over the hemispheric convexity. The tumours compress rather than invade adjacent brain. They also occur in the spinal canal and orbit. Most are benign (despite their tendency to invade adjacent bone) but some undergo sarcomatous change. Histological types include: syncytial, transitional, fibroblastic and angioblastic.

- Meningial sarcoma and primary meningeal melanoma:

Exceedingly rare tumours.

Tumours derived from the Nerve Sheath Cell:

- Neurolemmoma/Schwannoma:

A non-invasive slowly growing tumour of the schwann cells, involving the eighth cranial nerve roots or the peripheral nerves. Different histological types exist: Antoni type A

Antoni type B

Antoni type A- shoals and whorls of tightly packed cells in groups or palisades.

Antoni type B- a mesh work of interlinked loosely packed stellate cells.

- Neurofibroma:

A tumour of schwann cells and fibroblasts producing a fusiform expansion through which nerve fibres run. It involves the spinal nerve roots or peripheral nerves but rarely affects cranial nerves and has greater tendency to undergo malignant change than schwannoma. This tumour is the type associated with Von Recklinghausen’s disease, although schwannomas and mixed tumours also occur.
Tumours derived from Blood vessels (Haemangioblastoma):

Occurs within the cerebellar parenchyma or spinal cord. In 1926 Lindau described a syndrome relating cerebellar and or spinal haeminangioblastomas with similar tumour seen in the retina and cystic lesions in the pancreas and kidneys (Von Hipple-Lindau disease).

Tumours derived from Germ Cells:

Germinoma: Primitive spheroidal cell tumour comparable to seminoma of the testis.

Teratomata: A tumour containing a mixture of well differentiated tissues, dermis, muscles and bone. These are uncommon tumours at the pineal region and they are not arising from pineal cells.

Tumours of maldevelopmental origin:

Craniopharyngioma: Arises from cell rests of the buccal epithelium and lies in close relation to pituitary stalk. Usually a nodular tumour with cystic areas containing greenish fluid and cholesteatomous material.

Epidermoid/dermoid cyst: Rare cystic tumours arising from cell rests predetermined to form epidermis or dermis.

Colloid cyst: A cystic tumour arising from an embryological remnant in the root of the third ventricle.

Anterior pituitary gland:

Pituitary adenoma: A benign tumour usually secreting excessive quantities of prolactin, growth hormone.
Adenocarcinoma: A malignant tumour occasionally arises in the pituitary.

Local extension from adjacent tumours:-

- Chordoma: A rare tumour arising from cell rests of the notochord. May occur anywhere from the sphenoid to the coccyx, but commonest in the basi-occipital and the sacrococcygeal region, invading and destroying bone at these sites.

- Glomus Jugulare Tumour (syn-chemodectoma): Vascular tumour arising from ‘glomus jugulare’ tissue lying either in the bulb of the internal jugular vein or in the mucosa of the middle ear. The tumour invades the petrous bone and may extend into the posterior fossa or the neck.

- Other local tumours include chondroma, chondrosarcoma and cylindroma.

Primary malignant lymphoma (syn:microgliomatosis):

Forms around parenchymal blood vessels. May be solitary or multifocal. It generally occurs in immunocompromised patients i.e. AIDS. Metastatic lymphoma (non-Hodgkin’s) is less common, involves the meninges and is rarely intraparenchymal[8].

Metastatic tumours:-

May arise from any primary site but most commonly spread from the bronchus or breast[16]. In 1993 a revised WHO classification of brain tumours was published by international committee, as novel tumour entities the desmoplastic infantile ganglioma (DIG) and pleomorphic xanthoastrocytoma (PXA) have been included[20]. Several histopathological variants of meningiomas have been added of which only the
papillary meningioma and the atypical meningioma are characterized by increase rate of recurrence. Meningeal haemangiopericytomas and hemangioblastoma are classified as tumours of nonmeningiothelial origin. The glioblastoma multiforme, which had previously been listed as an embryonal tumour, is now recognized as astrocytic glioma. Immunohistochemistry has greatly advanced the practical diagnosis and classification of brain tumours. There are specific markers for all normal and neoplastic cells except for oligodendrogliaoma. The prognosis and therapeutic approaches to brain tumours greatly depends on histopathological grading. The WHO proposes four tumour grades I, II, III, IV. As a rule grade I and II tumours are viewed as benign neoplasms, grade III and IV tumours as malignant. There are attempts to use new biological parameters for grading of the brain tumours. Antibodies to proliferation associated proteins reflect tumour growth. Molecular genetic approaches to tumour associated genes and gene loci are particularly promising new tools for the future.

**Clinical Features:**

Brain tumours present in two patterns, not necessarily mutually exclusive, one consists of non focal symptoms of increased intracranial pressure i.e. such as headache, nausea and vomiting, confusion and lethargy. The other consists of symptoms or signs of focal brain dysfunction, these may be summarized as follows:

1. Frontal lobe:
   - Generalized seizures and focal motor seizures.
   - Expressive aphasia (dominant side).
   - Behavioral changes.
   - Dementia.
• Gait disorder; incontinence.

2. Basal Ganglia:-
• Hemiparesis (contralateral).
• Movement disorders (rare).

3. Parietal lobe:-
• Receptive aphasia (dominant side).
• Visual field defect. This include:-
  Lower homonymous quadrantanopia.
• Cortical sensory dysfunction ‘contralateral’:-
  Localization of touch.
  Two point discrimination.
  Astereognosis.
  Sensory inattention

4. Occipital Lobe:-
• Homonymous hemianopia (contralateral).
• Visual disturbance.

5. Temporal Lobe:-
• Complex partial (psychomotor) seizures.
• Generalized seizures.
• Behavioral changes.
• Olfactory and complex visual auras.
• Upper homonymous quadrantanopia.

6. Corpus Callosum:-
• Dementia (anterior).
• Behavioral changes (posterior).
• Asymptomatic (mild).

7. Thalamus:-
• Sensory loss (contralateral).
• Behavioral changes.
• Language disorder (dominant side).

8. Mid brain/pineal:-
• Paresis of vertical eye movements.
• Pupillary abnormalities.
• Precocious puberty (boys).

9. Sella/optic nerve/pituitary:-
• Endocrinopathy.
• Bitemporal hemianopia.
• Monocular visual defects.

10. Pons/Medulla:-
• Cranial nerve dysfunction.
• Ataxic nystagmus.
• Weakness/sensory loss.
• Spasticity.

11. Cerebellopontine angle: -
• Deafness (Ipsilateral).
• Loss of facial sensation ‘Ipsilateral’ and 7th cranial nerve
• Ataxia

12. Cerebellum: -
• Ataxia (Ipsilateral).
• Nystagmus

Such signs of focal brain dysfunction may have convincing localizing value even before an image of the brain is made by computed tomography or MRI. The onset is usually gradual and the course is
progressive, but sometimes it may present acutely like CVA because of bleeding into the tumour\textsuperscript{(19,10)}.

**Symptoms and signs of increased intracranial pressure:**

These may be caused by cerebral mass, reactive cerebral oedema, or obstruction of cerebrospinal fluid pathway. The major features are:

- **Headache:**
  
  This is common but not invariable manifestation of brain tumour\textsuperscript{(19)}, and pain is due to stretch of the meninges by the tumour. Typically the headache is most severe in the morning and may disturb sleep, although as intracranial pressure rises the pain becomes more constant. The localization of the headache does not generally correlate with the site of tumour, although posterior fossa tumours often cause pain in the occipital or nuchal area\textsuperscript{(19)}. Headache in supratentorial tumours was claimed to have correlation with site of tumour\textsuperscript{(19,21)}. Headache is increased by conditions that cause increased intracranial pressure, such as coughing, bending, straining, it was reported to occur in 88\% in some series\textsuperscript{(3)} and in 60\%\textsuperscript{(19)} and 71\%\textsuperscript{(21)} in other series.

- **Impairment of conscious level:**
  
  This ranges from listlessness and drowsiness to coma and is related to the level of ICP. Cerebral tumours occupy space within the rigid skull, but compensatory mechanisms involving alteration in the volume of fluid in cerebrospinal fluid spaces and venous sinuses may delay the onset of raised ICP. Benign tumours may therefore attain a large size before causing a rise in ICP. Raised ICP develops early in rapidly expanding tumours or even acutely if the cerebrospinal circulation is obstructed by, for example, posterior fossa masses or interventricular tumours. Raised ICP may also cause personality change, including apathy irritability, withdrawal and inattention\textsuperscript{(19)}. 
• Papilloedema:-
This is significant but not invariable sign of ICP and may develop acutely or insidiously. Swelling of optic nerve head may be accompanied by haemorrhages in the optic disc, but often causes little subjective visual disturbance. Perimetry may reveal peripheral constriction of visual field or enlargement of blind spot, but visual acuity is usually preserved. Rarely acute blurring of vision or transient blindness, precipitated by postural changes (visual obscuration) may be an early feature and indicate severely raised ICP. Papilloedema progresses in parallel to the level of ICP and eventually results in visual failure due to extensive retinal haemorrhage, or secondary optic atrophy. Rarely but significantly, papilloedema may not develop in raised ICP, particularly if this of acute onset as may occur in obstruction of cerebrospinal fluid pathway (19). It was reported to occur in 75%.

• Vomiting, bradycardia, arterial hypertension:-
These develop as ICP continues to rise. These features usually parallel the other clinical signs, but sudden vomiting may be an early feature of tumours of cerebellar hemisphere (19), and it may occur in up to 65% of patients (3).

• False localizing signs:-
These are signs which are present regardless the site of tumour, and are due to raised ICP. The rise in ICP may not be uniform within the cerebral substance and sudden alterations in pressure relationships within the skull may lead to displacement of parts of the brain. Downward displacement of the temporal lobes due to a large hemisphere mass may result in stretching of the third and sixth cranial nerves, or pressure on the contralateral cerebral peduncle may result in ipsilateral upper motor neuron signs. Another form of displacement is the downward movement of cerebellar tonsils, so that they impact within the foramen magnum, thus compressing the medulla. This coning
may result in brain stem haemorrhage or acute obstruction of the cerebrospinal fluid pathway, and is often associated with loss of consciousness and paresis of the six and third cranial nerves with dilatation of the pupil on the side of the lesion. The patients may adopt a decerebrate posture and death almost invariably ensues. This type of brain displacement may occur spontaneously in relation to critical levels of intracranial pressure, but is particularly likely to develop if the pressure dynamics are disturbed by lumbar puncture\(^{(19)}\).

- **Epilepsy:**

  About 20\% of tumour patients develop fits sometime in their lives\(^{(10)}\). Infiltration by tumour cells of an area of cerebral cortex often invokes excitatory responses in neighbouring neurons and may result in an epileptic focus. The resulting seizures may be generalized or focal in nature. The development of focal motor or sensory seizures in adult life should always suggest the possibility of a tumour. It was reported in 40\% of patients with glial tumours\(^{(22)}\) and in 25\% with meningioma\(^{(8)}\).

  In addition to all these symptoms the patient with brain tumour may present with other symptoms, for example a patient with Sturge Weber Syndrome or neurofibromatosis may have the characteristic skin lesions in addition to brain tumour. Turcot syndrome which is inherited as AR is characterized by adenomatous polyposis of the colon and CNS tumours. Patients with this syndrome may present with symptoms related to adenomatous polyps. Nagane et al reported two rare cases of triple primary malignant neoplasm (PMN), including malignant brain tumours ‘glioblastoma’ with adenocarcinoma of the stomach and carcinoma of the bladder in one case and adenocarcinoma of the lung and adenocarcinoma of the rectum in the other. They mentioned that 44 such cases have already been reported\(^{(23)}\). Another
investigator Ahsan et al. (24) conducted an exploratory study of brain tumours that occurred as a second primary malignancy to identify potential risk factors for brain tumours and they concluded that the association of brain tumours with bladder, colorectal and endometrial cancers in women and an increase occurrence of CNS lymphoma as a second malignancy in men are new findings that have not been described previously (22). Although association of syringomyelia with brain tumour is relatively rare, some Japanese investigators (23) have reported a case of syringomyelia with left posterior fossa meningiothelial meningioma. Surprisingly enough, postoperative CT and MRI demonstrated disappearance of syringomyelia (25). Shinoda et al. reported a case of painful tic (trigeminal neuralgia and ipsilateral hemifacial spasm) caused by cerebellopontine angle epidermoid tumour (26). After removal of the tumour neuralgia and facial spasm disappeared. Painful tic caused by brain tumour is rare (eight cases in literature plus this case) but epidermoid tumour is not rare as a cause of this complaint (seven in eight cases). They stated that on encountering a case of painful tic of unknown origin, it is prudent to consider the same kind of tumour as a cause.

It has been found that patients with brain tumours are at great risk of developing deep vein thrombosis and pulmonary problems. Thromboembolism is a quite common problem in patients with brain tumours. For this reason prophylactic treatment with mechanical devices and pharmacological agents has been suggested (27). Primary lymphoma of the central nervous system occurs in about 10% of patients with acquired immunodeficiency syndrome and is the commonest brain tumour in this subpopulation (28).
Brain tumours, particularly neuroectodermal tumours and stomach cancer, in families of patients with Ewing's sarcoma were found to have an increased incidence and hypothesis suggesting that these tumours might share common aetiology, was made up(29).

Brain tumours may be a part of Gorlin's syndrome, which is also known as multiple basal cell syndrome, a familial tumour condition with autosomal dominant inheritance(30). Patients develop multiple basal cell carcinomas beginning in childhood(30). They also have typical dysmorphic faces, skeletal malformations, and particular type of epithelial cyst of the jaws. Recent evidence localizes Gorlin's syndrome locus on chromosome 9(30). Both tumours and malformations of central nervous system occur with Gorlin syndrome. Medulloblastoma is the primary brain tumour most frequently associated with this syndrome. Over 40 such cases have been reported, however only 7 cases of meningioma associated with Gorlin's syndrome have been described(30).

In regard to cerebral metastases which are considered to be the most common CNS tumours, metastases occur in one quarter of patients with systemic cancer. Spread to the calvarium, brain parenchyma and subarachnoid space occur through several mechanisms(9).

Haematogenous tumour embolism, direct extension of tumours originating in the head and neck(9) and tumour passage from the eye or through the choroid plexus to the brain and subarachnoid space(9).

It is important to mention that 60% of metastasis occurs in the setting of diagnosed systemic cancer(9). Cancer of lung in men and of breast in women account for the largest percentage although melanoma is the tumour with the highest likelihood of spread to the CNS. Twenty percent develop neurological symptoms before discovery of the primary
tumour\(^{(9)}\). At some point after diagnosis of systemic cancer 25% of patients with lung carcinoma, 6 to 20% of patients with breast carcinoma and about 5% with those of melanoma develop tumours in brain\(^{(9)}\).

More often than is the case with primary brain tumours, those of metastatic origin occur in a setting of seizure activity, increasingly severe head pain and motor weakness. These difficulties evolve over days to weeks\(^{(19)}\). Ten percent of patients with cancer develop neurologic difficulties in the absence of an intracranial mass on CT. Most of these will be found to have abnormalities on MRI. Neurologic symptoms in patients with systemic cancer should not always be considered sequelae of metastasis because cerebrovascular disease may be due to:

- Multiple cerebral infarctions.
- Encephalopathy due to DIC leading to infarcts in patients with lymphoma and leukaemia.
- Paraneoplastic syndromes\(^{(9)}\).

Neurologic symptoms can be complications of tumour therapy such as headache, sleepiness, ataxia, dementia or gait apraxia, atherosclerosis with increased risk of strokes and peripheral nerve involvement. Other complications of tumour therapy may include restrictive lung disease and pneumocysts carinii pneumonia\(^{(31,32)}\).

**Investigations**:

Brain imaging:

Brain imaging by MRI or CT scans is an indispensable component of modern diagnosis of the presence but not the type of brain tumours. One type of tumour can look like another or even resemble non neoplastic
mass lesions, such as brain abscess, fungal infection, parasitic invasion, demyelinating disease or strokes.

For definitive diagnosis and adequate treatment planning, one must obtain a tissue diagnosis whenever possible. This can be done either by direct surgical biopsy or in the case of some non neoplastic conditions by judging CT or MRI responses to particular therapies.

**MRI:**

MRI is almost always superior to CT scanning in diagnosing intracranial mass lesions. MRI outlines posterior fossa structures and tumours with a clarity that CT cannot achieve, because of x-ray distortions due to the bony structure of that region. In several types of tumours, particularly the low grade gliomas, MRI may show extensive brain infiltration in cases that fail to produce any image abnormality on CT or, at most, a vague low density\(^{10}\). Although either MRI or CT should be used with contrast enhancement in cases of suspected brain tumour, the passage of such contrast agent beyond the blood brain barrier into the tissues does not necessarily imply the presence of a histologically malignant tumour\(^{10}\). For example, malignant glioma almost always show contrast enhancement, so do meningiomas which are entirely benign if they can be fully removed surgically. MRI is better than CT in detecting multiple lesions\(^{10}\).

Gadolinium MRI characteristics of brain tumours:-

- Metastases are remarkably variable, some inhahe brighty and solidly with gadolinium. Others are in ring configuration many are invisible with contrast CT.
- Acoustic neuromas, these are invariably intensely contrasted by gadolinium, even more reliably than by CT.
- Meningiomas shows the same findings as for acoustic neuromas.
- Pituitary adenomas always enhance less than the normal pituitary gland. MRI is superior in every way to CT, especially when thin slices and magnified views are ordered.

- Glioblastoma are almost always in ring configuration.

- Anaplastic astrocytomas are sometimes solidly bright, they are often patchy, may be non contrasting, and may look like low grade astrocytoma.

- Low grade astrocytomas do not enhance. They are often invisible by CT or are imaged only as vague low density masses.

- Oligodendrogliomas generally do not enhance unless anaplastic and are often invisible on CT unless they are calcified.

Primary brain lymphomas usually exhibit homogenous enhancement and are smoothly rounded. Periventricular location is common. They are multiple in about a fourth of cases. This lesion often does not look like glioblastoma but is easily mistaken for metastases if multiple.

MRI initially provided two types of images, designated T1 and T2. For brain tumours, the former generally showed a well demarcated area of low density and the latter, bright whiteness that encompassed a more extensive region owing to the signal of the surrounding brain oedema. With the availability for general usage in 1988 of gadolinium contrast for MRI, a new set of criteria of usage and differential diagnostic considerations in brain imaging have quickly evolved (see above). TI gadolinium imaging is the most precise way to image brain tumour and often patients can be followed during and after treatment with that type of study alone. Such an approach is easier for patients because it reduces the length of time otherwise spent on T2 scanning. Now and then T2 images are useful. For example, T2 images, besides showing the extent of
oedema, also delineate the demyelinating effects of radiation upon white matter \(^{(10)}\). CT scans done without contrast enhancement are of little value in the diagnosis of brain tumours or other mass lesion. Although it is true that haemorrhage, calcifications, hydorcephalus and shift can be well seen on a non contrast CT scan the interpretation of even these conditions is tentative because each can have an underlying causative structural abnormality such as brain tumour, which may fail to appear on a non contrast CT study. Allergy to CT dye is rare and readily manageable. Currently available nonionic CT dyes have an extremely low incidence of side effects. There is little risk that currently used CT dyes will cause renal dysfunction in normally hydrated patients who are not known to have kidney disease\(^{(10)}\).

CT attenuation of cerebral tumour (relative to normal brain) may appear as follows:\(^{(33)}\):

1. Hyperdense neoplasms:
   a) Meningioma 95%.
   b) Microglioma (primary lymphoma).
   c) Metastases 30%.
   d) Gliomas 10% (most glioma showed mixed attenuation).
   e) Ependymoma.
   f) Papilloma.
   g) Medulloblastoma 80%.
   h) Pituitary adenoma 25%.
   i) Craniopharyngioma (if solid).
   j) Acoustic neuroma 5%.
2. Isodense neoplasms:-
   a) Acoustic neuroma 95%.
   b) Pituitary adenoma.
   c) Glioma 10%.
d) Metastases 10%.

e) Chordoma.

f) Pinealoma.

3. Hypodense tumours:

a) Craniopharyngioma.

b) Glioma (95% of astrocytomas).

c) Metastases.

d) Prolactinoma.

e) Haemangioblastoma.

f) Lipoma.

g) Epidermoid.

h) Dermoid.
## CT appearances of cerebral tumours

<table>
<thead>
<tr>
<th>Tumour</th>
<th>Attenuation</th>
<th>Surrounding oedema</th>
<th>Contrast enhancement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glioma</td>
<td>Increased or decreased (if cystic, necrotic)</td>
<td>Yes</td>
<td>95% if high grade relatively infrequent in low grade, often irregular ring, but may be homogenous or patchy.</td>
</tr>
<tr>
<td>Metastases</td>
<td>Increased or decreased often multifocal.</td>
<td>Yes, extensive</td>
<td>Marked, may be irregular ring, homogenous or patchy.</td>
</tr>
<tr>
<td>Meningioma</td>
<td>Increased, multifocal in 5%</td>
<td>Minimal, perifocal (moderate in 20%).</td>
<td>Marked, homogenous.</td>
</tr>
<tr>
<td>Microglioma primary lymphoma</td>
<td>Increased (occasionally decreased, infiltrating) multifocal 50%.</td>
<td>Yes</td>
<td>Marked, homogenous.</td>
</tr>
<tr>
<td>Pituitary adenoma</td>
<td>Isodense 65%, increased 25%</td>
<td>No</td>
<td>Marked, homogenous.</td>
</tr>
<tr>
<td>Prolactinoma</td>
<td>Hypodense</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Craniopharyngioma</td>
<td>Decreased if cystic, increased if solid</td>
<td>No</td>
<td>±moderate, homogenous (solid) or ring (cystic).</td>
</tr>
<tr>
<td>Pinealoma</td>
<td>Isodense</td>
<td>No</td>
<td>Marked, homogenous.</td>
</tr>
<tr>
<td>Acoustic neuroma</td>
<td>Isodense 95%</td>
<td>No</td>
<td>Marked, homogenous.</td>
</tr>
<tr>
<td>Epidermoid</td>
<td>Decreased</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Medulloblastoma</td>
<td>Increased</td>
<td>Minimal perifocal</td>
<td>Moderate, homogenous.</td>
</tr>
<tr>
<td>Haemangioblastoma</td>
<td>Decreased</td>
<td>Minimal</td>
<td>Moderate, homogenous.</td>
</tr>
<tr>
<td>Dermoid</td>
<td>Decreased</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Lipoma</td>
<td>Decreased</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Papilloma</td>
<td>Increased</td>
<td>No</td>
<td>Marked, homogenous ± patchy.</td>
</tr>
<tr>
<td>Glomus jugulare</td>
<td>Isodense</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

• Angiography:—

Seldom is used in the diagnosis of brain tumours, although it may reveal tumour blush or vessel displacement with enlarged early draining veins. In a few circumstances, neurosurgeons, in preparation for surgery, require a more precise knowledge of the pattern and position of blood vessels that can be obtained only by angiography. The procedure
is also used to embolize highly vascular meningiomas, or to study cerebral dominance by injection of sodium amytal into the carotid artery (the wada test) in left handed individuals who are to have surgery near language areas. Preoperative determination of cerebral localization helps surgeons to plan the extent of surgery and avoid postoperative language deficits.

- Isotop scanning:-
  
  Useful in detection of supratentorial intracranial pathology if CT scanning is unavailable but will not distinguish the nature of the lesion.

- CSF examination:-

  Lumbar puncture is contraindicated if the clinician suspects intracranial tumour. If CSF is obtained by another source, e.g. ventricular drainage or during shunt insertion it may help to rule in or out an inflammatory disorder mimicking brain tumour. Another is to establish the diagnosis of benign intracranial hypertension in patients with uninformative MRI images. In addition, spinal fluid cytology may be useful for determining instances of malignant meningitis secondary to metastatic neoplasms, in association with spinal spread of medulloblastoma in children and in identifying primary lymphomas of the brain in cases in which MRI changes are ambiguous.

- EEG

  The routine electroencephalogram (EEG) has no role in the diagnosis of brain tumours, and does not assess in the choice of anticonvulsant drugs for brain tumour patients. However specialized intraoperative monitoring may be useful in identifying and removing epileptogenic areas adjacent to brain tumours, or to avoid resection of critical brain regions adjacent to tumours.
• Plain skull x-ray:-

* General features of increased intracranial pressure:-
  Separation of cranial sutures in infants.
  Beaten silver appearance or finger prints.
  Sellar changes.
  Enlargement of sella tursica.
  Rarefaction and destruction of the dorsum sellae and posterior clinoids.
  Lateralizing signs denoting the site of the tumour.
  Shift of calcified pineal body or falx cerebri.
  Signs denoting the site of the tumour.
  Localized calcification.
  Localized erosion and destruction of the skull bones (8).
  Osteolytic lesions.

• Positron emission tomography (PET):-
  Is able to quantify biochemical functions, such as oxygen and glucose utilization, within tumour as well as normal brain tissue (10). PET scanning is a powerful research tool of limited availability for routine clinical purposes. Its spatial resolution is inferior to CT and MRI in-patients with brain tumours who develop recurrent symptoms after radiation therapy. PET can differentiate with about 70% accuracy.
radiation-induced injury from tumour recurrences\textsuperscript{(10)}. These disorders appear identical on MRI.

- Tumour markers:
  Attempts to find a substance in blood or CSF which reflects growth of a specific tumour have been limited, but the link between elevated alfa fetoprotein and human chorionic gonadotrophines with germinomas of the third ventricle aids diagnosis. The development of monoclonal antibodies, with further improvement in their specificity may provide a useful approach to tumour localization and identification in the future\textsuperscript{(8)}.

Manjula et al. found that ceruloplasmin is elevated in all cases of brain tumours except in meningioma and they concluded that ceruloplasmin may have a role to play as an acute phase reactant protein in brain tumours\textsuperscript{(34)}.

**Treatment:**

In almost every instance when a brain tumour is suspected on the basis of combined results of history, physical findings and imaging studies the first consideration is surgical resectability. There are exceptions such as cases of multiple brain metastases in a patient with known systemic cancer.

**Medical treatment:**

The medical treatment of cerebral tumours can never be anything more than temporary or palliative. Relief of raised intracranial pressure is often required when surgery is not possible or when life is threatened before investigation has revealed the diagnosis. The main therapeutic agent is dexamethasone, which can reduce symptoms caused by peritumoral oedema, it has a long biologic half life and steady action upon
the brain, which made it the steroid of choice for treating patients with brain tumours. It should be started with an oral dose of 16 mg, followed with 8 mg twice daily, it is well absorbed by the mouth, and its action by that route is almost as rapid as when given intravenously. If focal neurologic symptoms are due to peritumoral vasogenic oedema, dexamethasone induces improvement within 48 hours and usually sooner. If there is no benefit, the neurologic symptoms are likely to be due to damage of the brain tissue by the tumour and not to oedema.

In instances of extreme intracranial pressure, the speed and action of dexamethasone are not sufficient to reduce the brain swelling quickly enough to prevent complications. In such instances hyperosmotic solution of mannitol must be given. The usual dosage is 0.5 to 2 grams per kilogram given intravenously over 15 minutes, followed by additional boluses of 25 grams as needed. The osmotic action of mannitol occurs within minutes. Clinical improvement may be dramatic. It is unusual for brain tumour patients preoperatively to decompensate so severely from increased intracranial pressure that intubation becomes necessary. Nevertheless this does occur. In such cases the Paco₂ must be decreased by passive hyperventilation to approximately 25 mmHg. The effect constricts the cerebral vasculature and promptly induces a major reduction of intracranial pressure which can be life saving.

About 20% of patients with brain tumours develop seizures sometimes in their lives even if they do not have seizures at the time of diagnosis. It is conventional and probably effective to treat all patients with supratentorial tumours with anticonvulsants before surgery. Most patients with acoustic neuromas or other posterior fossa tumours have a low probability of convulsive seizures and do not need such drugs. Phenytoin is the best initial drug, it can be administered intravenously or
orally, unlike either carbamazepine or valproic acid which can be used only orally. An intravenously given drug is specially useful for continuation during the preoperative period. If required, patients may be switched easily to alternative oral drugs later. Phenytion should be started orally giving 1000 mg over 12 hours, or intravenously, with 1000 mg given over 1 hour. Thereafter the usual dosage is 300 to 400 mg daily, administered in one dose or split between breakfast and dinner along with dexamethasone. Periodic blood level need to be checked to adjust the dosage to ensure concentration of 10 to 20 microgram/ml.\(^{(10)}\)

**Surgery:**

- The principal aims of surgery should be: -
  - To establish a pathological diagnosis.
  - Effective symptomatic relief.

  Complete surgical excision should be the ultimate goal, but even potentially curable tumours such as meningiomas or acoustic neuromas may reside in position that make complete resection technically impossible. Malignant gliomas lack microscopic boundaries, even though they may appear by imaging studies to have well defined limits, how much success can be achieved with these tumours depends on several factors, including the tumour's proximity to indispensable areas, the skill and the experience of neurosurgeon, and the preoperative level of neurologic function. The combination of current standards of neurosurgical anaesthesia, the capacity to control intracranial pressure, and the recent addition of lasers to other operative tool such as operating microscope have greatly increased the surgeon's capacity for well chosen radical resection correspondingly, the relative risks of surgery have become less age dependent than was previously the case. The
greatest surgical risk is to neurologic function and the fear of unacceptable postoperative neurologic deficit. For this reason, radical operations upon tumours involving language area, sensorimotor regions, the basal ganglia, corpus callosum and brain stem are generally avoided. However, practical removal in these areas by stereotaxic methods may be surprisingly effective. MRI images facilitate such surgery by showing that the tumour has pushed aside critical brain structures and that macroscopic tumour edge can be delineated. It has been repeatedly shown that resection of the maximal amount of tumour consistent with functional preservation provides patients with better and longer lives. A number of tumours cannot be even partially resected because they invade indispensable areas of the brain. Most are intraxial tumours, such as gliomas. Although imaging techniques may produce a characteristic picture suggestive of a particular histologic diagnosis, treatment planning demands a tissue diagnosis. All brain regions may be approached by MRI guided stereotaxic biopsy. The tissue specimens are small, but they are almost invariably adequate to establish a diagnosis. Haemorrhage occurs in about 2% of patients subjected to stereotactic surgery and these patients need to remain in hospital for less than 48 hours. Open biopsies of the brain tumours are not justifiable. If the skull and dura are to be opened, the surgeon should be prepared to do a gross total resection, or at least, a major removal of as much tumour as is consonant with preservation of neurologic function. Dexamethasone should be administered at adequate levels for at least 5 days to minimize surgically induced brain oedema.

Stereotactic surgery:

The first stereotactic frame is attributed to Hoarsely and Clark, who developed an apparatus, which was used on experimental animals, for
carrying out cerebellar stimulation\textsuperscript{(22)}. A prototype derived from this frame was made for use in humans but was never put to clinical use. The underlying principle of stereotaxy is that any point in space can be identified in relation to three planes running perpendicular to each other. In frame based stereotaxy a device is fixed to the head, so that coordinates of any points with the confines it’s self or an imaginary extension of it can be accurately localized. This requires computed tomography or magnetic resonance imaging to be performed after the frame has been applied to the head, so that the position of the region of interest can be related to the frame. With the coordinates thus determined, multiple targets can be selected so that biopsies can be taken as required. In the operating theatre one or more burr holes may be placed and the biopsies taken. Communication between the surgeon and pathologist ensures that abnormal tissue is obtained for analysis. This technique may be used not only for brain tumour biopsy but also for localizing craniotomies for minimally invasive excision of tumours. It can also be used in tumour management for catheter placement, for drug or isotope delivery or cyst drainage. This method of surgery has much wider applications and is used for the functional treatment of movement disorders and pain. An extension of the stereotaxic process is a preoperative scan without the use of the frame and then use a mathematical algorithm to correlate with the surface markings of the skull at the time of surgery. This so called ‘frameless stereotaxy’ is potentially less clumsy and time consuming although, like frame based stereotaxy, it has to be remembered that the images are not contemporaneous with operative findings and therefore any shift of intracranial structures upon craniotomy can not be taken into account by the device\textsuperscript{(22)}. 
Radiation therapy:

All forms of external beam radiation whether photons emitted from $^{60}$Co sources or x-rays generated from linear accelerators, act similarly. They produce fast moving electrons and free radicals in biologic tissue that interrupt chemical bonds between DNA base pairs. Affected cells either die or become so altered that their mitotic rate is greatly diminished. Radiation therapy is given in small daily fractions to build to a total dose. It appears safer and more effective to do this than to give larger fraction over shorter periods. Hyperfractionation defined as two (or more) doses during a day, does not seem to be worthwhile. Therapeutic brain irradiation with particulate radiation such as neutrons has been attempted experimentally at facilities with cyclotrones. Such density ionising radiation has shown no therapeutic advantage, over x-rays. Interstitial (implanted) radiation therapy (brachy therapy) is usually given in the form of $^{125}$I or $^{192}$Ir in 'seeds' placed by stereotaxic techniques. This method permits localized high dosage radiation with sharp edges and sparing of the adjacent brain. Considerable controversy exists about the utility of interstitial radiation therapy, but it can be effective in well selected patients. Other non operative radiosurgical techniques include 'the gamma knife' and linear accelerators adapted to provide focused therapeutic beams. Efficacy has been shown for metastases but not for gliomas. External beam radiation therapy has value in controlling the growth of malignant gliomas and metastatic brain tumours. It doubles median survival time for both types of tumours. Radiation therapy has little, if any value for recurrent meningiomas and acoustic neuromas. These are almost invariably better handled by reoperation. Primary brain lymphomas are so responsive to radiation therapy that many neurologists and radiation therapist continue to use it alone despite the fact that chemotherapy may
prove to provide superior initial treatment. It has already been mentioned that solitary brain metastases are best managed by surgical resection before radiation therapy.

The complication of radiation therapy are often reported as infrequent, perhaps 2-5% of cases. These figures are unrealistically low if one includes effects on long term survivors. A high percentage of patients receiving whole brain radiation develop dementia or impaired mobility\(^{(10)}\).

**Chemotherapy:**

For brain tumours it has had disappointing record. The reasons are many, but inadequate drug delivery, tumour cell heterogeneity and inherent resistance are among the important ones\(^{(10)}\). Almost all effort have been directed towards the primary brain tumours especially the gliomas. Established brain metastases however, respond about as well as systemic metastases do in many cancers, especially breast and small cell lung cancer. BCNU (bischloroethylnitrososurea), the most frequently used drug, remains the most effective single agent available to treat the malignant astrocytomas. The combination of procarbazin, CCNU (cyclohexylchloroethylnitrosurea), vincristine is the most effective multidrug regimen for the malignant astrocytomas, and it is probably superior to BCNU. It has an unusually beneficial effect against oligodendrogliomas. No more than 10% of patients with malignant gliomas have meaningful and durable responses to chemotherapy, whether it is given immediately after radiation therapy (when its effect is especially hard to assess) or at the time of recurrence. Efforts to improve response to chemotherapy by delivering drugs through the carotid artery have not been successful. BCNU has intolerable toxicity when given by the intracranial route. Cisplatin is studied for possible utility as intrarterial drug in highly selected patients.
The pharmacokinetics of drugs used in brain tumour chemotherapy are not well understood. Knowledge about their ability to gain adequate concentration within the tumours is minimal and almost nothing is known about chemosensitivity. Nonetheless, occasional remarkable responses to chemotherapy do occur in patients with gliomas\(^{(10)}\). Among other primary intracranial brain tumours, primary brain lymphoma has a reasonably good response rate. The drugs used are those given regularly for systemic lymphoma. Patients with primary brain lymphoma do better with chemotherapy added than with radiation alone. On average, 3 to 4 year survival can now be expected\(^{(10)}\).

Several additional forms of medical treatment for brain tumours have been attempted experimentally. These include slow release of BCNU from implanted biodegradable polymers and the administration of interferons, other biologic response modifiers and radionuclides coupled with monoclonal antibodies. None has met with appreciable success to date. In all probability, much new biologic knowledge, such as sequential genetic events that influence malignant transformation and progression of brain tumours, will be required before new medical treatments become practical medical realities\(^{(10)}\).

**Prognosis:**

Gliomas can rarely be completely excised and infiltration may spread beyond the radiologically evident boundaries of the tumour. Recurrence is common even if the mass of the tumour is apparently removed, although survival in some cases may be prolonged for several years. Partial excision may be useful in alleviating raised intracranial pressure, but survival in highly malignant gliomas (glioblastoma multiforme) is measured in months even if such a decompressive
procedure is attempted. Ependymomas and medulloblastomas may be excised with minimal residual disability, but often recur with seeding of the tumour via the cerebrospinal fluid. Oligodendrogliomas are often slowly growing and relatively benign in the early stages but may transform to a more malignant form and behave as gliomas. The prognosis in benign tumours is very good provided complete surgical excision has been achieved.\(^{19}\)

Specific types of brain tumours:-

Gliomas:-

Are derived from glial cells and three main types are recognized.\(^{35}\)

1. Astrocytoma.
2. Oligodendroglioma.
3. Ependymoma.

Astrocytoma:-

This is the commonest primary intracranial tumour (11.9\%)\(^{8}\) and also by far the commonest glioma (36\%)\(^{7}\).

Low grade astrocytoma and well differentiated astrocytoma:- Median age at onset is the third to fourth decade of life, it may occur in all parts of CNS but most common sites are the frontal and temporal lobes.\(^{14}\)

Malignant astrocytoma, Anaplastic astrocytoma and Glioblastoma multiforme:-

The age at onset is usually 40-60 years with a male to female ratio of 2 to 1.\(^{8,5}\) Malignant astrocytoma occurs anywhere in the neuroaxis and affect both hemispheres with equal frequency.\(^{14}\)
Poor prognostic features are: histological evidence of tumour necrosis, older age, duration of symptoms less than 6 months. Familial examples constituting only 1% of cases.

**Oligodendrogliaoma:-**

These are slow growing tumours. The median age of onset is 40 years, they are typically frontal lobe in location but may occur in posterior fossa or spinal cord. Approximately 6% are histologically malignant.

**Ependymoma:-**

Ependymomas are located near the ependymal surfaces. Most are in the posterior fossa, but many are supratentorial. The peak age incidence in children is from birth to 4 years of age. It may be seen in young adults. Subarachnoid seeding occur in high grade tumours and tumours of the posterior fossa.

**Meningioma:-**

Intracranial meningiomas are twice as common in females as in males. Their incidence may be increased after ionising radiation and in NF2. Many meningiomas show a deletion on chromosome 22. They are primarily present in the 40-60 age group. They constitute about one fifth of all primary intracranial tumours, occasionally they are multiple, specially in NF2. A reactive hyperostosis develops in adjacent bone, forming a swelling on the inner table. Hyperostosis affecting the outer table may produce a palpable lump. Tumour tissue may infiltrate the adjacent bone. They are principally benign tumours, although a malignant form exists. Common sites for intracranial meningiomas are:
parasagittal (24%), followed by lateral convexity (18%) and in the
sphenoid ridge in equal proportion. Other common sites include,
olfactory groove (10%), tuberculum, CPA, petrous ridge, foramen
magnum, lateral ventricle and optic nerve sheath.

**Craniopharyngioma:**

This accounts for (2.5%) of primary intracranial tumours. Are
more common in children than in adults and often reach substantial size
before detection. They may extend into the hypothalamus, third
ventricular region, optic chiasm and frontal lobes. They are typically
calcified and cystic, permitting preliminary diagnosis by MRI or CT. The
usual initial features are headache, diabetes insipidus, somnolence
endocrinopathies, visual disturbances, visual field deficits and in children
dwarfism. Growth starts near pituitary stalk, but may extend in many
directions. Despite excellent survival rates, treatment causes diabetes
insipidus, panhypopituitarism, obesity, radionecrosis and intellectual
impairment.

**Pituitary Adenoma:**

This is found in adult life and accounts for 6.8% of all primary
intracranial tumours and for 10% in other reports. Abu Salih and
Abdul-Rahman reported that adenoma accounts for 10.6% of all
intracranial tumours. It was common as they mentioned in third decade
and males were affected more commonly than females. They arise from
the anterior portion of the gland and are usually benign. Acromegaly
occur in 10-15% of all pituitary adenomas, hypogonadism in 25-40%
Cushing’s disease in 10-15% and hypopituitarism in 10-15%.
Acoustic neuroma:–

Is the commonest infratentorial tumour, constituting 6% of all primary intracranial tumours and 80% of CPA lesions\(^8\). They are usually present in middle age (40-50) years and occur more frequently in females\(^8\). Bilateral neurilemmomas occur in 5% of patients and are characteristic of type NF\(_2\). They are benign, slowly growing tumours which arise primarily from the vestibular portion of the VIII cranial nerve and lie in CPA. Rarely these tumours arise from the fifth cranial nerve. Abu Salih and Abdul-Rahman reported a low incidence of acoustic neuromas in Sudan\(^2\).

Haemangioblastoma:–

This occurs primarily in middle aged\(^8\). It is slightly more prevalent in males. In some patients this tumour occurs in the spinal cord and retina in addition to the brain. It may be associated with other pathologies such as polycythaemia and cysts in the pancreas and kidneys. Cerebellar signs and the effects of CSF obstruction developed insidiously\(^8\). Occasionally subarachnoid haemorrhage occurs. In female patients, symptoms often appear during pregnancy. Polycythaemia due to increased erythropoetin production is common\(^8,14\).

CT scan shows a well defined low density cystic region in the cerebellum with strongly enhancing nodule in the wall. Occasionally multiple lesions are evident\(^8\).

Pineal tumours:–

Pineal and parapineal tumours are mixed group and the natural history of the individual components is uncertain since surgical access to them is often limited or hazardous and many have therefore been treated by shunt procedures and irradiation without biopsy.
This group includes teratomas, so called pineal gliomas, true pinealomas, and germinomas, the latter being the most common form. Some teratomas are ectopic in that they develop in the floor of the third ventricle and spread to the parapineal region. Pineal tumours quite often spread within cerebrospinal fluid pathways including the spinal canal. Histologically germinomas resemble ovarian dysgerminomas and seminomas and have a marked preponderance in males\(^3\).

**Cholesteatomas:**

These are developmental inclusion lesions in which epithelial remnants are incorporated into deeper layers\(^6\). They include epidermoid and dermoid cysts. The epidermoid cysts have a thin connective tissue capsule and contain material rich in cholestrol from the break down of keratin which is shed by the desquamating epithelial cells. Dermoid cysts may also contain follicles, sebaceous and sweat glands. These rare tumours are found predominantly in the cerebellopontine angle and in the parasellar region. They have a tendency to recur. Failure to remove the whole capsule results in further desquamation of cells and a gradual accumulation of the contents of the cyst\(^6\).

**BRAIN ABSCESS:**

**Definition:**

Brain abscess describes encapsulated or free pus in the substance of the brain. Abscess may vary in size from a microscopic focus of inflammatory cells to a major encapsulated area of necrosis occupying a major part of cerebral hemisphere. They may be single or multiple and caused by local extension or haematogenous spread\(^3\).
Incidence:-

Brain abscess constitutes approximately 0.7% at all neurosurgical operations\(^3\). The condition occurs two to three times more frequently in males than in females\(^{10}\). It is an uncommon disorder accounting only for 2% of intracranial mass lesions\(^{10}\). Brain abscesses, however, often progress more rapidly than tumours and more often affect meningeal structure\(^{10}\).

Aetiology:-

The causes of intracranial abscess in approximate order of frequency are:-

1. Infection of the middle ear or nasal sinuses and infection of the middle ear is from four to nine as common cause as is the sinus infection. The frontal sinus is the most often involved, the sphenoidal sinus is the next.

2. Pyemia or bacteraemia: this is now rare following wide spread use of antibiotics. But cerebral abscess may still arise as a consequence of infective endocarditis. It is rare in subacute endocarditis in view of the low virulence of the infecting organisms. Unsuspected bacteraemia in acute osteomyelitis and in other pyogenic infections including simple cutaneous sepsis is some times the cause.

3. Metastasis from intrathoracic suppuration:- It may complicates bronchiectasis. There is clear association between cerebral abscess and cyanotic congenital heart disease. Single or multiple abscesses may also develop in the immunocompromised patients, as after cardiac transplantation when the organisms may be aspergillus, toxoplasma, candida, klebsiella, cryptococcus, coccidioides, listeria, mucor or rhizopus.
4. Fracture of the skull is liable to cause abscess when injury leads to free communication between the body surface and the brain specially when fragments of bone, clothing or missile penetrate the latter.\(^{(3)}\)

Excluding cerebral abscesses occurring in AIDS (which are multiple and are most often caused by toxoplasma gondii); about 60% of intracerebral abscesses are caused by middle ear infection and are situated in the temporal lobe or cerebellum. About 20% are secondary to frontal sinusitis, which results in frontal lobe abscess. Approximately 10% are due to bacteremia or septicaemia from the lung (abscess, empyema or bronchiectasis), the heart (infective endocarditis), dental sepsis or other peripheral sites. With penetrating skull trauma accounting for a small proportion of cases and in about 10% of cerebral abscess no source of infection is found. Cerebral abscesses are more likely to occur in patients with debilitating illness and in the immunocompromised\(^{(36)}\).

According to another source, about 25% of brain abscesses are due to the disease of the middle ear, mastoids, or paranasal sinuses\(^{(9)}\). Another 25% are due to the contaminated, penetrating wounds or postoperative infection, and about 25% are metastatic\(^{(9)}\).
The following table shows the site, source and organism in cerebral abscess:

<table>
<thead>
<tr>
<th>Site</th>
<th>Source of infection</th>
<th>Likely organism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>Frontal sinusitis</td>
<td>Streptococcus</td>
</tr>
<tr>
<td>Temporal</td>
<td>Otitis media</td>
<td>Streptococcus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proteus</td>
</tr>
<tr>
<td>Cerbellar</td>
<td>Otitis media</td>
<td>Streptococcus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bacteroides</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proteus</td>
</tr>
<tr>
<td>Parietal</td>
<td>Embolic</td>
<td>Streptococcus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bacteroides</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proteus</td>
</tr>
<tr>
<td>Any site</td>
<td>Trauma</td>
<td>Staphylococcus</td>
</tr>
</tbody>
</table>

Mampalam *et al* mentioned that the aetiology of brain abscess was as follows in the period between 1970-1986:

- Local infection account for about 19%
- Cardiac for about 17%
- Surgery 16%
- Trauma 9%
- Pulmonary 9%
- Immunocompromise 6%
- Other 5%
- Unknown 21%

Cerebral abscess occurs in 1-5% of staphylococcus aureus endocarditis.
Pathology:-

It begins with vascular seeding of the brain parenchyma, producing early cerebritis during the first 1-3 days. Inflammatory infiltrates of polymorphonuclear cells, lymphocytes and plasma cells follow within 24 hours. By 3 days the surrounding area shows a marked increase in perivascular inflammation. The late cerebritis phase develops approximately 4 to 9 days after infection during which time the centre becomes necrotic, containing a mixture of debris and inflammatory cells. Neovascularity is maximal at this time. Early reactive astrocytes surround the zone of infection and proceed to early capsule formation between approximately 10 to 13 days. At this time, the necrotic centre shrinks slightly and a well developed peripheral fibroblast layer evolves. The late capsule stage continues to evolve between 14 days and 5 weeks with continual shrinking of the necrotic centre and relative decrease in the inflammatory cells. The capsule thickens as reactive astrocytes proliferate.

Bacteriology:-

- Staphylococcus Aureus is the commonest isolate in trauma related cases.
- Toxoplasma is most common organism in HIV associated disease and bacterial abscesses are rare.
- Anaerobic organisms but aerobic and microaerobic streptococci, staphylococcus aureus, bacterioids, proteus and other gram negative bacilli may also be found. Actinomyces, nocardia and candida are less frequent offenders. Infection is often polymicrobial. Culture negative abscesses from surgical specimens occur in 30% of antibiotic treated patients and in 5% of patients operated on before antibiotic administration.
Clinical features:-

Signs of infection may be minimal or absent. Almost half of affected patients maintain a normal body temperature and fewer than a third show a peripheral white cell count above 11,000 per microlitre\(^{(10)}\). Generally symptoms are composed of:-

1. Fever and general symptoms.
2. Symptoms of increased intracranial pressure (see brain tumours).
3. Focal symptoms (seen brain tumours).

Onset is gradual specially in metastatic abscesses\(^{(3)}\). The period of evolution may be as brief as many hours or as long as many days to weeks, with more indolent organisms. Seizures may occur with abscesses involving the cortical gray matter\(^{(10)}\). Presenting features in 43 cases of brain abscesses as reported in literature\(^{(10)}\).

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>72%</td>
</tr>
<tr>
<td>Lethargy</td>
<td>71%</td>
</tr>
<tr>
<td>Nausea, vomiting</td>
<td>35%</td>
</tr>
<tr>
<td>Seizures</td>
<td>35%</td>
</tr>
<tr>
<td>Ocular palsy</td>
<td>27%</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>12%</td>
</tr>
<tr>
<td>Papilledema</td>
<td>10%</td>
</tr>
<tr>
<td>Haemiparesis</td>
<td>9%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>60%</td>
</tr>
<tr>
<td>Nuchal rigidity</td>
<td>49%</td>
</tr>
<tr>
<td>Confusion</td>
<td>26%</td>
</tr>
<tr>
<td>Visual disturbance</td>
<td>21%</td>
</tr>
<tr>
<td>Weakness</td>
<td>12%</td>
</tr>
<tr>
<td>Stupor</td>
<td>12%</td>
</tr>
<tr>
<td>Dysphasia</td>
<td>9%</td>
</tr>
<tr>
<td>Dizziness</td>
<td>7%</td>
</tr>
</tbody>
</table>

Investigations:-

Cerebrospinal fluid examination:-

CSF examination is not useful in diagnosis because the findings range from normal to those of purulent meningitis\(^{(10)}\), more important because abscess often expand rapidly, lumbar puncture may aggravate impending transtentorial herniation. If possible procedure should be
deferred until after brain images are obtained which may eliminate the need for CSF analysis\(^{(10)}\).

**Neuroimaging:**

CT and MR are imaging studies of choice for brain abscesses and monitoring their response to therapy. MRI is especially useful for posterior fossa abscesses, as it provides an artifact free view of the brain stem and cerebellum. In addition, MRI with intravenous gadolinium contrast is superior in demonstrating cerebritis surrounding oedema, the extent of the mass effect, or associated venous thrombosis. MRI with or without gadolinium is preferable to CT scan for demonstrating multiple lesions. The evolution of the abscess can be followed radiologically. In the early cerebritis stage, CT images reveal a low density lesion with ring enhancement. In the late cerebritis and early capsule stage, well-formed, ring enhancement is typically thin walled and uniform with subtle medial thinning adjacent to the ventricular system. Thick, nonuniform, or nodular enhancement should raise the possibility of an alternative cause. Delayed contrast scans show diffusion of the contrast material into the lucent center. In the late capsule stage, well formed ring enhancement may be seen with no delayed diffusion of the contrast. Other ring enhancing lesions that may mimic the image of the brain abscess include primary and metastatic tumour, a resolving infarct or haematoma and rarely demyelinating disease\(^{(10)}\).

**Treatment:**

Pyogenic brain abscesses are treated with antibiotics combined with surgical aspiration or excision.

**Medical treatment:**

In selected cases, antibiotics alone are appropriate, as in the case of surgically inaccessible, multiple abscesses, or abscesses in early cerebritis stage. If the causal organism is not identified, antibiotic coverage should be directed towards the most likely organisms (streptococci and anaerobes).
A suggested regimen includes penicillin G, 4 million units given intravenously every 4 hours and metronidazole 15 mg/kg intravenously over one hour, followed by 7.5 mg/kg intravenously or orally every 6 hours. If staphylococcal infection is suspected (because of a history of trauma or intravenous drug abuse), vancomycin should be added. Concomitant corticosteroids therapy may attenuate the oedema surrounding abscesses.

Aspiration offers the advantage of identifying the infecting organism and may be performed stereotactically with CT guidance, while the patient is under local anaesthesia.

Surgical therapy: This requires craniotomy. It is required when significant mass effect is present, when the abscess adjoins the ventricular surface (raising the possibility of catastrophic rupture into the ventricular system), when abscess arises in the posterior fossa (with the potential of brain stem compression) or when abscess reaches a large size (>cm diameter) or become refractory to medical treatment.

The resolution of abscesses can be followed by serial CT or MRI. Antibiotics must be continued, until the abscess cavity resolves completely, usually within 6 to 8 weeks. Of note is that ring enhancement may persist despite clinical and CSF normalization.

Abscess due to toxoplasma gondii is treated with daily doses of sulfadiazine 12 to 15mg/kg orally and pyrimethamine 25 to 50 mg orally. Another alternative regimen is pyrimethamine orally and clindamycin 900 to 1200mg I/V every 6 hours for patients allergic to sulfa drugs.

Prognosis:-

Current mortality rate is 5-15% depending on the locale and the nature of preexisting illness.
Hydatid disease is caused by larvae of parasites of the genus Echinococcus, the adult of which is found in carnivores, which are the definitive hosts. The intermediate hosts are infected by swallowing eggs passed in the faeces of the definitive host. Two main forms of hydatid disease occur in man\(^{(38)}\). Echinococcus granulosus (unilocular hydatid) and E. multilocularis (multilocular hydatid).

E. granulosus:- It develops in dogs but generally not in foxes and uses ungulates as intermediate hosts. There are strains which are host adapted horse/dog is probably not infective for men. Other strains e.g. sheep/dog, buffalo/dog, camel/dog and deer and moose/wolf are all infective to man and further strains may be developing\(^{(38)}\).

E. Multilocularis:- Matures in foxes and other canines and uses rodents as intermediate host.

Geographical distribution:-

The most extensive and endemic areas of human infection are found in the sheep raising countries; South Australia, New Zealand, Tasmania, parts of North, South and East Africa the southern half of the south America, particularly Argentina and southern Brazil. In addition human infection is frequently found in south-west states of the USA, Southern and Eastern Europe, Iraq, Syria, Lebanon, Turkey, Mongolia, Turkestan, North China, Southern Japan and North Vietnam\(^{(38)}\).

Incidence:-

Amongst 1802 patients in Australian hydatid register, hydatid disease of the brain was found in 1\(^{(6)}\). According to another source it occurs in 2\% of patients with hydatid disease\(^{(33)}\).
Clinical Features:-

Generally are those of space occupying lesion and hydatid involvement of the brain is marked by slow mass effect, hydrocephalus and often seizures, occasionally metastatic lesions in the brain are the first to cause symptoms by local inflammation or mass effect\(^{(10)}\).

Diagnosis:-

Thirty percent may show eosinophilia. Confirmatory evidence of infection may be obtained by serology (sensitivity of 60% to 90% depending on the test used)\(^{(10)}\).

Neuroimaging:-

CT scan:- the appearances are cystic, spherical with a sharp border, a central absorptive value similar to CSF, no perifocal oedema and usually with significant ventricular distortion and a shift of midline structures. There is lack of enhancement and of the perifocal oedema seen in cerebral abscesses or of solid portions and perifocal oedema seen in cystic tumours\(^{(6)}\).

Cysticercosis:-

Cysticercosis represents human tissue infection with intermediate cyst forms of the pork T.solium tape worm. Cysticercosis is acquired by ingestion of T.sodium eggs in contaminated foods. The prevalence of infection is approximately 1 to 10% in endemic areas of Latin America, India, Asia, Indonesia and parts of Africa. Because of its potentially life threatening complications, cysticercosis has greater clinical significance than does intestinal T.sodium tape worm infection, particularly if cyst disease involves the CNS. Cysticerci are bladder-like, fluid-filled cysts, containing an invaginated protoscolex. They are often surrounded by a dense fibrous capsule of host origin. Many patients have minimal, if any,
symptoms of infection. However, symptomatic neurocysticercosis requires medical attention. This syndrome has an estimated mortality of up to 50%\textsuperscript{(10)} and any neurologic, cognitive, or personality disorder in an individual from an endemic area should be considered a possible manifestation of undiagnosed cysticercosis. Neurocysticercosis may be divided into following discrete syndromes:-

In the acute invasive stage of cysticercosis, the patient may experience fevers, headache, myalgia and eosinophilia. Heavy infection at this stage may be associated with picture of 'cysticercal encephalitis' associated with coma and rapid deterioration. prevalence

After cysticerci become established, parenchymal CNS cysticercosis (50% of cases) is associated with seizures, intellectual impairment and personality changes. Compression due to swelling or inflammation around the cysts may result in focal deficits, signs of cerebral oedema and or hydrocephalus. Seizures may be focal or generalized.

Subarachnoid cysticercosis (30% of cases) is frequently associated with obstruction of cerebrospinal fluid flow and there may be intracranial hypertension. Sensorial changes may include apathy, amnesia, dementia, hallucination and emotional disturbance. Like other forms of basilar meningitis, pericysticercal inflammation at the base of the brain may cause obstruction or vasculitis of the cerebral arteries, leading to intermittent ischaemia or stroke.

Intraventricular cysticercosis (15% of cases) is the most difficult to diagnose and treat. Symptomatic cysts are most frequent in the fourth ventricle, where they cause outflow obstruction and increased intracranial pressure without localizing signs. An aggressive variant of ventricular neurocysticercosis is called racemose cysticercosis, frequently involves basal cisterns\textsuperscript{(10)}. Another syndromes are spinal and ocular cysticercosis.
In Australia Young et al found only 4 cases of neuro-cysticercosis over 10 year period (1984-1994) and most of the patients were emigrants \(^{(39)}\). They found that epilepsy, aseptic meningitis and raised ICP are common symptoms.

**Diagnosis:**
- Definitive diagnosis is by examination of tissue cysts obtained by biopsy.
- High index of suspicion in endemic areas \(^{(39)}\).
- Neuroimaging - CT, MRI (multiple, low density, enhanced and unenhanced lesions). Infection with *T*. *solium* is present in about 25% of patients with neurocysticercosis\(^{(10)}\).
- CSF shows hypoglycorrhachia, elevated total protein level, lymphocytic and eosinophilic pleocytosis (5-500 cell/microlitre).
- ELISA has sensitivity of 75%-100%\(^{(10)}\).

**Treatment:**

Surgery may be risky or technically difficult and should be reserved for patients with intraventricular cyst, for whom shunting may offer some palliation with anticonvulsant and anti-inflammatory drugs.

Drug therapy with praziquantel 50 mg/kg in 3 divided doses for 14-30 days or albendazole 15 mg/kg for 15 days, has been associated with alleviation of symptoms and regression of cyst size and number in patients with viable cysts in the cerebral parenchyma\(^{(10)}\).

Del Brutto et al mentioned that administration of albendazole in patients with neurocysticercosis (single lesion) presenting as space occupying lesion may obviate unnecessary surgical procedures in selected cases\(^{(40)}\).

Other parasitic infections that cause features of space occupying lesions are: -
Sparganosis: -

Results from infection by plerocercoid larval stages of spirometra species tape worms of cats and other carnivores. Infection is usually by oro-fecal route.

Munckhof et al reported the first case of cerebral sparganosis in Australia and they concluded that clinicians should consider the possibility of unusual parasitic infections in refugees who present with intracranial space occupying lesions (SOL) especially those from developing countries\(^{(45)}\).

Chamadol et al mentioned that cerebral sparganosis is a rare condition and the clinical features are mainly due to parasitic granulomatous SOL\(^{(42)}\).

Treatment: -

The treatment of choice is ethanol injection and/or surgical removal, as limited experimentence, with medical antihelminthic therapy has shown no beneficial effect\(^{(10)}\).

Gnathostomiasis:-

This is an intestinal nematode of dogs and cats. Fishes are intermediate hosts. The infection is endemic in rodents in the Far East and Thailand. Human infection is also reported in South America\(^{(43)}\). Infective larvae are ingested by humans in raw or undercooked fish. Larvae do not complete their life cycle in humans but migrate through the body. In CNS gnathostomiasis haemorrhagic tracts may be seen in the brain\(^{(10)}\). Suntharasmas et al mentioned that human gnathostomiasis is characterized
by space occupying lesions and/or haemorrhages as a result of migration of the larva of gnathostoma spinigerum, this rare cerebral invasion may be fatal. They mentioned that albebdazole therapy stimulate outward migration of gnathostoma to the dermis in the man\(^{(43)}\).

**Schistosomiasis:-**

Nervous system involvement in schistosoma mansoni is rare\(^{(10)}\). The main clinical presentation, as in schistosomiasis haematobia, is transverse myelitis\(^{(10)}\). The preferential involvement of the spinal cord may be due to anatomic location of adult worm. The underlying pathologic lesions are usually granulomas forming around the eggs in the spinal cord\(^{(10)}\). Cerebral schistosomiasis japonica is a unique syndrome reportedly occurring in 2-4% of infected individuals in the endemic countries. Schistosoma japonicum infection of the central nervous system preferentially affects the brain\(^{(10)}\). Pollner JH, reported a case who presented with space occupying cerebral lesion and schistosomal granulomas were found on pathological examination\(^{(44)}\). Schistosoma haematobium was identified in urine and serologically. The lesion responded to therapy with praziquantel and corticosteroids. They mentioned that CNS is an unusual site of ectopic infection in schistosomiasis. Cerebral lesions are caused primarily by schistosoma Japonicu and spinal cord lesions are due primarily to schistosoma haematobium and mansoni\(^{(44)}\).

**Toxoplasmosis:-**

Toxoplasma gondii is a protozoon that commonly affects mammals and birds throughout the world. Toxoplasma gondii infection in humans is usually asymptomatic.
Toxoplasmic encephalitis is the most common manifestation, and is also the most frequent cause of intracerebral mass lesion in-patients with AIDS and is almost always due to reactivation of a chronic infection.

Diagnosis:

Is by PCR, serology, ELISA. PCR has been used successfully on CSF (10). Laing et al mentioned that treatment is effective but outcome of treated disease cannot be predicted from the presenting clinical or radiological features (45).

Cerebral aspergilloma:

Fardoun et al mentioned that the incidence of mycotic infection of CNS seems to have increased in the last few years and the diagnosis is being made more and more frequently (46). Reporting a case of aspergilloma presented as space occupying lesion they mentioned that 25 cases of Aspergillus granulomas have been reported up to 1990. This disease is included in the entity of neuromycosis, its specificity compared to other types of cerebral mycotic localization as for example; abscess, meningitis, mycotic aneurysm lies in the fact that it presents as granulomatous mass in the hemisphere mimicking a brain tumour without any specific neuroradiological findings. They stressed the necessity of early and vigorous management mainly in patients with risk factors to try to reduce high mortality that has been universally reported in almost all cases (46). Haran retrospectively studied intracranial aspergillus granulomas over 12 years period and found 3 distinctive types of presentations, namely rhinocerebral form, space occupying lesion form and patients with stroke like presentation (47).
Nocardiosis:-

Nocardiosis is a subacute or chronic bacterial infection that evokes a suppurative response. The most common sites of primary infection are, first, the lung, then the skin, from which the bacteria may disseminate haematogenously to CNS and other tissues. The infection often pursues a more acute and aggressive course in immunocompromised patients. The predominant human pathogen is a gram positive, aerobic actinomycete, many of which are weakly acid fast in tissue or initial isolation. However in 20-40% of patients with pulmonary nocardiosis, dissemination to the CNS occurs and therefore CT scan of the head should be considered (10). Loculated brain abscesses either singular or multiple, are common and are often accompanied by headache and focal findings. Meningitis is infrequent (10). Herkes et al reported three cases of cerebral nocardiosis, they mentioned that it is an uncommon but increasingly diagnosed infection in Australia (4). One of their cases presented with posterior fossa nocardial abscess. They mentioned that primary cerebral nocardiosis is rare, with only two cases of primary posterior fossa nocardiosis reported. They highlighted the difficulty of diagnosis and the need for aggressive treatment with combined approach of surgical drainage and antibiotic therapy. The antibiotic regime of choice is a subject of controversy. Rifampicin, cephalosporines, imipenem, sulphonamides and other agents are used with varying success (48).

Human Cytomegalovirus (HCMV):-

It has been shown that CNS damage associated with congenital human cytomegalovirus infection (HCMV) is permanent, but subclinical congenital infection is less commonly associated with permanent sequelae. Acquired infection although uncommon in allograft recipients it is not infrequent in HIV infected patients. Encephalopathies
HCMV. The most common and important disease associated with CMV is retinitis\(^{(10)}\). However, Moulinger A\(^{(49)}\) reported many cases of AIDS associated cytomegalovirus infections presenting as cerebral space occupying lesions. In these patients MRI showed ring enhanced masses and marked oedema\(^{(49)}\). They stated that HCMV infection should be considered as a cause of ring enhanced, space occupying mass in patients with HIV infection. The patients' condition improved with specific therapy. Moreover earlier identification of these unusual tumour forms of HCMV infections by means of MRI should result in improved outcome. Also HSV may produce features of SOL\(^{(9)}\).

**African Trypanosomiasis:**

Sonan et al. mentioned that hemiplegic forms of African trypanosomiasis are unusual, from 1963 to 1987 only 14 cases have been reported\(^{(50)}\). They mentioned that it could present with features gestive of SOLs\(^{(50)}\).

**Ichthyoid Cysts:**

These are benign lesions filled with cerebrospinal fluid-like fluid. These lesions are probably developmental in origin and become symptomatic either because of their progressive enlargement or because of haemorrhage into the cyst. These cysts occur throughout the roaxis, and generally no communication is demonstrated between the and the subarachnoid space, although occasionally during surgery, an ichthyoid cyst is observed being filled through an apparent one way valve. Ichthyoid cyst may spontaneously regress but if they do become...
Lesions largely depend on their location whether over the sylvian fissure, over the cerebral convexity, in the interhemispheric regions, in the sella and suprasellar region, around the optic nerve and quadrigeminal plate or CPA in the region of clivus, over the cerebellar vermis, or cerebellar hemisphere, or within the lateral or fourth ventricle. Arachnoid cysts have also been described extending across the region of the foramen magnum from the posterior cranial fossa into the upper cervical spine posterolateral to the spinal cord. The midline lesions often lead to obstruction of CSF and result in focal symptoms and raised intracranial pressure.

The arachnoid cyst wall is histologically indistinguishable from normal arachnoid membrane. Moderate thickening of the arachnoid and an increase in connective tissue is common.

Ultrastructural studies confirm the similarity of the cyst membrane, with the normal meningeal counterpart, including the cell connections, and the occurrence of the basal laminal structures. In many cases arachnoidal cysts are incidental findings noted on CT or MRI of the head performed for a reason unrelated to the cyst. In about 15% of middle fossa arachnoid cysts, an asymptomatic lesion may become symptomatic as a result of bleeding in association of the cyst and raised intracranial pressure. This event may occur after minor head trauma. Treatment is by cystoperitoneal shunt after evacuation of the haematoma or with it at the same time\(^{51}\).

Cerebral convexity cysts occurring in adults, present as seizures, headache, raised ICP and sometimes marked reactive thickening of the overlying skull with the erosion of the inner table. These cases can be managed by the wide excision of the membranes and the establishment of communication between the cyst interior and the CSF of subarachnoid
Clinical features are those of SOL, but also other features may be seen for example Ross *et al* reported a case of periodic sweating associated with a subarachnoid cyst and multifocal dystonia

Ernst *et al* tried to answer the question which appearances on CT or MRI are indication for surgical treatment of intracranial arachnoid cysts? and they concluded that the only indication for surgery is the presence of obstructive hydrocephalus. The other features are not significant and had no influence on the decision to operate.

Buczex *et al* mentioned that arachnoid cysts of CNS are uncommon lesions of considerable interest and importance, the origin and exact nature of which remain uncertain (congenital, post-traumatic, infectious or other). They formed a well defined pathological entity and account for less than 1% of CNS space occupying lesions.

Aneurysms and arteriovenous malformations: -

**Aneurysm:**

Aneurysm is a widening of a vessel involving the stretching of fibrous tissue within the media of the vessel. These can be classified morphologically into saccular, fusiform or mycotic. The pathogenesis of saccular aneurysms reflects a combination of congenital, acquired and hereditary factors. Congenital defects in the muscle and elastic tissue of the arterial media seen at autopsy in 80% of normal vessels of circle of Willis, gradually deteriorate as they are exposed over time to the haemodynamic stress of pulsatile blood flow. These defects lead to microaneurysmal dilatations (< 2 mm) of the circle of Willis arteries in 15% of population. Larger than (> 5mm) aneurysms are found in 5% of population, characteristically distributed at the arterial bifurcations. A modest increase in incidence of familial saccular aneurysms as well as
their association with polycystic kidney disease, Ehler Danlos Syndrome and other connective tissue disorders implicate hereditary factors\(^{(10)}\).

*Fusiform aneurysms:* -

They acquired their name from the spindle shape dilatation and elongation that occur in large arteries at the site of arteriosclerotic narrowing. They develop most frequently in the basilar artery, but may also affect the internal, middle and anterior cerebral arteries of an individual with widespread arteriosclerosis and hypertension. Rarely they rupture and are difficult to treat, when they do because their shape and stiff walls preclude easy surgical clipping. Progressive dilatation and the tortuous elongation of the vessel cause neurologic dysfunction most frequently by compressing surrounding structures. Typically ectatic aneurysms of the basilar artery compress cranial nerves V, VII, VIII causing facial pain, hemifacial spasm and hearing loss with vertigo respectively. Fusiform aneurysms may imitate the features of CPA angle tumours, or they may mimic pituitary and suprasellar mass lesions.

*Mycotic Aneurysms:*-

These are caused by septic degeneration of arterial wall muscle and elastic tissue. They form in distal cerebral arteries at the point where small septic cardiogenic emboli lodge. They are frequently multiple and can be found in either the anterior or posterior cerebral circulation\(^{(10)}\).

Clinical features:-

A part from those related to subarachnoid haemorrhage due to rupture of aneurysm, it may present with features of space occupying lesion. Certain syndromes may result, for example compression of oculo-
motor nerve by an expanding aneurysm of the posterior communicating artery at its junction with either the internal carotid or the posterior cerebral artery, and less frequently, of the superior cerebellar artery can cause ipsilateral ophthalmoparesis, ptosis, and later pupillary dilatation with loss of pupillary light reflex. Orbital pain frequently, but not always, accompanies these signs. The clinical picture may resemble diabetic involvement of cranial nerve III, but the latter usually spares the pupil. Other compression syndromes from cerebral aneurysms include amnesia combined with varying degrees of the paresis of the third cranial nerve and quadriparesis from large strategically placed, basilar-tip aneurysm. Giant (>2.5 cm) aneurysms of internal carotid artery lying within the cavernous sinus can cause unilateral ophthalmoplegia and orbital pain by compressing cranial nerves III, IV, VI and the first division of the V.

Giant aneurysm of supraclinoid portion of the internal carotid artery can produce unilateral loss of vision or field defects through compression of the optic nerve or tracts\(^{(10)}\).

Bien et al mentioned that CT diagnosis of cerebral aneurysm is possible. In the nonthrombosed giant aneurysm, CT shows a homogenous, primarily hyperdense space occupying lesion with strong enhancement. The partially thrombosed giant aneurysms appear hyperdense with hypodense or isodense portion in the plain CT scan.

The completely thrombosed giant aneurysms were isodense to hypodense. CT scan diagnosis is possible in every case of partially or non thrombosed aneurysm. But cerebral angiography remains the definitive study to detect the lesions\(^{(55)}\).

The definitive therapy for a ruptured saccular aneurysm consists of surgical clipping of the aneurysm to prevent rebleeding. Medical therapy aims to reduce the risk of rebleeding and cerebral vasospasm and to
Sedation. Nimodipine orally 60 mg every 4 hours for 21 days, is required. Aneurysms should be clipped as soon as possible, but it may be worthwhile to avoid operation when maximal cerebral vasospasm is likely during days 3-10\(^{10}\). Other methods of treating aneurysms is coiling or glueing\(^{10}\).

Symon L mentioned that according to his surgical experience with giant intracranial aneurysms, over a period of 10 years, the most satisfactory method of handling the lesion is to remove the intraaneurysmal clot and clip the neck of the aneurysm\(^{56}\).

**Mycotic aneurysm:**

Unruptured mycotic aneurysms should be treated with antibiotics appropriate for infecting organism and followed angiographically. Single aneurysm and those in surgically accessible areas should be considered for prompt surgical clipping\(^{10}\).

**Vascular Malformation:**

Congenital vascular malformations of the brain and spinal cord fall into five categories according to vessel size and type.

V\(_e\)nous angiomas: The most common cerebrovascular malformation and usually lie close to the surface of the brain. They seldom produce seizures or headaches.

A cerebral varix is a single dilated vein and very rarely causes clinical symptoms.
haemorrhage from these small vessels can occasionally be fatal.

Cavernous angiomas: These are large sinusoidal channels served by large feeding arteries and veins. Many of these channels thrombose and the remainder have very low blood flow, which makes their visualization on angiograms difficult. They are readily detected by CT scan and rarely bleed, but they may cause headache and seizures.

Arteriovenous Malformation (AVM):-

The most common symptomatic vascular anomaly. The prevalence of AVM's among general population is uncertain, but autopsy studies of unselected patients indicate that 4 to 5% harbor some form of vascular malformation, of which only 10 to 15% produce symptoms\(^{(10)}\). Familial cases of AVM's are rare, indicating that the problem reflects sporadic abnormalities in embryologic development\(^{(10)}\).

Clinical features:-

Apart from manifesting with intracranial haemorrhage a lower portion causing seizures or progressive neurologic disability as first symptoms. It may be responsible in the brain and its covering for about 5-9% of all intracranial space occupying lesions and 20-40% of the sources of surgically treated intracranial haemorrhages\(^{(57)}\). Diagnosis is by CT scan and MRI. Angiography remains the definitive test to identify the AVM and delineate its feeding arteries and draining veins.

Medical treatment is indicated for unruptured AVM's and it includes, treatment of headache, anticonvulsants for control of seizures and control of hypertension, avoidance of antiplatelet and anticoagulants.
Interventional therapeutic options include surgical resection of the AVM embolization of the feeding arteries or radiation induced thrombosis\textsuperscript{(10)}.

**Cerebral Vasculitis:**

Scolding \textit{et al} studied 8 patients with cerebral vasculitis and found three clinical patterns:

1. Acute or subacute encephalopathy.
2. A picture with some similarity to multiple sclerosis.
3. Features of rapidly progressing space occupying lesion\textsuperscript{(58)}.

**Sarcoidosis:**

Sarcoidosis, a multisystem granulomatous disease, begins most frequently in people between 20 and 40 years old. The cause is unknown, but alterations in the immune system are clearly involved in the pathogenesis. Organ involvement is usually asymptomatic and the disease most frequently regress spontaneously, but it may progress to a more chronic state of fibrosis with severe functional impairment of various organs. Neurological involvement of CNS according to some sources occurs in 2-5\% of patients\textsuperscript{(36,59)}.

In addition to cranial nerve involvement, mononeuropathy and polyneuropathy, meningitis, diabetes insipidus from hypothalamic involvement and personality change have been observed.

In addition to all these neurological manifestation, granulomas of the brain may produce space occupying lesion and cause headaches, seizures or focal symptoms. Definitive diagnosis can be made by biopsy. However, diagnosis by biopsy sometimes becomes problematic for neurologic disease caused by sarcoidosis when other tissues do not
provide a positive diagnosis, since the involved tissue is often not easily accessible. However, Morgunov et al reported a case with CT scan evidence of space occupying lesion, but granuloma typical of sarcoidosis was found on stereotactic biopsy of the parietal brain region\textsuperscript{(60)}. Transbronchial biopsy provides positive histological evidence of a granuloma of patients with clinically extrapulmonary sarcoidosis in whom the chest x-ray is normal\textsuperscript{(36)}.

- Neuroimaging - CT and MRI may help\textsuperscript{(36)}.
- Other tests Keveim test, angiotensin-converting enzyme, tuberculin test and gallium scanning may also help in the diagnosis.

In regard to treatment it has been assumed that central nervous system involvement is particularly chronic and poorly responsive to steroid therapy\textsuperscript{(36)}.

**Gumma:**

Late benign syphilis or gumma was the most common complication of late syphilis in OSLO study of untreated patients (1891-1951) \textsuperscript{(3)}. In the penicillin era gumma is rare. They typically develop from one year to ten years after the initial infection and may involve any part of the body, although they may be very destructive they respond to treatment and therefore are relatively benign. Histologically the gumma is a granuloma. The histologic findings are non specific and may be associated with central necrosis, surrounded by epithelioid and fibroblastic cells and associated giant cells, as well as vasculitis. In some cases T.pallidium is ordinarily not demonstrable by silver stains, but can sometimes be recovered by inoculation of rabbits. Roda et al mentioned that cerebral gumma is exceptionally rare at present time \textsuperscript{(61)}. Gummas are expression of localized meningiovascular forms of neurosyphilis and their clinical symptoms and
signs are similar to those of any other space occupying intracranial lesion. Computed tomography scans show a ring enhancing lesion with intense cerebral oedema. They stated that surgical excision, whenever possible, is the treatment of choice since it achieves the removal of the mass and its histological verification\(^{(3)}\).

**Tuberculoma:**

It has been agreed that tuberculoma is rare in developed countries \(^{(8,62)}\). The incidence of tuberculoma, showed great variation and results of studies are quite different. For example, Gropper et al mentioned that it accounts for 5-30% of all intracranial masses\(^{(63)}\). Liu et al stated that it accounts for 0.90% of intracranial tumours\(^{(64)}\) and Irfan et al showed that it constituted 5.5% of all SOLs\(^{(4)}\). Bouchama et al mentioned that in developing countries 5-8% of space occupying lesions of central nervous system are tuberculomas\(^{(65)}\). Lana-peixoto et al mentioned that two thirds of the patients were younger than 20 years\(^{(66)}\). Boucetta et al mentioned that most of the patients were young\(^{(67)}\). Abdul-Ghaffar mentioned that 97% of patients were males\(^{(68)}\).

**Clinical Features:**

Usually are those of space occupying lesion. The patient may have history of tuberculosis, or may have no evidence of current extracranial tuberculosis\(^{(63,64)}\).

Boucetta _et al_ \(^{(67)}\) showed that intracranial hypertension syndrome is predominant in 75% of patients \(^{(67)}\). The supratentorial localization predominated in 70% of the cases and 16% of lesions are multiple \(^{(67)}\).
An exceptionally rare intrasellar tuberculoma in a patient with clinical evidence of hypopituitarism was reported by Ranjan et al\(^{(69)}\). Gropper mentioned that tuberculoma of the brain stem accounts for 2.5-8% of all intracranial tuberculomas\(^{(61)}\). An exceptionally rare case of tuberculoma of the cavernous sinus was reported by Grayeli\(^{(70)}\). An unusual case of hypothalamic tuberculoma in a man who presented with symptoms of hypopituitarism was reported by Flannery et al, who mentioned that the incidence of tuberculoma accounts for 15-50% of neurosurgical cases in developing countries\(^{(71)}\).

Surprisingly enough that intracranial tuberculomas can sometimes develop or increase in size during therapy. Afghani et al mentioned that 23 cases of tuberculoma that increased in size and other 17 cases that appeared after treatment was reported in literature\(^{(72)}\). It has been assumed that this may be due to interaction between hosts immune response and the direct effect of mycobacterial products and it does not represent failure of antituberculous therapy\(^{(72)}\). A similar case was reported by Wong et al\(^{(73)}\). Another group of investigators reported similar case\(^{(65)}\). Occasionally tuberculoma may occur in conjunction with tuberculous meningitis\(^{(74)}\).

Investigations:

ESR and chest x-ray and mantoux test often fail to confirm the diagnosis\(^{(8)}\). Although the sensitivity of CT in the diagnosis of intracranial tuberculoma is 100% and its specificity is 85.7% the positive value is only 33% (confident limit 24-42%)\(^{(75)}\). The negative predictive value is 100%. The low positive predictive value for a diagnosis of intracranial
tuberculoma on CT scan alone indicates the need for a confirming histological diagnosis\(^{(75)}\).

Consugglus \textit{et al} mentioned that CT scanning with contrast enhancement is very useful. He claimed that the presence of a target lesion is considered to be pathognomonic for tuberculoma, however, he recommended obtaining a definitive histological diagnosis with CT guided stereotactic surgery prior to commencing antituberculous treatment\(^{(76)}\). It was agreed that CT scan clearly demonstrates an enhancing lesion, but this often resembles astrocytoma or metastasis. Tuberculoma has no distinguishing features\(^{(62,64)}\).

Kioumeher \textit{et al} mentioned that in regard to MRI all intraparenchymal tuberculomas showed characteristic T2 shortening not found in most other space occupying lesions\(^{(77)}\). In the appropriate clinical setting tuberculoma should be considered\(^{(77)}\).

Inoue \textit{et al} mentioned that MRI may reveal three concentric layers within the tumour and the histological appearance corresponded well to MRI findings\(^{(78)}\). The central core was caseous necrosis and the middle layer contained langhans' giant cells and epithelioid cells with substantial oedema. The outer layer was the capsule that consisted of collagen fibres\(^{(78)}\).

Bouchoma \textit{et al} stated that brain biopsy although not free of risk may be of great help in establishing a diagnosis of tuberculoma especially when no evidence of intracranial tuberculois is present and because CT scan is only suggestive\(^{(65)}\). White \textit{et al} mentioned that diagnosis is often made at surgery\(^{(79)}\).
Treatment:

With antituberculous drugs especially short course chemotherapy almost all patients with intracranial tuberculoma can be cured and only few patients need surgical operation\(^{(80)}\).

Awada et al mentioned that all tuberculoma disappeared on CT scan after 12 months of therapy\(^{(81)}\). Most of the oedema images disappeared by a month and they suggested that a long treatment regimen of 15-18 months may not be necessary in most intracranial tuberculomas occurring in nonimmunocompromised patients. They stated that medical trial in suspected cases should last for at least 2 months before considering other aetiologies and surgical exploration. It is agreed that the response to treatment is encouraging and very good and operative indications for intracranial tuberculoma should be applied strictly\(^{(81)}\). Surgical excision is necessary in-patients with raised intracranial pressure secondary to the lesion not responding to medical therapy.

Mycetoma:

Mycetoma is a chronic, localized subcutaneous infection characterized by draining sinus tracts, that frequently discharge purulent material containing granule\(^{(10)}\). The disease most often affects the lower extremities with the majority of cases involving the foot. The organisms are usually introduced by a thorne. Rare sites such as eye lids, testes, mandible, paranasal sinus, head and neck and the spine has also been described. Neurologic deficits are quite rare and less well described\(^{(10)}\).

In the study conducted by Arbab et al, they mentioned that the commonest causative organism was streptomyces somaliensis (66.7%). Males were affected more often than females (22.2%) and the sources of
infection was unknown in the majority of cases and only known in (33.3%) of cases. The most common mode of presentation was headache and scalp swelling (88.9%) the next common presentation was epilepsy (55.6%). Other focal neurological disorders such as hemiplegia and cranial nerve affection were also found. CT findings of the cranium showed osteosclerotic rather than osteolytic changes. They concluded that mycetoma of the cranium may present with various neurological disorders and may simulate cerebral neoplasm or psudotumour cerebri.
CHAPTER TWO
OBJECTIVES

1. To determine types of intracranial space occupying lesions.
2. To study clinical presentation of intracranial space occupying lesions.
3. To study CT scan changes in different SOLs.
CHAPTER THREE
One hundred and eighteen patients with different types of SOLs were included in this study.

1-b: Place:

The patients seen in this study were those admitted in El Shaab Teaching Hospital in both neurology and neurosurgery wards and Khartoum and Ibn Khaldoun Hospitals or seen in neurosurgical referred clinic in El Shaab Teaching Hospital.

1-c: Duration of the study:

This study was done in the period between August 1997 and October 1998.

2- Definition:

The term intracranial SOL is generally used to identify any lesion whether vascular or neoplastic or inflammatory in origin, which increases the volume of the intracranial content and thus leads to rise in the intracranial pressure (ICP).

3- Inclusion Criteria:

All patients are adult Sudanese patients.
All patients had CT scan proved SOLs.

Exclusion criteria:

- Patients with intracranial haemorrhage and subdural haematoma were not included.
- Children were not included in this study.
4- Material and methods:

- A questionnaire was used. It consisted of the following:

a. Personal data: name, age, sex, nationality, tribe, residence and occupation.

b. Symptoms:
   - These include fever, symptoms of raised intracranial pressure, headache, vomiting, blurring of vision, epilepsy, mental symptoms such as coma, confusion and others such as dementia or impaired memory.

c. Also sensory symptoms: such as paraesthesia or sensory loss.

d. Motor symptoms:
   - Weakness, unsteadiness, stiffness and abnormal movements 'tremor'.

e. Bulbar symptoms and symptoms referred to cranial nerves:
   - Dysphagia, nasal regurgitation, visual loss, diplopia, squint, ptosis and deafness.

f. Other focal symptoms such as: aphasia, apraxia, behavioral changes and incontinence.

g. Past medical history of chronic cough, trauma to the head, schistosomiasis and epilepsy.

h. Family history of similar condition.

- Clinical examination:

This included thorough physical examination and detailed neurological examination. The neurological examination consisted of assessment of higher functions, cranial nerves and fundi, examination of the neck, motor and sensory systems, examination of the skull and back and the gait.

All patients seen were either inpatients or regular attendants of neurosurgical referred clinic in El Shaab Teaching Hospital. The questionnaire was filled by the author.
5- Investigations:

a) Routine investigations:
   Haemoglobin, white blood count, ESR, urea, electrolytes and urine analysis.

b) Specific investigations:
   Mantoux test, sputum for AAFB, chest x-ray, serology for evidence of mycetoma and pituitary hormones were done in selected patients when indicated.

c) Investigations essential for diagnosis of intracranial SOL:
   - All patients had brain CT scan.

d) Other investigations:
   Biopsy - histological confirmation was obtained in those who had access to the surgery.

6. Statistics:
   - The statistical significance was tested using the Chi-Square test.
RESULTS

118 patients were included in this study.

Age and sex distribution:-

Figure 1 shows age distribution. Thirty five patients (29.66%) were in the age group 18-30, 38 patients (32.2%) in the age group >30-42, 17 patients (14.4%) between >42-54, 18 patients (15.25%) were between >54-66 and 10 patients (8.47%) were above the age of 66. Sixty seven patients (56.8%) were males while 51 patients (43.2%) were females (figures 2 and 3).

Clinical presentation:-

Headache was present in 102 patients (86.4%), epilepsy in 55 patients (46.6%), visual deterioration in 67 patients (56.8%), mental symptoms in 31 patients (26.3%), fever in 44 patients (37.2%), vomiting in 45 patients (38%) and cranial nerve involvement was found in 38 patients (31.9%). They were difficult to be assessed in 8 patients (16.77%). Seventh cranial nerve was affected in 18 patients (15.25%), sixth cranial nerve in 8 patients (8.77%) and more than one nerve in 9 patients (7.5%) (table 1 and 2). Table 3 shows fundal changes.

Papilloedema was detected in 37 patients (31.3%), optic atrophy in 14 patients (11.86%). Hemiparesis or hemiplegia in 34 patients (28.8%). Cerebellar ataxia was found in 15 patients (12.7%). Scalp swelling was found in 6 patients (5.08%).

All patients had cranial CT scans. Sites of the lesions were as follows:-

In the tempoparietral region in 20 patients (16.9%). In the cerebellum and cerebellopontine angle in 18 patients (51.1%), in the sellar and/or suprasellar region in 11 patients (9.32%), in parietal in 17 patients (14.3%), frontal in 11 patients (9.32%), frontoparietal in 12 patients
In temporal region in 8 patients (6.77%), in parasagittal region in 6 patients (5.08%), in occipital in 5 patients (4.2%). In the convexity in 4 patients (3.38%), in the region of pineal body, pons and parietooccipital in 2 patients for each site (1.69%) and in temprooccipital and in the region of the posterior communicating artery in 1 patients (0.84%) for each site.

Histopathological confirmation was obtained in 30 patients 25.3% (table 5). Types of the SOLs (figure 6).

- Meningiomas in 34 patients 28.8%
- Gliomas in 31 patients 26.3%
- Abscess in 12 patients 10.2%
- Tuberculoma in 10 patients 8.5%
- Adenoma in 8 patients 6.8%

In 23 patients (19.5%) other lesions were detected (table 4). They include:

- Hydatid disease in 3 patients 2.5%
- Metastases to the brain in 3 patients 2.5%
- Arachnoid cyst in 3 patients 2.5%
- Cerebellar cyst in 2 patients 1.69%
- Mycetoma in 2 patients 1.69%
- Haemangioblastoma in 2 patients 1.69%
- Pineocytoma in 1 patient 0.84%
- Cholesteatoma in 1 patient 0.84%
- Acoustic neuroma in 1 patient 0.84%
- Craniopharyngioma in 2 patient 1.69%
- Dermoid cyst in 1 patient 0.84%
- Malignant sarcoma in 1 patient 0.84%
- Aneurysm of posterior communicating artery in 1 patients 0.84%
Menengiomas:

Menengiomas were found in 34 Patients.

Age and sex distribution:

Figure 8 shows age distribution. Four patients (11.7%) were in the age group 18-30, 13 patients (38.2%) were in the age group >30-42, 5 patients (14.7%) were between >42-54, 10 patients (29.4%) were between >54-66 and 2 patients (15.88%) were above the age of 66. No patient was below the age of 20. Nineteen patients (55.9%) were females and 15 patients (44.1%) were females (figure 3).

Clinical presentation (figure 9):

Headache was present in 29 patients (85.3%), epilepsy in 18 patients (52.9%), visual deterioration in 24 patients (70.5%), mental symptoms in 10 patients (29.4%), fever in 2 patients (5.9%), vomiting in 12 patients (35.3%). History of trauma was present in 16 patients (47.06%), cranial nerve involvement in 10 patients (29.4%), [seventh cranial nerve was affected in 4 patients (11.76%), sixth cranial nerve in 3 patients (8.7%), other cranial nerve in 3 patients (8.7%) and more than one cranial nerve in 4 patients (11.7%)]. Papilloedema was detected in 9 patients (26.5%), optic atrophy in 5 patients (14.7%). It was difficult to be examined in 2 patients (5.9%). Hemiplegia or hemiparesis was found in 8 patients (23.5%). Cerebellar ataxia in 2 patients (5.9%) and scalp swelling in 3 patients (8.8%) (see the photo).

CT findings (figure 10):

The meningiomas were located in the following sites, in CPA in 2 patients (5.9%), in parasellar in 3 patients (8.8%), frontal in 4 patients (11.7%), in parietal in 5 patients (14.4%), temproparietal in 4 patients (11.7%), occipital in 3 patients (8.8%) and pontine in 1 patient (2.9%). In the convexity in 4 patients (11.7%), in frontoparietal in 3 patients (9.7%)
and multiple meningiomas were detected in 1 patient (2.9%). The precontrast scan revealed hyperdense lesions in 31 patients (91.2%) and isodense lesions in 3 patients (8.8%) (table 6). The contrast scan showed enhancement in 34 patients (100%). Shifting of midline structures was found in 9 patients (26.5%) and calcification in 3 patients (8.8%). Histological confirmation was obtained in 13 patients (41.9%) (figure III shows meningioma).

**Gliomas:**

Gliomas were found in 31 patients.

**Age and sex distribution:**

Eight patients (25.8%) were in the age group 18-30, 7 patients (22.6%) were between >30-42, 5 patients (16.1%) between >42-54, 5 patients (16.1%) were in the age group >54-66 and 6 patients (19.35%) were above the age of 66. Nineteen of patients (61.3%) were males and 12 patients (38.7%) were females (figure 3).

**Clinical presentation (figure 12):**

Headache was present in 26 patients (83.3%), epilepsy in 16 patients (51.6%), mental symptoms in 14 patients (45.2%), visual deterioration in 22 patients (70.9%), fever in 14 patients (45.7%) and vomiting in 10 patients (32.25%). Cranial involvement was seen in 13 patients (41.9%), [seventh cranial nerve in 7 patients (22.6%), sixth in 2 patients (6.45%), other cranial nerves in 4 patients (12.9%) and affection of more than one nerve in 2 patients (table 1 and 2)]. Papilloedema was detected in 10 patients (32.25%), optic atrophy in 1 patient (3.2%) and it was difficult to examine in 2 patients (6.45%) (table 3). Cerebellar ataxia was detected in 4 patients (12.9%) and hemiparesis or hemiplegia in 12 patients (38.7%).
CT findings (figure 13):

The gliomas were found in the following sites; in the cerebellum in 6 patients (17.6%), frontoparietal in 1 patient (3.2%), temproparietal in 9 patients (29%), frontal in 3 patients (9.7%), parietal in 3 patients (9.7%), occipital in 2 patients (6.5%), temporal in 5 patients (16%), parietooccipital in 1 patient (3.2%) and in the brain stem in 1 patient (3.2%). The precontrast scan showed hypodense lesions in 8 patients (25.8%), hyperdense lesions in 16 patients (51.52%), isodense lesions in 2 patients (6.45%) and mixed attenuation in 5 patients (16%) (table 6). The contrast scan showed enhancement in 18 patients (58%), oedema in 21 patients (67.7%), shifting of midline structures in 17 patients (45.8%).

Histopathology:-

The diagnosis was verified histopathologically in 6 patients (19.35%) (table 5).

Types of gliomas (figure 14):

- Astrocytoma in 18 patients 58.1%
- Glioblastoma in 4 patients 12.8%
- Cystic glioma in 2 patients 6.4%
- Ependymoma in 2 patients 6.4%
- Oligodendroglioma in 1 patient 3.2%
- and type of glioma was not known in 4 patients 12.8%

Abscesses:-

Abscesses were found in 12 patients.

Age and sex distribution:-

Figure 15 shows age distribution. Eight patients (66.6%) were in the age group 18-30 years. One patient (8.3%) was in the age group >30-42, 2 patients (16.6%) were between >42-54 and one patient (8.3%) in the
age group >54-66. Ten of the patients (83.3%) were males and 2 (16.6%) patients were females (figure 3).

**Clinical presentation (figure 16):**

Headache was present in 11 patients (91.6%), epilepsy in 6 patients (50%), 4 patients (33.3%) had deterioration of vision, mental symptoms in one patient (8.3%), fever in 11 patients (91.6%), vomiting in 5 patients (41.6%), cranial nerve involvement in 6 patients (50%), [seventh cranial nerve in 3 patients (25%), sixth nerve in 2 patients (8.3%) and in one patient (16.6%) there was affection of more than one nerve (table 1 and 2)]. Papilloedema in 4 patients (33.3%) and optic atrophy was not detected (table 3). Hemiparesis or hemiplegia in 4 patients (33.3%) and cerebellar ataxia in 3 patients (25%).

**CT findings (figure 17):**

The lesions were found in the following sites; in the cerebellum in 3 patients (25%), temproparietal in 2 patients (16.6%), temporal in 2 patients (16.6%), parietal in 3, patients (25%), frontoparietal in one patient (8.3%) and in parieto-occipital in one patient (8.3%). Multiple lesions were detected in 2 patients (16.6%). The non contrast CT scan shows hypodense lesions in 12 patients (100%). The contrast CT scan showed ring enhancement in all 12 patients (100%). Oedema was shown in 12 patients (table 6). Figure I shows brain abscess.

**Other investigations:**

Total white blood cells count in 9 patients (75%) was between 6000-9500 and in 3 patients (25%) it was less than 6000. The mean value of TWBC was 6800±1844 SD.
Tuberculoma:-

Tuberculomas were found in 10 patients.

Age and sex distribution:

Figure 18 shows age distribution. Five patients (50%) were in the age group 18-30 and 5 patients (50%) were between >30-42. Six patients (60%) were males and 4 patients (40%) were females (figure 3).

Clinical presentation (figure 19):-

Headache was present in 9 patients (90%), epilepsy in 4 patients (40%), visual deterioration in 2 patients (20%), fever in 4 patients (40%), vomiting in 3 patients (30%) and mental symptoms in 2 patients (20%). Cranial nerve involvement in 1 patient (10%) (table 2), papilloedema in 2 patients (20%) (table 3) and hemiparesis or hemiplegia in 4 patients (40%).

CT findings (figure 20):-

The lesions were in the following sites: in parietal in 5 patients (50%), tempoparietal 2 patients (20%), frontoparietal 2 patients (20%) and parasagittal in one patient (10%). The noncontrast CT scan showed isodense lesion in 4 patients (40%), hypodense in 5 patients (50%) and hyperdense in 1 patient (10%) (table 6). The contrast scan revealed enhancement in 10 patients (100%), oedema was found in 6 patients (60%), shifting of the midline structure in 1 patient (10%).

Histopathology:-

Histopathological confirmation was done in 3 patients (30%).
Other investigations:-

- Mantoux test was positive in 6 patients (60%) (mantoux test was considered to be positive if it is more than 10mm). It was negative in 3 patients (30%) and not done in 2 patients (20%).
- Sputum for AAFB was negative in 8 patients (80%) and not done in 2 patients (20%).
- ESR was ranging between 35-120 mm/hr. The mean ESR was 75.8±27 SD. 6 patients (60%) had ESR of 80mm/hr and above, one patient (10%) had ESR of 60mm/hr and 3 patients (30%) had ESR below 60mm/hr.
- Chest x-ray was clear in 4 patients (40%) and not done in 6 patients (60%).

Pituitary adenoma:–

Pituitary adenomas were found in 8 patients.

Age and sex distribution:–

Figure 21 shows age distribution. One patient (12.5%) in the age group 18-30, 4 patients (50%) were between >30-42 and 3 patients (7.5%) were between >42-54. 4 patients (50%) were males and 4 patients (50%) were females (figure 3).

Clinical presentation:–

Figure 22 shows main clinical presentation. Headache was present in 8 patients (100%), visual deterioration in 8 patients (100%) and mental symptoms in one patient (12.5%). Acromegalic physique was found in 3 patients (37.5%) and galactorrhoea in 2 patients (25%). Visual field defects in 3 patients (37.5%), papilloedema was found in 1 patient (12.5%) and optic atrophy in 5 patients (62.5%).
CT findings:-

The non contrast CT scan showed hyperdense lesion in 6 patients (75%), hypodense in one patient (12.5%) and isodense in 1 patient (12.5%) (table 6). The contrast scan showed enhancement in 6 patients (75%). Calcification was seen in one patient (12.5%), oedema and shifting of midline structures was not found. Suprasellar extension was found in 2 patients (25%), erosion of clinoid processes was detected in 5 patients (62.5%).

Other investigations:-

Anterior pituitary hormones were done in 6 patients (75%). Prolactin was found to be high in 4 patients (50%) and normal in 2 patients (25%). Other pituitary hormones were normal in 5 patients (62.5%), FSH and LH were low in one patient (12.5%).

Histopathology:-

In 3 patients (37.5%) the lesion was verified histopathologically.

Other space occupying lesions:-

Other space occupying lesions were found in 23 patients.

Age and sex distribution:

Figure 23 shows age distribution. Nine patients (39%) in the age group 18-30, 8 patients (34.8%) were between >30-42, 2 patients (8.7%) were between >42-54 and 2 patients (8.7%) were above the age of 66. Thirteen of the patients (56.5%) were males and 10 patients (43.5%) were females (figure 3).

Clinical presentation:-

Figure 24 shows main clinical presentation. Headache was present in 18 patients (78.3%), epilepsy in 11 patients (47.8%), visual deterioration in 7 patients (30.4%) and mental symptoms in 3 patients (13%). Fever in 10 patients (43.5%) and vomiting in 13 patients (56.5%).
affection of more than one cranial nerve in 2 patients (8.6%) (table 2)]. Papilloedema in 11 patients (47.7%), optic atrophy in 3 patients (13%) and it was difficult to examine the fundi in 3 patients (13%). Hemiparesis or hemiplegia in 6 patients (26%) and cerebellar ataxia in 6 patients (26%).

CT scan findings:

Figure 25 shows sites of the lesions. In the cerebellum in 4 patients (17.4%), frontoparietal in 5 patients (21.7%), tempoparietal 3 patients (13%), frontal in 4 patients (17.4%), pineal in 2 patients (8.7%), in CPA in 3 patients (13%), temporal in 1 patient (4.2%) and parietal in 1 patient (4.2%).

Metastases:

Three patients had cerebral metastases accounting for (2.5%) of all SOLs and for (3.3%) of all brain tumours. Two patients (66.3%) were males and one patient (33.3%) was a female. One patient (33.3%) was 48 years old, 1 patient (33.3%) was 63 years old and 1 patient (33.3%) was 70 years old. In 2 patients (66.6%) the primary was identified histopathologically, 1 patient (33.3%) had breast carcinoma, 1 patient (33.3%) had bronchial carcinoma and in 1 patient (33.3%) it was diagnosed on clinical and radiological base. Multiple lesions on CT scan were detected in 2 patients (66.6%).

Arachnoid cysts:

Arachnoid cysts were found in 3 patients (2.5%) of all SOLs. The age of the patients was as follows. One patient (33.3%) was 30 years, 1 patient (33.3%) was 35 years old and 1 patient (33.3%) was 40 years old. Two patients (66.6%) were males and 1 patient (33.3%) was a female.
Two patients had cranial mycetoma. This accounts for 1.69% of all SOLs. All patients (100%) were of the age 35. Two patients (100%) had scalp swelling and 2 patients (100%) had positive serology for streptomyces somaliensis.
Fig (1) Age distribution
Fig (2) Sex distribution

Females
43.2

Males
56.8
Fig (3) Sex distribution of S.O.Ls
Fig (4) Main clinical presentation

- Headache
- Visual deterioration
- Epilepsy
- Vomiting
- Fever
- Cranial neuropathy
- Fundal changes
- Motor weakness
- Mental symptoms
- Cerebellar ataxia

Percentage: 86.4, 56.8, 46.6, 38, 37.2, 31.3, 31.3, 28.8, 26.3, 12.7
Fig (5) location of lesions

- Tempro parietal 16.9
- Cereb. & cereb. pon 18.1
- Parietal 14.3
- Fronto-parietal 10.0
- Sellar & para sellar 9.3
- Frontal 9.3
- Temporal 6.7
- Parasagittal 5.1
- Occipital 4.2
- Convexity 3.4
- Tempo-occipital 1.8
- Pineal 1.7
- Pons 1.7
- Parietoccipital 1.7
- Tempro occ. 0.84
- PCA 0.84
Fig (6) Types of S.O.Ls

- Meningiomas: 28.8%
- Gliomas: 26.3%
- Abscesses: 10.2%
- Tuberculomas: 8.5%
- Adenomas: 6.8%
- Other lesions: 19.5%
Fig. (7) The nature of the lesions
Figure 8: Age distribution in meningiomas.
Fig (9) Main clinical presentation in meningiomas

- Headache: 85.3%
- Visual deterioration: 70.5%
- Epilepsy: 52.9%
- Mental symptoms: 36.3%
- Vomiting: 36.3%
- Cranial neuropathy: 29.4%
- Fundal changes: 26.5%
- Motor weakness: 23.5%
- Fever: 5.9%
- Cerebellar ataxia: 5.9%
Fig (10) location of meningiomas

- Parietal: 14.4%
- Parasagittal: 14.4%
- Temporal-parietal: 11.7%
- Frontal: 11.7%
- Convexity: 11.7%
- Occipital: 8.8%
- Parasellar: 8.8%
- Frontoparietal: 8.8%
- C.P.A.: 5.9%
- Pontine: 2.9%

%
Fig (12) Main clinical presentation in gliomas

- Headache: 85.3%
- Visual deterioration: 70.9%
- Epilepsy: 61.6%
- Fever: 45.7%
- Mental symptoms: 44.2%
- Cranial neuropathy: 41.9%
- Motor weakness: 38.7%
- Vomiting: 32.3%
- Fundal changes: 32.3%
- Cerebellar ataxia: 12.9%
Fig (13) location of gliomas

- Temporo parietal: 29%
- Cereb. & cereb.pon: 17.6%
- Temporal: 16%
- Frontal: 9.7%
- Occipital: 4.2%
- Fronto parietal: 3.2%
Fig (14) Types of glioma

- Astrocytoma
- Glioblastome
- Cystic glioma
- Ependymoma
- Oligodendroglioma
- Type not known

No
Fig (15) Age distribution in abscesses

Age groups in years

18-30
>30-42
>42-54
>54-66
>66

%
Fig (16) Main clinical presentation in abscesses

- Headache: 91.6%
- Fever: 91.6%
- Epilepsy: 60%
- Cranial neuropathy: 60%
- Vomiting: 41.6%
- Visual deterioration: 33.3%
- Fundal changes: 33.3%
- Motor weakness: 33.3%
- Cerebellar ataxia: 25%
- Mental symptoms: 8.3%
Fig (17) location of abscesses

- Cerebellum: 25%
- Parietal: 25%
- Temporal: 16.6%
- Temporo parietal: 16.6%
- Occipital: 8.3%
- Fronto parietal: 8.3%
Fig(18) Age distribution in tuberculomas
Fig (19) Main clinical presentation in tuberculomas

- Headache
- Fever
- Epilepsy
- Motor weakness
- Vomiting
- Visual deterioration
- Fundal changes
- Mental symptoms
- Cranial neuropathy
Fig (20) location of tuberculomas
Fig (21) Age distribution in pituitary adenomas
Fig (22) Main clinical presentation in pituitary adenomas

- Headache
- Fundal changes
- Acromegalic physique
- Visual field defect
- Galactorrhea
- Mental symptoms
Fig (23) Age distribution of other S.O.Ls
Fig (24) Main clinical presentation in other S.O.Ls

- Headache
- Fundal changes
- Vomiting
- Epilepsy
- Visual deterioration
- Cranial neuropathy
- Motor weakness
- Cerebellar ataxia
- Mental symptoms

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Headache</td>
<td>78.3%</td>
</tr>
<tr>
<td>Fundal changes</td>
<td>60.8%</td>
</tr>
<tr>
<td>Vomiting</td>
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</tr>
<tr>
<td>Epilepsy</td>
<td>47.8%</td>
</tr>
<tr>
<td>Visual deterioration</td>
<td>30.4%</td>
</tr>
<tr>
<td>Cranial neuropathy</td>
<td>21.7%</td>
</tr>
<tr>
<td>Motor weakness</td>
<td>26%</td>
</tr>
<tr>
<td>Cerebellar ataxia</td>
<td>26%</td>
</tr>
<tr>
<td>Mental symptoms</td>
<td>13.1%</td>
</tr>
</tbody>
</table>
Fig (25) location of other S.O.Ls

- Fronto parietal: 21.7%
- Cerebellum: 17.4%
- Frontal: 17.4%
- Temporo parietal: 13%
- CPA: 13%
- Pineal: 8.7%
- Temporal: 4.2%
Table 1
Cranial nerves involvement in SOLs

<table>
<thead>
<tr>
<th>Cranial nerve</th>
<th>Frequency</th>
<th>Percentage</th>
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<tr>
<td>Abnormality</td>
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<tr>
<td>Normal</td>
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<td>Difficult</td>
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<td>6.72%</td>
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### Table 2

<table>
<thead>
<tr>
<th>Cranial nerves involvement in SOLs</th>
<th>Meningioma</th>
<th>Glioma</th>
<th>Abscess</th>
<th>Tuberculoma</th>
<th>Adenoma</th>
<th>Other SOLs</th>
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<tr>
<td>9th</td>
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<td></td>
</tr>
<tr>
<td>10th</td>
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<td></td>
</tr>
<tr>
<td>11th</td>
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</tr>
<tr>
<td>12th</td>
<td>1</td>
<td></td>
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</table>
## Table 3

**Fundal changes in SOLs**

<table>
<thead>
<tr>
<th>The changes</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
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<tbody>
<tr>
<td>Papilloedema</td>
<td>37</td>
<td>31.3%</td>
</tr>
<tr>
<td>Optic atrophy</td>
<td>14</td>
<td>11.86%</td>
</tr>
<tr>
<td>Difficult to assess</td>
<td>8</td>
<td>6.77%</td>
</tr>
<tr>
<td>Normal</td>
<td>59</td>
<td>50%</td>
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<tr>
<td>Total</td>
<td>118</td>
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Table 4

Other space occupying lesions

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<thead>
<tr>
<th>SOL</th>
<th>No. of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Cerebral metastases</td>
<td>3</td>
<td>2.5%</td>
</tr>
<tr>
<td>2 Arachnoid cyst</td>
<td>3</td>
<td>2.5%</td>
</tr>
<tr>
<td>3 Hydatid disease</td>
<td>3</td>
<td>2.5%</td>
</tr>
<tr>
<td>4 Cerebellar cyst</td>
<td>2</td>
<td>1.69%</td>
</tr>
<tr>
<td>5 Mycetoma</td>
<td>2</td>
<td>1.69%</td>
</tr>
<tr>
<td>6 Haemangioblastoma</td>
<td>2</td>
<td>1.69%</td>
</tr>
<tr>
<td>7 Pineocytoma</td>
<td>1</td>
<td>0.84%</td>
</tr>
<tr>
<td>8 Craniopharyngioma</td>
<td>2</td>
<td>1.69%</td>
</tr>
<tr>
<td>9 Acoustic neuroma</td>
<td>1</td>
<td>0.84%</td>
</tr>
<tr>
<td>10 Dermiod cyst</td>
<td>1</td>
<td>0.84%</td>
</tr>
<tr>
<td>11 Cholesteatoma</td>
<td>1</td>
<td>0.84%</td>
</tr>
<tr>
<td>12 Malignant sarcoma</td>
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<td>0.84%</td>
</tr>
<tr>
<td>13 Aneurysm of PCA</td>
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### Histopathology in SOLs

<table>
<thead>
<tr>
<th>SOL</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
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<tbody>
<tr>
<td>Meningioma</td>
<td>13</td>
<td>11.01%</td>
</tr>
<tr>
<td>Glioma</td>
<td>6</td>
<td>5.08%</td>
</tr>
<tr>
<td>Tuberculoma</td>
<td>3</td>
<td>2.5%</td>
</tr>
<tr>
<td>Adenoma</td>
<td>3</td>
<td>2.5%</td>
</tr>
<tr>
<td>Hydatid disease</td>
<td>2</td>
<td>1.69%</td>
</tr>
<tr>
<td>Craniopharyngioma</td>
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<td>1.69%</td>
</tr>
<tr>
<td>Cholesteatoma</td>
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<td>0.84%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
<td><strong>25.3%</strong></td>
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Table 6

Precontrast and contrast findings in SOLs

<table>
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<tr>
<th>Tissue Type</th>
<th>Hyperdense %</th>
<th>Isodense %</th>
<th>Hypodense %</th>
<th>Mixed %</th>
<th>Contrast Enhancement</th>
</tr>
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<tbody>
<tr>
<td>Renalingioma</td>
<td>91.2%</td>
<td>8.8%</td>
<td>-</td>
<td>-</td>
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</tr>
<tr>
<td>Tcoma</td>
<td>51.52%</td>
<td>6.45%</td>
<td>25.8%</td>
<td>16%</td>
<td>16%</td>
</tr>
<tr>
<td>Abscess</td>
<td>-</td>
<td>-</td>
<td>100%</td>
<td>-</td>
<td>100%</td>
</tr>
<tr>
<td>Tuberculoma</td>
<td>10%</td>
<td>49%</td>
<td>50%</td>
<td>-</td>
<td>100%</td>
</tr>
<tr>
<td>Neroma</td>
<td>75%</td>
<td>12.5%</td>
<td>12.5%</td>
<td>-</td>
<td>75%</td>
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</tbody>
</table>

CHAPTER FIVE
DISCUSSION

The commonest SOL in this study is meningioma (28.8%), followed by glioma (26.3%), then abscess (10.2%). Tuberculoma is the fourth common SOL accounting for (8.5%), followed by adenoma (6.8%) and other SOLs account for (19.5%). These findings are not comparable to that reported in literature, that the commonest type of SOL is glioma (32%), followed by meningioma (13.7%), pituitary tumours (13.2%) and tuberculoma (5.5%)\(^4\). However, it is important to know that our study in contrast to other studies (that have been just mentioned) was confined to adult population. Different neoplastic conditions appeared to be the two most common SOLs, in both studies, while meningioma is the commonest in this study, it is the second common SOL in the study reported in literature. Brain abscess seem to occupy similar position in both studies, even frequency among SOLs seems to be comparable to that reported in literature.

The age distribution among the study population is wide. All age groups were affected. This may be explained by the fact that many pathologies were included in the SOLs. The age group >30-42 seems to be the most commonly affected (32.2%), followed by the age group 18-30 (29.6%) and the least to be affected are those of the age above 66. These findings are not comparable to that mentioned in literature, that SOLs are more common in the age group 11-20. This difference can be explained by the fact that this study was confined to adult population. In this series SOLs were found to be more common in males (56.8%). This is comparable to that reported in literature\(^4\), and this can be explained by the fact that most of SOLs with the exception of meningioma, are predominating in males. Moreover this difference in occurrence of SOLs
between males and females was tested using Chi-Square test and the P value was $>0.05$ which is statistically not significant. From the results provided it is obvious that neoplastic lesions account for the majority of SOLs (76.3%).

**Presenting symptoms:**

The most common presentation among study population was that of increased intracranial pressure, thus headache was the commonest complaint being present in (84.9%). The next common complaint was visual deterioration, which was found in (56.8%), and the epilepsy was the third common complaint in these patients accounting for (46.6%). Fever was found in (47%). Headache did not occur in all patients and this goes with literature mentioning that headache is common but no invariable$^{(19)}$. The frequency of headache mentioned in literature varies greatly. It ranges between 71-88%$^{(21,14,3)}$, anyhow, the occurrence of headache seems to be similar to that reported in literature. More than half of our patients suffered from visual deterioration. This can be explained by late presentation of our patients to the physicians. Epilepsy was found to be higher in our patients than that mentioned in literature$^{(10,22)}$. Vomiting was found less than reported in literature$^{(3)}$. The common presence of fever can be explained by the fact that gliomas and abscesses constituting a significant proportion of SOLs in this study (36.5%) could be responsible for the fever, moreover malaria which is endemic in our country may be the cause of fever in some of these patients. Cranial nerve involvement occurred in (31.13%) and the commonest nerve to be affected is the seventh cranial nerve (28.8%). The commonest finding in the optic fundi was papilloedema (31.3%), followed by the optic atrophy which was present in (11.86%). Cerebellar ataxia was present in (15%). Scalp
meningioma, mycetoma and a patient with malignant sarcoma.

Sites commonly appeared to be affected on CT scan are tempoparietal in (16.9%). The next common site was cerebellum and CPA (15.1%) then parietal region (14.3%). Frontoparietal region was the site of the lesion in (10%), followed by sellar and suprasellar in (9.32%). These findings are not comparable to that reported by Abu Salih and Abdul Rahman, that the commonest location is the frontal\(^{(2)}\). However, their study was confined to patients with brain tumours.

**Meningioma:**

This was found to be the commonest SOL (28.8%) and it was the most common primary brain tumour accounting for (39%) of all primary brain tumours. These findings are not comparable to those in literature that meningiomas constitute about fifth of all primary intracranial tumours\(^{(8,14)}\), however Abu Salih and Abdul Rahman found that meningioma was the commonest brain tumour\(^{(2)}\). The age group >30-42 was affected more commonly, followed by the age group >54-66 and those above the age of 66 appeared the least to be affected. Meningiomas in this series were not reported to affect those below the age of 20. This is not comparable to that reported in literature that meningiomas primarily present in the age group 40-60, but appeared to be comparable to that reported by Abu Salih and Abdul Rahman, whose series showed low incidence of meningioma in the age group 51-60 and they did not report a single case above the age of 61. In this study females were affected more commonly (55.9%) than males, this is in keep with literature that meningiomas are more common in females\(^{(8,10,14)}\). But differs from that reported by Abu Salih and Abdul Rahman, who showed that meningiomas are more common in males (66.1%)\(^{(2)}\).
The commonest symptom is the headache (55.3%), it is comparable to that reported in literature\(^3\), but higher than that reported by some authors\(^{14,21}\), and even higher than that reported by Abu Salih and Abdul Rahman\(^2\). The next commonest symptom to be encountered was visual deterioration (73%). This is comparable to that reported by Abu Salih and Abdul Rahman, an explanation could be the fact that our patients tend to present late. The next common complaint was epilepsy (52.9%). It was more than reported in literature\(^8\) and slightly less than that reported by Abu Salih and Abdul Rahman\(^2\). Vomiting was reported in (35.3%) and mental symptoms in (29.4%).

The relationship between head trauma and meningioma was assessed using Chi-Square test and the P value was >0.05 which is statistically insignificant. Cranial nerves involvement was evident in 29.4% and the commonest nerve to be affected was the seventh, followed by sixth nerve. Papilloedema was detected in (26.5%). It was greatly less than what was reported by some authors\(^3\), but it has also been mentioned in literature that papilloedema is significant but not invariable\(^{19}\). Optic atrophy noticed in (14.7%) and it was post-papilloedemic in all cases. Hemipareisis or hemiplegia occurred in 32.5%. Ataxia was seen in (5.9%).

The commonest site where lesions were found, was parasagittal region in (14.7%). The next common sites were parietal and temproparietal (11.7%), convexity in (11.7%), then frontoparietal and parasellar in (8.8%) and followed by CPA in (5.9%). These findings are comparable to that reported in literature but differ from that reported by Abu Salih and Abdul Rahman, who reported that the commonest site was the frontal\(^2\). Multiple lesions were seen in 2.9% only. It has been mentioned in literature that multiple lesions may be seen occasionally\(^8\).
The majority of meningiomas were hyperdense lesions on CT scan (91.2%) and some isodense lesions were also seen (8.8%). This is comparable to literature mentioning that meningiomas are hyperdense lesions and of multifocal attenuation in 5%\(^{(33)}\). Isodense lesions were also described\(^{(8)}\), anyhow 2 out of 3 patients with isodense meningiomas that reported on CT scan had their meningioma confirmed histopathologically. Enhancement was found in all patients (100%). Shifting of midline structures was seen in (26.5%). Calcification was seen (8.8%).

**Gliomas:**

It was the second common SOL accounting for (26.3%), also it was the second common primary brain tumour. This is not comparable to reports in literature that glioma is the commonest SOL\(^{(4,7,19)}\). This finding is comparable to what reported by Abu Salih and Abdul Rahman that glioma was the second common brain tumour\(^{(2)}\). The most common type of glioma detected in this study is astrocytoma (58.1%), followed by glioblastoma (12.9%), cystic glioma (6.45%), then ependymoma (6.45%) and oligodendroglioma was the least one (3.2%). These findings are in keep with literature stating that astrocytoma is the commonest glioma\(^{(15)}\). Astrocytoma constitute about (20.7%) of all primary intracranial tumours and seems higher than that reported in literature\(^{(8,14)}\). Glioblastoma comprised (4.6%) of all primary intracranial tumours which is greatly less than that reported in literature\(^{(8,14)}\). Why gliomas are less common than meningioma? I think this is because patients with gliomas may die before being seen by physicians, moreover these patients may be diagnosed as having malaria or encephalitis because of the fever that they may have. In this study gliomas affect commonly those in the age group 18-30, then those in the age group >30-42. This differs from that reported in literature, that gliomas commonly affect those between 40-60 years\(^{(8)}\). Abu Salih and
Abdul Rahman mentioned that gliomas commonly affect those in the second decade, followed by those in the third decade. This is also not comparable to our finding, but we should remember that our study was confined to adult population. Males (61.3%) were affected more common than females (38.7%), this is comparable to reports in literature mentioning that gliomas are more common in males\cite{8,9,10,14}. In Abu Salih and Abdul Rahman series (68.4%) of patients were males\cite{2}.

Headache was reported in (83.3%) of all glioma patients. In the view of different reports in literature about occurrence of headache in gliomas, headache in this study seems to be higher than some reports but less than others\cite{36,14,21}, it was higher than that reported by Abu Salih and Abdul Rahman. The second commonly encountered symptom is visual deterioration (70.9%) which is higher than that reported by Abu Salih and Abdul Rahman\cite{2}. Epilepsy was found in more than half of the patients (51.6%) which is higher than that reported in literature\cite{22}, and also higher than that reported by Abu Salih and Abdul Rahman. Fever was reported in (45.2%) and vomiting in (32.25%). The commonest finding on physical examination was cranial nerve involvement (41.9%). The seventh cranial nerve was affected more commonly than the others, followed by sixth cranial nerve. Motor weakness (hemiparesis or hemiplegia) was the next common finding (38.7%). The next finding was that of fundal changes. Papilloedema was the commonest fundal change to be found (32.25%). Optic atrophy was an uncommon finding being present only in (3.2%). Ataxia was present in (12.9%).
CT findings:-

The commonest location of the glioma was found to be in tempoparietal (29%), followed by temporal (16%), cerebellar in (17.6%) and in frontal and parietal in equal proportion of patients (9.7%). This is different from that reported in literature\(^{(14)}\), that the commonest sites are frontal, temporal and parietal and also different from that reported by Abu Salih and Abdul Rahman. The precontrast scan revealed that lesions were hyperdense most of the time (38.7%). Hypodense lesions were the second to be encountered (25.8%) and then lesions of mixed attenuation (16%) and the isodense lesions were the least to be encountered. This is not comparable to literature stating that most gliomas show mixed attenuation and isodense lesions may be found in only (10%)\(^{(33)}\).

Abscess:-

This constitutes (10.2%) of all intracranial SOLs. It was the third common SOL. It is comparable to that reported in literature that abscess is the third common intracranial SOL comprising (13.2%) of all SOLs\(^{(4)}\). Brain abscess affects mostly those in the age group 18-30 in (66.6%), followed by those in the age groups >30-54 in (16.6%) and in equal proportion (8.5%) in those in the age groups >30-42 and >54-66.

The males were predominantly affected (83.3%), this in keeping with literature reporting that abscess is 2 to 3 times common in males\(^{(10)}\). Headache and fever were the commonest symptoms occurred in equal proportion of patients (91.6%), followed by epilepsy in (50%), this is higher than reports in literature mentioning (72%) for headache, (60%) for fever and (35%) for epilepsy. Vomiting was the next commonest complaint (41.6%), which is higher than reported in literature\(^{(10)}\), visual deterioration in (33.3%) which is higher than reported in literature. Confusion was seen in only (8.3%) which is less than reported in literature (26%) and
aphasia (8.3%) also less than reported in literature\(^{(10)}\). Cranial nerve involvement was evident in half of the patients.

Papilloedema was detected in (33.3%) which is higher than that reported in literature\(^{(10)}\). Hemiparesis or hemiplegia were detected in (33.3%), which is higher than that mentioned in literature (9%). Ataxia was seen in (25%). Commonly the abscess was located in the cerebellum (25%) and parietal in equal proportion (25%), followed by equal proportion (16.6%) in temporo-parietal and temporal. The least site to be affected were frontoparietal and parieto-occipital in equal proportion (8.3%).

Multiple lesions were seen in only (16.6%). All lesions (100%) were hypodense. Ring enhancement was also seen in all patients as did oedema (100%). Shifting of midline structure was detected in most of the patients (58.3%). The mean value of WBC was 6800 which is less than that reported in literature, this may be because our patients tend to have low WBC comparatively with Europeans and because some patients started treatment already when seen by the author.

**Tuberculoma:**

Accounts for (8.5%), of all cases, it seems comparable to that reported by Bauchama et al (5-8%)\(^{(65)}\), and higher than that reported by Qureshi et al (5.5%)\(^{(4)}\), and also higher than that reported by Liu C et al\(^{(64)}\) and because tuberculoma was reported to be rare in European countries, it seems to be higher than in these countries.

Those in the age group 18-30 and those in the age group >30-42 appeared to be affected equally. This is not comparable to that mentioned in literature that about two thirds of the patients were younger than 20 years\(^{(66)}\) and this may be due to the fact that our study was confined to
adult population, anyhow, our result is comparable to that reported by Boucetta et al, who mentioned that most of the patients were young\textsuperscript{(67)}.

Most of the patients were males (60%). It is comparable to that reported in literature and more over some reports mentioned that most of patients were males (77\%)\textsuperscript{(68)}. Headache was commonest complaint (90\%), followed by epilepsy and fever in equal proportion (40\%) and vomiting was evident in (30\%). Visual deterioration and mental symptoms were seen in equal proportion (20\%). Hemiparesis or hemiplegia were common findings (40\%) and papilloedema in (20\%). Confusion and cranial nerve involvement were the next to be encountered (10\%). Although symptoms of raised intracranial pressure were marked in our patients, they were less than that reported in literature\textsuperscript{(67)}.

Tuberculomas detected on CT scan were commonly located in the parietal (50\%), followed by tempoparietal and frontoparietal in equal proportion (20\%) and least common in parasagittal (10\%). This is different from that reported in literature, that the tuberculoma often found in the cerebellum\textsuperscript{(8)}, but it seems comparable to other reports, mentioning that most intracranial tuberculomas were found in cerebral hemisphere and one third of them were located in the cerebellum. Although multiple tuberculomas (16\%) were reported in literature. They were not reported in this series. Commonly the lesions were hypodense ones (50\%), isodense lesions were the next common lesion (40\%) and hyperdense lesion in the minority (10\%) and in this hyperdense lesion, histopathological confirmation was obtained. In literature tuberculoma was reported often as a lesion of multifocal attenuation and it may be hypodense or isodense.

In this series all lesions showed enhancement, however, target lesion which was claimed to be characteristic by some authors\textsuperscript{(89)}, and pathognomonic by others\textsuperscript{(62)}, was not seen in this series. Oedema was
found in most of patients (60%) and shifting of midline structures in only (10%).

Mantoux test was positive in most patients (60%), and negative in the minority (%30). Sputum for AAFB was negative in most patients (80%). The ESR was 80 mm/hr and above in most of the patients (60%), 60 mm/hr and below in (40%), chest x-ray was normal in most of the patients (80%), although Mantoux test and sputum and chest x-ray failed to confirm the diagnosis most of the time, ESR was raised to some extent in this series.

I think the real incidence of tuberculoma may be higher than what reported in this series, because tuberculosis is still common in our country, also because of the fact that tuberculoma lacks specific clinical, and radiological features, it can be missed easily, therefore, high index of clinical suspicion should be maintained.

**Pituitary adenoma:**

This constitutes (6.8%) of all intracranial SOLs and (8.7%) of all primary intracranial tumours. It has been mentioned by some authors that adenoma comprised (13.2%) of all SOLs which is higher than what reported in this series. The incidence of adenoma among patients with brain tumours was found to be higher than reported in literature\(^4\), however, it is comparable to that reported by Abu Salih and Abdul Rahman\(^2\). Commonly affected patients were those in the age group >30-42, followed by those in the age group >42-54, and those in the age group 18-30 appeared least to be affected. This is not comparable to that reported by Abu Salih and Abdul Rahman who mentioned that adenoma commonly affect those in the third decade, followed by those in the second decade. It has been mentioned in literature that adenoma is common in adult life\(^6,10\).
In this series half of the patients were males. This differs from that reported by Abu Salih and Abdul Rahman, who mentioned that males were affected more than females (61.5%). Headache and visual deterioration were the commonest symptoms and they were found in all patients. The commonest sign to be found was optic atrophy (62.5%) denoting late presentation of our patients. Acromegalic physique was next common finding, which was evident in (37.5%). Visual field defects were common (37.5%), other patients denied any constriction of visual fields, however, these patients were examined crudely for visual field defects by confrontation method, and perimetry was not done for them. Galactorrhoea was detected in (25%). It is clear that the commonest presentation of patients with pituitary adenoma in this series is headache, visual impairment and endocrinopathy.

All tumours has sellar location. Suprasellar extension was found in (25%) of patients. Erosion of clinoid processes was seen in most of the patients (62.5%). The majority of the lesions were hyperdense lesions (75%). Hypodense and isodense lesions were seen in equal proportion and in the minority of the patients (12.5%). Enhancement was seen in the majority of patients (75%). Some nonenhancing lesions were noticed also and the cause of these nonenhancing lesions could be prolactinomas. Calcification was noticed in only (12.5%).

Prolactin was found to be high in half of patients and normal in quarter of patients. This is higher than reported in literature stating that hyperprolactinaemia may be present in (25-40%) of patients with pituitary adenoma. Other pituitary hormones were normal in (62.5%). Acromegaly was diagnosed on clinical ground only, because of the absence of facilities used in determination of growth hormone, anyhow, it seems higher than reported in literature.
Other Space Occupying Lesions:

These account for (19.5%). The commonest types of the lesions in this group were cerebral metastases, meningitis disease of the brain, arachnoid cyst and cerebellar cyst, all occurred in equal proportion (2.5%). The next common type of the lesion in this group was found to be cranial mycetoma and haemangioblastoma in equal proportion (1.69%). The least encountered lesions in this group were cholesteatoma, pineocytoma, acoustic neuroma, carniopharyngioma, epidermoid cyst, malignant sarcoma and aneurysm of posterior communicating artery in equal proportion of patients (0.84%). The age distribution is wide covering all age groups, but commonly affected age group was 18-30, followed by the age group >30-42, then the age group >42-54 and the age group >54-66 and those above the age of 66 appeared least to be affected (8.7%). Males were more affected than females. Clinical presentation was dominated by raised ICP, thus headache was the commonest complaint (78.3%), followed by vomiting (56.5%) and epilepsy in (47.8%). Mental symptoms were evident in (13%). The commonest finding on physical examination was papilloedema (47.8%). Motor weakness (hemiparesis or hemiplegia) was the next to be encountered (26%). Cranial nerve involvement (21.7%), cerebellar ataxia was present in (26%). In addition to symptoms, there were other findings in certain lesions, for example scalp swelling was seen in 2 patients with mycetoma. In 2 patients with cerebral metastases chest signs (clinical and radiological) were found, and in the third patient with metastases post-mastectomy scar was noticed.

Commonly lesions were found to be located in the frontoparietal region (21.7%), followed by cerebellum and frontal lobe in equal proportion (17.4%), followed by CPA and temproparietal region in (8.7%)
and the least sites to be affected were temporal and parietal regions in equal proportion (4.2%).

All patients with mycetoma had serology positive for streptomyces somaliensis. Two out of three patients with cerebral metastases had the primary confirmed histopathologically (breast carcinoma and bronchial carcinoma). In the third patient clinical findings (the patient being excessive smoker, cough, dyspnoea) and the radiological features (lung mass on chest x-ray and multiple SOLs on brain CT scan) were highly suggesting of bronchial carcinoma.

**Cerebral metastases**:

These account for (2.5%) of all intracranial SOLs, and for (3.3%) of all brain tumours. This is less than reported in literature and also less than reported by Abu Salih and Abdul Rahman. Two of these patients had primary tumour in the lung and in the third patient the primary tumour was in the breast. This is in keeping with literature mentioning that the lung and the breast were the commonest primary tumours to have metastases to the brain.

**Mycetoma**:

Accounts for (1.69%) of all intracranial SOLs. One patient was male and the other one was a female. Both of them were in the fourth decade. The commonest presentation was that of scalp swelling and raised ICP. This is comparable to that mentioned by Arbab et al. The two patients showed positive serology for streptomyces somaliensis, which was reported by Arbab et al to be the commonest causative organism.

**Hydatid disease**:

Accounts for (2.5%) of all intracranial SOLs. Females were more affected than males. Age distribution was wide. The incidence seems to
be higher than that reported in literature for parasitic cyst among intracranial SOLs\textsuperscript{(3)}. In all patients no evidence of extracranial hydatid cyst was detected.

**Arachnoid cysts**

Account for (2.5\%) of all SOLs. This is higher than that reported in literature\textsuperscript{(56)}. Females were more affected than males.
CHAPTER SIX
CONCLUSIONS

- Neoplastic conditions account for (75.6%) of all intracranial SOLs.
- The commonest intracranial SOL is meningioma, followed by glioma and then abscess.
- Tuberculoma is the fourth common intracranial SOL and therefore high index of clinical suspicion should be maintained and it should always be considered in the differential diagnosis of cerebral SOLs.
- The commonest clinical presentation was that of raised intracranial pressure.
- Intracranial SOLs are more common in males.
- Intracranial SOLs were commonly located in temproparietal.

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CHAPTER SEVEN
RECOMMENDATIONS

1. To extend study about tuberculoma.
2. Introduction of stereotactic techniques for biopsy taking.
3. To train more neurologists and neurosurgeons to deal with cases in the district hospitals.
4. To introduce MRI to help in early diagnosis of cerebral SOLs.
CHAPTER EIGHT
REFERENCES


APPENDIX
Cerebellar abscess with perifocal oedema, enhancement and obstructive hydrocephalus

Frontoparietal: Front \( \sigma \) parietal cystic lesion noted continuous e\' horns of the lateral ventricles suggestive of arachnoid cyst, no mass effect or shift seen.

Frontoparietal meningioma, marked mass effect and shifting of the midline structure, irregular bone thickening noted adjacent to the mass. Homogenous enhancement was seen.
A patient with Scalp Swelling due to Meningioma.
(with permission of Dr. M.A. Arbab)
**QUESTIONNAIRE**

Name: .......... Age: ................. Sex ..............

Residence: .......... Tribe: .............. Occupation: ........

<table>
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<tr>
<th>Symptoms</th>
<th>Yes=1</th>
<th>No = 0</th>
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</thead>
<tbody>
<tr>
<td>Headache:</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Vomiting:</td>
<td>Yes</td>
<td>No</td>
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<tr>
<td>Nausea:</td>
<td>Yes</td>
<td>No</td>
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<tr>
<td>Epilepsy:</td>
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<td>No</td>
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<tr>
<td>Fever:</td>
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<td>No</td>
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<td>Loss of consciousness:</td>
<td>Yes</td>
<td>No</td>
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<td>Neck stiffness:</td>
<td>Yes</td>
<td>No</td>
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<tr>
<td>Mental symptoms (Specify):</td>
<td>Yes</td>
<td>No</td>
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**Sensory symptoms:**

| Loss:                              | Yes   | No     | Site:...... |
| Paraesthesia:                      | Yes   | No     | Site:...... |

**Motor symptoms:**

| Unsteadiness:                     | Yes   | No     | Site:...... |
| Tremor:                           | Yes   | No     | Site:...... |
| Stiffness:                        | Yes   | No     | Site:...... |

**Symptoms due to cranial nerve involvement:**

| Anosmia:                          | Yes   | No     |
| Visual impairment                 | Yes   | No     | (specify) |
| Diplopia:                         | Yes   | No     |
| Squint:                           | Yes   | No     |
| Deviation of the mouth:           | Yes   | No     |
| Deafness: Yes □ | No □ |
| Dysphagia: Yes □ | No □ |

**Other symptoms:**
- Speech disturbance: Yes □ No □
- Behavioral changes: Yes □ (specify) ........................ No □
- Incontinence: Yes □ No □

**Past medical history:**
- TB: Yes □ No □
- Trauma: Yes □ No □
- Epilepsy: Yes □ No □
- Otitis media: Yes □ No □
- Sinusitis: Yes □ No □
- Schistosomiasis: Yes □ No □

**Family history:**
- Similar disease: Yes □ No □ Who:.............
- Other disease: Yes □ No □ Who:.............

**Drug history:** .................................................................

**Physical examination:**
- Fever: Yes □ No □ Anaemia: Yes □ No □
- Jaundice: Yes □ No □ Breast lump: Yes □ No □
- Lymph-nodes: Yes □ No □ Thyroid nodule: Yes □ No □
- CVS: Normal □ abnormal □ Specify: ......................
- R.S: Normal □ abnormal □ Specify: ......................
- Abdomen: Normal □ abnormal □ Specify: ......................
- Skin condition: Normal □ abnormal □ Specify: ......................
- Abdomen: Normal □ abnormal □ Specify: ......................
Nerve system:

Oriented: Yes ☑ No ☐ Dementia: Yes ☑ No ☐
Confused: Yes ☑ No ☐ Motor dysphasia: Yes ☑ No ☐
Comatose: Yes ☑ No ☐ Sensory dysphasia: Yes ☑ No ☐
Global dysphasia: Yes ☑ No ☐
Cranial nerve lesions:

R ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐
L ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

Fundoscopy:

R: Normal ☐ Abnormal ☐ Specify: ......................
L: Normal ☐ Abnormal ☐ Specify: ......................

Neck:

Neck stiffness: Yes ☑ No ☐
Weakness of flexor: Yes ☑ No ☐
Weakness of extensor: Yes ☑ No ☐

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Reflexes, tone, sensation (3= increased, 2= normal, 1= reduced)

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<th>Supinatar</th>
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<td>Position</td>
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Abdominal reflexes

**Planter reflex:** (R) .................. (L) ..................  
**Coordination:** (R) .................. (L) ........... R=L, R>L, R<L  
(R) .................. (L) ..................  

Gait: Normal □ Abnormal □ Specify: .........................  
The back: Normal □ Abnormal □ Specify: .........................  
The skull: Normal □ Abnormal □ Specify: .........................  

Investigations:

- HB%............ PCV............. ESR............  
- TWBC............ P............. L............. M............. E............. B.............  
- CT scan of the brain: ........................................  
  .................................................................  
- Histopathology: ..................................................  
- Other investigations: ...........................................  
- Treatment:  
  - Medical (specify)..............................................  
  - Surgery (specify)..............................................