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Risk Factors & Outcome Associated With Polyhydramnios in Sudanese Women

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Dedication

To My Family

Parents

Sister & Brothers

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Abstract

Objectives:

To detect the associated risk factors, the role of ultrasound in the antenatal diagnosis and fetal outcome in cases of polyhydramnios.

Setting:

Soba University Hospital.

Subject :

100 patients of confirmed polyhydramnios who had been followed in the fetomaternal unit in Soba University Hospital from 1/4/2001 to 1/6/2002.

Results :

Polyhydramnios increased with advanced maternal age, (42%) of the patients were grandmultiparae. Twenty percent of them had bad obstetrical history. Most of the patients (78%) were diagnosed after the gestational age of 28 weeks.

Seventy nine percent of the patients had mild polyhydramnios, (15%) had modrate polyhydramnios and (6%) of the cases polyhydramnios were severe. thirty nine percent of the patients had associated risk factors in the current pregnancy.

These risk factors were as follows:- diabetes in (16%), Rhesus iso-immunization complicated with hydrops fetalis in (4%), congenital malformation in (15%) and 4% had multiple pregnancy.

Sixty five percent of the cases had no complication and did not need any tretament, those with complications were managed by different methods such as control of diabetes, Amnioreduction and Indomethacin therapy.

Thirty eight percent of the patients delivered preterm, (44%) delivered by caeserean section, most of them were emergency caeserean section.

Twenty five percent of the neoborns weighted more than 4kg. Fifteen percent of them had apparent congenital malformation, 43% of the them needed different types of special management. Perinatal mortality was found to be (19.2%).

بسم الله الرحمن الرحيم

ملخص الأطروحة

تمت هذه الدارسة المستقبلية الوصفية في مستشفى سوبا الجامعي الذي يغطي معظم سكان ولاية الخرطوم بالإضافة إلى الحالات المحولة من مختلف أنحاء السودان في الفترة من ٢٠٠١/٤/١م إلى ٢٠٠٢/٦/١م.

وقد شملت هذه الدراسة عدد (١٠٠) حالة تم تشخيصهم بزيادة السائل الأمنيوثي بواسطة الموجات الصوتية.

تهدف هذه الدراسة للتعرف على العوامل المسببة لزيادة السائل الأمنيوثي ودور الموجات الصوتية في التشخيص المبكر لهذه الحالات ومن ثم معالجة الحالات القابلة للعلاج. كما تهدف الدراسة أيضاً لمعرفة تأثير زيادة السائل الأمنيوثي على الأجنة والمواليد.

بعد تحليل بيانات هذه الدراسة وجد أن نسبة زيادة السائل الأمنيوثي تتناسب تناسباً طردياً مع زيادة عمر الأم وكذلك مع تكرار الولادات ، معظم الحالات (٦١%) لم يوجد سبب مباشر لزيادة السائل الأمنيوثي ، بينما في (٣٩%) من الحالات كانت لديهم أسباب مباشرة في هذه الزيادة المتمثلة في الآتي:

(١٦%) لديهم مرض زيادة السكر في الدم ، (١٤%) لديهم تشوهات خلقية بالأجنة ، (٤%) لديهم تعدد في الأجنة ، (٤%) لديهم مضاعفات العامل الريصي.

معظم الحالات (٧٨%) تم تشخيصها بعد الأسبوع الثامن والعشرين من الحمل ، وفي (٧٩%) كانت زيادة السائل الأمنيوثي بسيطة و لم تحدث لهم مضاعفات و لم يحتاجوا إلى علاج أثناء الحمل ، (٣٥%) من المرضى حدثت لديهم مضاعفات متمثلة في الآتي : الولادة المبكرة – الآلام الحادة – وفاة الأجنة واحتاجوا لعلاجات مختلفة متمثلة في الآتي : عقار الأندوميسازين ، سحب السائل الأمنيوثي الزائد وتنظيم السكر في الدم.

(٢٥%) من المواليد كانت أوزانهم أكثر من ٤ كيلو جرام ، كما أن (١٥%) كانت لديهم تشوهات خلقية ظاهرة و (٤٤%) احتاجوا إلى علاجات خاصة. نسبة الوفيات بين الأجنة والمواليد كانت (١٩,٢%).

توصي الدراسة بتحويل جميع حالات زيادة السائل الأمنيوثي إلى المستشفيات المتخصصة.

Introduction & literature review

Introduction:

Amniotic fluid plays a major role in fetal growth and development. Increased amount of amniotic fluid volume can interfere directly with fetal development, and it can be an indirect sign of an underlying disorder, Sonographic evaluation of the amniotic fluid can,thus, aid in the diagnosis of fetal structural anomalies and fetal compromise and can help in guiding pregnancy management decisions.

Definition:

It is a condition in which there is an excess of liquor amnii surrounding the unborn infant. In normal case the actual quantity of fluid varies but up to two litres is considered to be normal and above it constitutes hydramnios. Hydramnios is defined by some authors as amniotic fluid volume above the normal range for gestional age.

For practical clinical purposes it may be considered as :-

Single pool > 8cm

Aminotic fluid index > 90th centile [1].

Incidence:

Polyhydramnios occurs in 0.5-2% of all pregnancies [2]. From study done in Paris 1980, about hydramnios and fetal malformations, the incidence of hydramnios was o.29 % out of 17.528 pregnancies [3]. While in study done in switzerland in 1993 about the prevalence and etiology of hydramnios, the incidence was 1.08 % out of 17.750 pregnancies [4].

Polyhydramnios can be idiopathic (~ 60%), associated with maternal diabetes (~20%), associated with congenital fetal anomaly (~20%).

Even in the absence of an identifiable cause (>60%), polyhydramnios is associated with an increased rate of caesarean section, antepartum fetal death (0.6% vs 0.2%), Postpartum death (2.8% vs 0.4%), abruption (0.9% vs 0.3%), malpresentation (6.8% vs 2.9%), cord prolapse (2.2% vs 0.3%), and large for gestational age infant (24% vs 8%) [2].

Polyhydramnios occurs more often in multipara than in primigravada [5].

Physiology:

Amniotic fluid surrounds and protects the fetus in the amniotic cavity. It provides a cushion against the constricting confines of the gravid uterus, allowing the fetus room for movement and growth and protecting it from external trauma. The space around the fetus is necessary for the normal development and maturation of fetal lungs. It also promotes normal development of limbs by permitting periodic extension, thus preventing joint contractures. The fluid bathing the fetus helps maintain the fetal body temperature and plays a part in the homeostasis of fluid and electrolytes.

The mechanisms of amniotic fluid production and consumption, and the composition and volume of amniotic fluid, depend on gestational age. During the first trimester, the major source of amniotic fluid is the amniotic membrane, a thin membrane lined by a single layer of epithelial cells. Water crosses the membrane freely with no active transport mechanism, so that the production of fluid in the amniotic cavity is most likely accomplished by active transport of electrolytes and other solutes by the amnion, with passive diffusion of water following in response to osmotic pressure changes.

During the latter half of the first trimester and the early second trimester, as the fetus and placenta differentiated, develop, and grow, other pathways for amniotic fluid production and consumption come into play. These include movement of fluid across the chorion frondosum and fetal skin, fetal urine output, and fetal swallowing and gastrointestinal absorption. The chorion frondosum, the portion of the chorion that develops into the fetal portion of the placenta, is a site at which water is exchanged freely between fetal blood and amniotic fluid across the amnion. Fetal skin is permeable to water and some solutes, permitting direct exchange between the fetus and amniotic fluid until keratinization occurs at 24 to 26 weeks' gestation.

Fetal urine production and swallowing both begin at 8 to 11 weeks' gestation and become the major pathway for amniotic fluid production and consumption from the mid second trimester onwards. At 25 weeks the fetus produces approximately 100 mL of urine daily, with production increasing to about 600 ml/d by term and then declining somewhat after 40 weeks. Fetal swallowing leads to amnioic fluid consumption, as the swallowed fluid is absorbed by the fetal gastrointestinal tract. The volume of fluid swallowed increases with gestational age form 200 to 500 mL/d by term.

The fetal respiratory system may also provide a mechanism for

production and consumption of amniotic fluid, although the exact contribution of this system is unknown. Fluid may be absorbed or excreted across the alveolar capillaries or treachea.

There may be a net flow of fluid from the fetus across the respiratory tract into the amniotic fluid. Some exchange of alveolar fluid with amniotic fluid does occur, as manifested by increasing concentrations of fetal pulmonary phospholipids in the amniotic fluid as pregnancy progresses.

As the mechanisms for amniotic fluid production change during the course of pregnancy, the composition of the fluid changes concomitantly. In the first and early second trimesters, fluid production primarily involves passive flow of water across membranes or fetal skin, so that the amniotic fluid is similar in composition and osomolality to maternal and fetal serum. Thereafter, amniotic fluid becomes increasingly similar to fetal urine, which itself changes in nature as the fetus develops. Like fetal urine, amniotic fluid becomes increasingly hypotonic with respect to maternal and fetal serum from the mid second trimester onward. Sodium and chloride concentrations decrease, and urea and creatinine concentration increase.

The total volume of amniotic fluid increases throughout

gestation until 38 to 40 weeks, after which it decreases. At the end of the first trimester, normal fluid volume is approximately 60 ml, with a range of 35 to 100 ml. By 16 weeks, the mean amniotic fluid volume is approximately 200 mL, with a range for normal of 125 to 300 mL. At term it measures approximately 900 ml, with a wide normal range of 500 to 1200 ml. Fluid around the post-term fetus declines to 250 to 500 ml as the pregnancy proceeds.

In addition to its relationship with gestational age, fluid volume also correlates with fetal and placental weight and numbers. In particular, fetuses that are small-for-gestational age tend to have decreased amniotic fluid, while those that are large-for-gestational age tend to have increased fluid volumes.

Maternal factors play a small role in modulating amniotic fluid. Amniotic fluid volume correlates with maternal plasma volume, a relationship that is mediated predominantly by the fetus. Fluid is exchanged freely between maternal and fetal sera across the highly permeable placental. Alterations in maternal hydration lead to changes in net movement of fluid into or from the fetus. This, in turn affects fetal urine production and, hence, amniotic fluid volume. Increased maternal hydration leads to increased fetal hydration, increased fetal urine output, and increased amniotic fluid volume.

Maternal dehydration is associated with oligohydramnios that returns to normal following rehydration of the mother.

Amniotic fluid provides a reservoir for homeostasis of fetal hydration. Fetuses with excess water transfer fluid to the amniotic space, while dehydrated fetuses can conserve water by swallowing more amniotic fluid, absorbing more water across the gastrointestinal tract, and reducing urine production [6].

Aetiology :

Increased production or decreased consumption of amniotic fluid will result in polyhydramnis. The alteration of any factors that regulates the fetomaternal equlibrium may induce an abnormal increase in fluid volume. The factors involoved in this regulation are fetal swallowing, micturition, respiratory movements, and uteroplacental blood flow [7].

The majority of cases of polyhydramnios are mild or moderate in severity and are idiopathic. When the polyhydramnos is severe, an underlying fetal cause is likely. In modrate and mild polyhydramnios, approximately (20%) are due to fetal causes (the majority of which are central nervous system or gastrointestinal anomalies), (20%) are due to maternal causes, and (60%) are idiopathic. Among the subset of cases with severe polyhydraminos,

approximately (75%) are caused by a fetal abnormality and the remainder are due to maternal causes or are idiopathic.

The practical implication is that when a careful fetal survey reveals no abnormality in the setting of mildly increased fluid, a confident diagnosis of idiopathic polyhydramnios can be made; but when polyhydramnios is severe and no fetal anomaly is seen, repeat examination or referral to a tertiary center should be strongly considered [8].

The conditions associated with polyhydrmnios can be divided into fetal, maternal, placental, and idiopathic [7].

Fetal cases18-20% :

Most fetal causes are the result of decreased fluid consumption through the gastrointestinal tract. In particular, any fetal anomaly that impairs swallowing fluid from contacting the absorbative surface of the small intestine may cause polyhydramnios.

Fetal anomalies :

Fetal anomalies are responsible for approximately 12.7%of all the cases of polyhydramnios. The most commen lesions are:-

Abnormalities of the centr nervous system (neural tube defect):

Anencephalus is the most common congenital anomaly which is associated with polyhydramnios [7].

The incidence of anencephaly may reach 3.6 to 4.6 out of 10.000 birth. but the exact incidence is unknown, as most of these fetuses probably spontaneously abort [9].

The explanation of polyhydramnios due to anencephaly may be :

- Transudation of the cerebro-spinal fluid from the exposed meninges.
- Absence of swallowing of the liquor.
- fetal polyuria resulting from lack of antidiuretic hormone or irritation of the exposed center [10].

Risk factors include having a previous pregnancy with anencephaly. The specific nutrition risk factor is thought to be low serum levels of folic acid. Increasing the intake of folic acid for at least three months before becoming pregnant and during the first month of pregnancy reduced the risk of some neural tube defect. Therapeutic abortion may be considered following counseling after early in utero detection of anencephaly [9]. Polyhydramnios may also occur with other fetal abnormalities,

including spina bifida, hydrocephalyus, microcephaly, encephalocele and holoprosencephaly [7].

Gastrointestinal abnormalities :

Prenatally polyhydramnios will arouse suspicion of esophageal atresia or other obstruction of the gastrointestinal tract.

Esophageal atresia and trachio –osephagus fistula occur in approximately 2 or 3 infants per 10,000 births. This condition is frequently complicated by a spiration of saliva and secretions into the lungs, causing pneumonia,choking and possibly death. It is considered a surgical emergency. The disorder is usually detected shortly after birth when feeding is attempted and the infant coughs, chokes and turns blue (cyanosis). Immediate surgical repair of this disorder is indicated so that the lungs are not damaged and the baby can be fed.

As soon as the diagnosis is suspected, an attempt to pass a small feeding tube (nasogastric tube) through the mouth or nose into the stomach should be made. The cause of this birth defect is unknown; therefore prevention is unknown. Prompt diagnoses is necessary to prevent life-threatening complications [11].

Duodenal atresia is present in approximately1out of 6,000 newborns. There is an increased association of duodenal atresia

with Downs syndrome. Approximately 25% of duodenal atresia is found in infants with Downs [12].

From study done in England 2000 about early antenatal ultrasounic diagnosis of cogenital duodenal atresia, it was found that 87% of the total number of patient with duodenal atresia were diagnosed antinetaly by ultrasound (double bubble), while (13%) were diagnosed postnataly [13].

Infants born with duodenal atresia begin vomiting expulsively large amounts of material shortly after birth. The vomitus may be green (bile stained) and the volume often is greater than that which is fed to the infant. One or two initial meconium stools may be passed but no others. Untreated, these infants dehydrate and become critically ill very rapidly. Suspected atresia may be diagmosed after birth with an x-ray. Following surgery, infants may have significant feeding and absorption problems. Prevention is unknown [12].

Other gastro intestinal abnormalities included. Jejunal atresia, omphalocel, gastroschisis, mid-gut volvulus, annular pancreas and diaphragmatic hernia [7].

Genitourinary abnormalitis:

Partial or complete renal obstruction, most comonly ureteropelvic obstrution.

Skeletal malformation :

Including achondroplasia, osteoganesis imperfacta, multiple arthrogryposis and thanatophoric dwarfism.

Fetal Tumors :

Including cystic congenital adenomatoid malformation of the lung, sacrococcygeal teratoma, and malignant cervical teratoma.

Cardiac abnormalities :

Severe congenital heart disease and persistent cardiac arrhythmias account for cardiac-related polyhydramnios

Chromosomal abnormalities :

The most frequent are down syndrome and trisomies 13 and 7.

In study done in England 1994 it was found that there is association between Bartter syndrome (an autosomal recessive disorder of hyperaldosteronism and icreased plasma renin), and early occurrence of polyhydramnios which result from the increase fetal urine out put and amniotic aldosterone is a reliable marker for the prenatal diagnosis of this condition [14].

Genetic syndromes:

Myotonic dystrophy : From study done in United –Astates 1998, it was found that myotonic dystrophy shoud be consederd as diffrential diagnosis for polyhydramnios, Women with a familial history of myotonic dystrophy or ultrasonographic evidence of hypotonia, including positional abnormalities of the extremities, should be offered deoxyribonucleic acid testing for the myotonic dystrophy mutation [15].

Hematologic disorders:

Homozygous alpha thalassemia and fetomaternal hemorrhage.

Intrauterine infections:

Rubella, syphilis, or toxoplasmosis, other [7].

Miscellaneous:

From the study done in United-States 1997 about the association between intrauterine growth restriction and polyhydramnios, it was found that The combination of polyhydramnios and intrauterine growth restriction is ominous. The majority of fetuses have major anomalies or chromosome abnormalities, or both, even when other sonographic abnormalities are absent. Chromosome analysis and detailed fetal evaluation should be offered when polyhydramnios

and intrauterine growth restriction are identified prenatally [16].

Muliple pregnancy:

Multiple pregnancy accounts for 4.9% of all cases of Polyhydramnios.

Polyhydramnios has been reported in 25% of mononzgotic twins, with twin-to-twin transfusion syndrome. The recipient twin may develop polyuria, congestive heart failure. Hydrops, and Polyhydramnios. Usually the donor sac becomes oligohydramniotic. These events may be seen as early as the middle part of the second trimeste [7].

Maternal causes (18-20%):

Diabetes mellitus:

Polyhydramnios is present in 1.5% to 66% of all diabetic pregnancies. Diabetes mellitus may be responsible for approximately 14% of all cases of polyhydramnios [7].

Diabetes associated with an increased frequency of polyhydramnios and represents the most common maternal cause of elevated amniotic fluid volume. Polyhydramnios is especially frequent when the diabetes is poorly controlled. The underlying mechanism is unknown, and, in particular, no relationship has been found between polyhydramnios and fetal urine production rate in

diabetic mothers [17].

Rh isoimmunization:

Excessive production and accumulation of amniotic fluid are complication of erythroblastosis fetalis, usually indicating deterioration of the fetal status.

In the management of Rh-immunized patiants spectrophotometric analysis of the amniotic fluid is a reliable method for evaluating the severity of the fetal hemolytic process and for determining the optimal time for intra uterine transfution or for delivery of the infant.

When normal amniotic fluid is examined in a spectrophotometer using water as a blank, the optical density (OD) readings between 350 and 650 nm form an almost straight line. If the amniotic fluid contains bilirubin, the (OD) readings will show a peak at 450 nm,and the size of the peak will be proportional to the amount of pigment in the fluid. In case of polyhydiamnios the bilirubin conent of the amniotic fluid become diluted, resulting in a falsely low delta (OD) 450 valne that may be misleading when evaluating the severity of the disease. If polyhydramnios is suspected or diagnosed, the total volume of amniotic fluid should be determined using 51Cr, evans blue or sodium paraaminohippurate, and the OD 450 vlue should be

corected for dilution [7].

The use of prophlactic Rh(D) immune globin has made isoimmunization an uncommon cause of polyhydramnios [18].

Placental causes(<1%):

The most common placental causes of polyhydramnios are placentalchorioangioma and the circumvallate placenta syndrome [7].

Recurrent polyhydramnios :

From the study done in Australia 1993 about the significance of recurrent polyhydramnios it was found that recurrent polyhydramnios occurred in 1 in1. 720 pregnancies [19].

Geographic factors :

In study done in United- States 1994 for the efect of altitude on the amniotic fluid index, it was found that high altitude population had a greater proportion of weman with polyhydramnios when compared with those in low altitude (8.5 %: 1.0 %). This finding suggest that there is an association between high altitude an increase in the amniotic fluid index. The mechanism and clinical significance of this effect are unknown [20].

Idiophatic causes (65%):

Idiophathic polyhydramnios was the most frequent finding in several studies. Most mild cases of polyhydramnios are idiopathic. In a study of 102 cases of polyhydramnios, only 16% of the mild cases, defined as a vertical amniotic fluid pocket greater than 8 cm but less than 12 cm had a cause. In sever cases, defind as vertical pocket greater than 16 cm, an etiologic factor was identifiable in 91%. In approximately 65% of the cases, polyhydramnios is idiopathic [7].

From study done in United- states 1994 about the association of idiopathic polyhydramnios with fetal macrosomia it was found that idiopathic polyhydramnios is associated with large –for –gestational – age fetuses and macrosomia independent of maternal diabetes [21].

From study done in Paris 1980, about the hydramnios and fetal malformation, in 50 cases of hydramnios were found in 17. 528 pregnancies (0.29 %). These 50 cases of hydramnios gave birth to 53 fetuses which showed one or more of the following abnormalities:- (37.7%) were congenital malformation of which (60 %) were of the central nervous system, (11.3%) were twins, (11,3 %) were cases of Rhesus allo-immunisation, (1.9%) case of chorioangioma, (7.5%)

were unexplained itra-uterine deaths, (50.9%) infants that died later and (37.7%) were idiopathic. There was no maternal pathology to be found, often the obstetrical history of these women are pathological, in (3%) had a previous abortion and (18%) had a previous hydramnios [3].

From study done in switzerland in 1993 about sonographic prevelance and etiology of polyhydramnios in singleton pregnancy, it was found that in one hundred and forty-nine cases of hydramnios were detected among 13,750 parturients undergoing 25,000 routine ultrasonographic examinations. The prevalence of hydramnios was 1.08%. Ninety-seven patients (65.1%) had no apparent cause for the increased amount of fluid volume. Twenty-eight newborns (18.8%) were congenitally malformed, and 22 (14.8%) patients had diabetes mellitus. Rh incompatibility accounted for only (1.3%) of the cases [4].

Classification:

In most of the cases of hydramnios the excess fluid accumulates gradually (chronic polyhydramnois) and is only noticed after the 30th week. In afew exceptional cases polyhydramnios occurs before 24 weeks and more quickly (acute polyhydramnois) and many of these cases are asocciated with uniovular twins [5].

Clinical Feature:

Increased amniotic fluid volume produces uterine stretching and enlargement that may lead to preterm labour, as well as a variety of other maternal symptoms. The enlarged uterus itself may be painful. It can also compress adjacent organs, resulting in oedema from inferior vena cava compression, oliguria from ureteral compression, and dyspepsia from diaphragamatic elevation. These complications are more likely to occur when the polyhydramnios develops acutely than in the setting of chronic polyhydramnios [22].

Diagnosis:

Clinically, the diagnosis may be suspected by finding a uterine size larger than expected for the gestational age, easy ballottement of the fetus, difficulty in defining fetal parts and fetal heart tons [7]. Afluid thrill can be elicited. The fetus is unduly mobile and the presentation is unstable. The tightness of the uterus varies, but in case of acute polyhydramnios the uterus is very tense [5].

The clinical diagnosis of polyhydramnios should always be cconfirmed by ultrasound [7].

Sonographic assessment of amniotic fluid volume:

Method of assessment:

Several methods have been proposed for sonographic assessment of amniotic fluid volume. These include subjective assessment, measurement of the single deepest pocket, amniotic fluid index, planimetric measurement of total intrauterine volume, and variety mathematical formulae.

Subjective assessment:

Subjective assessment of amniotic fluid volume is accomplished by real-time scanning through the entire uterus and observing the amount of fluid in the gestational sac surrounding the fetus. The fluid volume is subjectively classified as normal, high or low for the gestational age. This approach is quick and efficient and takes into account gestational age-related variation in fluid volume. This method, however, may be unreliable in the hands of an inexperienced operator. Furthermore, the basis for classifying fluid as normal, high or low is difficult to document on hard copy. This can pose problems when the operator is not the person who renders the final interpretation of the scan or when the fluid volume is followed over repeated scans. From study done in Switzerland1993 about Subjective ultrasonographic assessment of amniotic fluid depth it

was found that Subjective ultrasonographic assessment of the amniotic fluid volume may serve as a screening test for the experienced ultrasonographer. However, when a decreased or increased amount of amniotic fluid volume is suspected, one may elect to use the amniotic fluid index for confirmation of the subjective impression [23].

Single-deepest-pocket measurement:

Assessment of amniotic fluid volume using the single-deepest-pocket method involves measuring the maximum vertical depth of any amniotic fluid pocket. A measurment below 2 cm is considered to represent oligohydramnius, and one above 8 cm represents polyhydramnios.

The method is simple and straightforward, but has little mathematical validity or rationale. The volume of a simple shape, such as a sphere or cube, is directly related to, and can be calculated from, a single measurement. In contrast, the volume of a highly irregular shape, such as that occupied by amniotic fluid, can not be calculated from, or even accurately approximated by, one measurement. In addition,the single deepest-pocket measurement can vary considerably if the fetus changes position. Furthermore, measurement of a deep but thin "pancake"-shaped collection

between the fetal legs or alongside the fetus may yield a normal value even in the presence of severe oligohydramnios. Finally, variation in amniotic fluid volume with, gestational age is not taken into account if a lower limit of 2 cm and an upper limit of 8 cm is used for all ages.

Four-quadrant amniotic fluid index:

The amniotic fluid index is determined by dividing the uterus into four quadrants by sagittal and transverse lines through the umbilicus and summing the vertical dimensions of the deepest pocket in each quadrant. When the sum results in a value below 5 cm, it is considered to signify oligohydramnios and one above 18 to 20 cm, polyhydramnios.

This approach is fairly quick and gives a better assessment of amniotic fluid volume than does, the single-deepest-pocket measurement, because the sum of four measurements will always correlate more closely with volume than will a single measurement. However, since the measurement of the deepest pocket in each quadrant bears little relationship to the amount of amniotic fluid in that quadrant, their sum will not accurately reflect the overall fluid volume. Furthermore, changes in fetal position and gestational age variation in fluid volume represent limitations for the four-quadrant

amniotic fluid index, as they do for he single-deepest-pocket measurement.

Planimetric measurement of total intrauterine volume:

Total intrauterine volume can be estimated by obtaining multiple scans through the uterus at regular intervals. The intrauterine area is determined on each scan and multiplied by the width of the interval. These values are summed to yield the total intrauterine volume.

The approach is slow and cumbersome, requiring tracing the uterine outline on each scan to compute the area. An articulated arm is needed to ensure that proper scan planes are obtained. The result of this time-consuming calculation is not the amniotic fluid volume itself, but rather the sum of amniotic fluid, fetal and placental volume.

Mathematical formulae for volume calculation:

A variety of formulae using sonographic measurements to approximate total intrauterine volume minus fetal and placental volumes, and volume of the largest pocket have been proposed. However, all these approaches make the assumption that the uterus (or fetus. Placenta, or deepest pocket) conforms to a regular shape,

such as a prolate ellipsoid. Unfortunately, such assumptions are oversimplifications that can lead to substantial inaccuracy [6].

Differential Diagnosis:

Chronic polyhydramnios has to be distinguished from multiple pregnancy. This may be difficult, especially as polyhydramnios may complicate multiple pregnancy; in such a cases the diagnosis of twin is easily missed. If the twins pregnancy is not complicated by polyhydramnios the essential clinical observations are the discovery of two heads and an unusual number of limbs. An ultrasound scan must be a routine in all cases of polyhydramnios to exclude multiple pregnancyor.

If pregnancy co-exists with a large ovarian cyst the diagnosis from polyhydramnios can be difficult. Ultrasound scan will show sacs, only one of which contain a fetus.

Acute polyhydramnios may simulate placental abruption with concealed haemorrhage, but in the latter condition the uterus is hard and tense and the fetal heart sounds are absent. In most cases of placental abruption there is at least a little external bleeding. Polyhydramnios should be differentiated also from maternal Ascites [5].

complications:

There is increased fetal and maternal morbidity and mortility associated with polyhydramnios.

Spontanous preterm labour may occur. The memberanes may rupture suddenly and there is a risk of prolapse of the umbilical cord. Because the fetus is unduly mobile malpresentation may occur. If a large quantity of amniotic fluid escape suddenly the placental site may diminish in area, and this can lead to abruptio placentae .After delivery there is a risk of postpartum haemorrhage.

The perintal mortiliy is greatly increased with polyhydramnios because there may be a fetal abnormality, and because of the possiblity of preterm labour, cord prolapse and malpresentation [5].

Management:

Ulterasound examination should be performed on every patient with polyhydramnios to detect congenital and and placental abnormalities, basic laboratory studies include maternal antibody screen, diabetic screening, and TORCH (toxoplasmosis, other viruses, rubella, cytomegalovirus, herpes) serology. Fetal karyotype must be obtained using amniocentesis, cordocentesis. Fetal swallowing studies are also indicated [7].

Polyhydramnios without symptoms and without any evidence of fetal abnormality requires no treatment [5].

Serial amniotic fluid decompression is the treatment of choice for severe polyhydramnios when a conservative management is intended.this method not only relieves maternal discomfort, but reduces excessive intrauterine pressure that can induce preterm labor [7].

Abdominal amniocentesis should be done after localizing the placenta with ultrasound, an epidural needle is inserted into the amniotic sac and fluid is withdrawn with an epidural catheter passed through the needle. Up to 2 liters of fluid may be removed, provided that it is only allowed to escape slowly. Since excessive decompression may lead to placental separation Although there is some risk of labour starting after amniocentesis. Unfortunately the fluid is often quickly replaced making repeated procedures necessary. There is always a slight risk of perforating afetal vessel and causing bleeding into the amniotic sac. Also taping may lead to infection [5].

As observed in study done in England 2000, which showed that serial amniocentesis may place patients at elevated risk for Candida chorioamnionitis and subsequent preterm delivery.

Clinicians should consider early diagnostic amniocentesis in patients in preterm labour with a history of prior amniocentesis, and the routine Gram stain and culture of amniotic fluid [24].

From study done in Polish in 2000 about the efficacy of serial amniocentesis in case of acute polyhydramnios in twin-twin-transfution-syndrome, it was found that Serial amniocentesis are effective in significant prolongation of gestation (the mean interval between diagnosis and delivery 24 days). The improvement of perinatal outcome in twin gestations complicated by hydramnios can be achieved by the combination of serial amniocentesis and the laser ablation of anastomoses [25].

An alternative medical treatment is the use of prostaglandin synthetase inhibitors. Indomethacin has been proven effective in reducing the amount of amniotic fluid. It probably acts by decreasing the fetal urinary output or by increasing the reabsortion of fluid via the lungs. A recommended dosage is 2.2 mg/kg/day administered orally every 6 hours. This trearment should be suspended at 32weeks of gestation to avoid neontal hemodynamic coplication such as fetal renal shutdown and premature closure of the ductus arteriosus. Close sonographic surveillance is recommended,

including Doppler assessment of blood flow through the ductus arteriosus [7].

In study done in Ireland 1993 about the efficacy and safety of indomethacin therapy for polyhydramnios, maternal and perinatal outcome of seven gravidas receiving 2.2-2.5 mg/kg per day of indomethacin for polyhydramnios are reported. Such therapy was started between 26 and 33 weeks of gestational age (mean, 30.4 weeks) and lasted for 20.1 days (range, 2-37 days). Median of amniotic fluid index ranged from 47 at the start of therapy (range, 32-53) to 15 (range, 2-50) when indomethacin was ended. Interval between the end of the therapy and the delivery ranged from 0 to 45 days (mean, 15 days). On average, pregnancies were prolonged by 5.1 weeks (range, 2-8 weeks) Oligohydramnios was seen in two instances; one patient developed constriction of the fetal ductus arteriosus, which returned to normality after indomethacin cessation; one newborn in which other causes of neonatal bleeding could be excluded, developed a disseminated intravascular coagulation and died 15 h after birth. Finally, one mother presented as acute renal failure immediately after indomethacin administration; this patient completely recovered after indomethacin withdrawal. Thus, the benefit of pregnancy prolongation should be balanced against the

increased risks for the newborn, mainly fetal ductus arteriosus constriction and possible bleeding disorders [26].

If the fetus is dead or there is growth congenital malformation which is incompatible with life, termination of pregnancy by high artifical rupture of membranes is indicated using Drew Smythe Cather.

In cases near term in which the women is in serious discomfort labor should be induced [10].

From the study done in United –States 1993 about intrapartum hydramnios at term and outcome. The study compares the outcome between 277 subjects with adequate fluid and 22 with hydramnios. The intrapartum amniotic fluid index, birth weight, incidence of macrosomia (birth weight > or = 4000 gm), shoulder dystocia, cesarean section for fetal distress or cephalopelvic disproportion, Apgar score, and neonatal malformations were noted in each of the 299 subjects. Umbilical arterial blood gas was analyzed in 189 patients. In this study, 22 women with hydramnios had a significantly higher risk of delivering a macrosomic fetus than the 277 patients with a normal index (36.3% vs 13.7%; p = 0.01). There was no significant difference in the incidence of shoulder dystocia, cesarean section for cephalopelvic disproportion, or fetal distress, Apgar scores < 7 at 1 and 5 minutes, fetal acidosis, or neonatal anomalies.

We conclude that hydramnios during the intrapartum period in patients at term is significantly associated with delivery of a macrosomic fetus, but not with delivery of a compromised neonate [27].

Objectives

1) Detection of associated risk factors of polyhydramnios.

2) Role of ultrasound in antenatal diagnosis of fetal anomalies associated with polyhydramnios.

3) Fetal outcome in cases of polyhydramnios.

Material and methods

This is a prospective descriptive study to find out the associated risk factors and the outcome in cases of polyhydramnios. It was carried out in the period from 1/4/2001 to1/6/2002 in Soba university hospital.

The hospital covers population in Khartoum state in adition to the referred cases from all other states of the country, delivers medical services through the medical departments which are surgery, medicine, paediatrics, orthopedics in adition to obstetrics and gynaecology.

The activities of the obstetrical and gynaecological department include outpatient, inpatient wards, labour room, major, minor and septic theatres and referred clinics in adition to special fetomaternal unit which following all high risk pregnancies by cardiotogography, biophysical profile and serial ultrasonography.

The Obstetrical and gynaecological department is covered by senior, junior consultants, registrars, house officeres, midwives, nurses and other paramedical staff.

The sudy population consisted of pregnant women attendig the referred clinic of the fetomaternal unit in obove mentioned poried, as suspected cases of polyhdramnios. confirmation of the diagnosis

was allways taken by the consultant obstetrician and fetologist.

Most of the cases of polyhydramnios were admitted in the hospital till they delivered, others patients were followed up as outpatient and they were admitted to the hospital for delivary.

For data collection standard questionnaire was designed, it includes history notes (identifications name, age, parity, personal and family history, amniotic fluid volume, presence of associated factors for polyhydramnios, complication, antenatal mangement, gestational age at diagnosis and delivaries, mode and indications of delivaries and status of the outcome).

All these patients had been followed up during their stay in the hospital untill their discharge, by regular monitoring of fetal well beeing.

According to the result of the diagnosis of polyhydramnios by ultrasound the data from the positive cases were collected and had been coded then transferred to a master sheet and fed to a microcomputer DELL PantiumIII-733MHz the operating system windows 98, professional statistical analysis was performed using statistical package SPSS 9.05. Some appropriate discriptive statistics like frequency tables, graphs, cross tabulation, bar and pie charts had been done.

Inference statistics are done to test hypothesis about the association beteewn variable for that purpose we used pearson chi-square test statistics.

The hypothesis

HO: There is no association between row varible levels and column varible levels.

H1: There is association.

If P. value ≤ 0.05 we reject HO.

That means there is significance.

RESULTS

Out of total number of patients in this study (100), only (4 %) were in age group 17-21, (12%) were 22-26, (18%) were 27-31, (28%) were 32-36, and (38%)were37-41 yreas (Fig.1).

Most of the patients were grandmultiparae, (42%)of them had more than 5 children, (38%) had 1-4 childern and (20%) were primigravidae (Fig.2).

The total number of patients who had past obstetrical history of stillbirths in this study were (6%) (fig.3), those with past history of early neonatal deaths were (2%) (Fig.4), and those with past history of abortion were (12%)of the total number of patients (Fig. 5).

These patients were diagnosed at different gestational ages, (22%) of them were diagnosed at gestational age less than 28 weekes, (31%) between 28-32 weekes, (40%) between 33-37 weekes and (7%) werer diagnosed between 38-42 weekes (Fig.6).

Regarding the presence of past history of risk factors, (3%) had a history of hypertension, (16%) of diabetes, (3%)had multiple pregnancies, (2%) delivered babies with congenital malformation and(2%) had previuos history of polyhydramnios, while (74%)had no past history of risk factors (table1).

When reviewing the family history of these patients, (10%) of them had a family history of hypertension, (14%) of diabetes,(7%) had both hypertension and diabetes, (4%) had a family history of multiple pregnancies and (1%) had a family history of congenital malformation, while (64%) had no related family history (table 2).

On determining the Rhesus factor status of the patients (92%) were found to be Rhesus factor positive and (8%) were Rhesus negtive (table 3).

Using the ultrasound to confirm polyhydramnios, (79 %0) of the cases had a deepest pool of the amniotic fliud volume in the range of 8.1-12 cm, (15 %9) in the range of 12.1-16 cm and in (6 %) the deepest pool was more than 16 cm (Fig.7).

Screening for associated risk factors in the current pregnancy it was found that(61%) of the cases had no related risk factors, while (39%) of them had risk factors (Fig. 8), these risk factors were diabetes mellitus in (16%), Rhesus isoimmunization complicated with hydrops fetalis in 4%, neural tube defects in 8%, gastrointestinal obstruction in(4%), achondroplaisa in (3%) and (4%) of the cases had multiple pregnancies (table 4).

Regarding the complications arising during the current pregnancy, (11%) devolped preterm labour, (6%) suffered from acute abdominal pain (2%) had hydrorrahae gravidarum, (12%) had

permature rupture of membranes and (4%) ended with intrauterine fetal deaths, the others (65%) had no complication (table 5).

Regarding the management provided antenataly for these patients, the (65%) with out complication need no treatment, the rest were managed differently, (16%) received treatment for diabetes, (4%) underwent amnioreduction, (13%) received indomethacin therapy, (1%) received combination therapy of amnioreduction and indomethacin, (1%) received combination therapy of amnioreduction and control of diabetes (table 6).

Regarding the gestational age at delivery, (5%) delivered at gestational age less than 28 weeks, (13%) between 28-32 weeks, (25%) between 33-37 weeks and (57%) delivered at gestational age between 38-42 weeks (Fig.9).

In relation to the mode of delivery (48%) delivered spontaneous vaginal delivery, (8%) had induced labour,(26%) were delivered by emergency caesarean section and (18%) by elective caesarean section (fig.10).

Concerning the indication for those who underwent induction of labor, (50%) of them were due to intrauterine fetal deaths, (25%) were due to complicated Rhesus iso-immunization and (25%) were due to anencephaly (table 7).

The indications for the emergency caesarean section were as follows :- (19.2 %) were due to footling breech, (19.2%) were due to cord prolapse, (7.7%) were due to abruptio placentae, (15.4%) were due to diabetes, (7.7%) were due to twins, the first was breech, (15.4%) were due to fetal compromise and (15.4%) were due to other indications (malpresentation, triplet, preivuos scars) (table 8).

The indications for the elective caesarean section were as follows:- (50%) were due to diabetes with macrosomia, (16.7%) were due to hydrocephalus, (27.8%) were due to repeated caeserean section and (5.5%) was twins the first was breech (table 9).

With regard to the outcome, (80%) of the newborns were alive, (2%) were fresh stillbirths, (4%) were macerated stillbirths and (14%) ended with early neonatal deaths (table 10).

According to the sex of the outcome, (57%) were females and (43%) were males (Fig.11).

regarding the birth weight, (35%) weighed less than 2.5 kg, (40%) ranged between 2.5-4 kg and (25%) weighed more than 4kg (Fig.12).

(85%) of the newborns had no apparent congenital malformation where as in (15%) of them apparent congenital malformation were observed (Figure 13).

The distribution of the apparent congenital malformation were as follows:- anencephaly in (26.6%) of cases, hydrocephalus in (20.1%), encephalocel in (6.6%), hydrops fetalis in (26.6%) and (20.1%) of cases were achondroplasia (table11).

Fivity seven percent of the newborns did not require any special management while (43%) of them received special management (Fig.14); The types of special management received were as follows: (9.3%) managed in incubaters, hypoglyceamia was corrected in (28%), (14%) managed surgically, (11.5%) of the cases were intubated, (4.7%) received blood exchanges and (32.5%) admitted in the nursary for observation (table 12).

Fig.(1): Distribution of patients according to age group

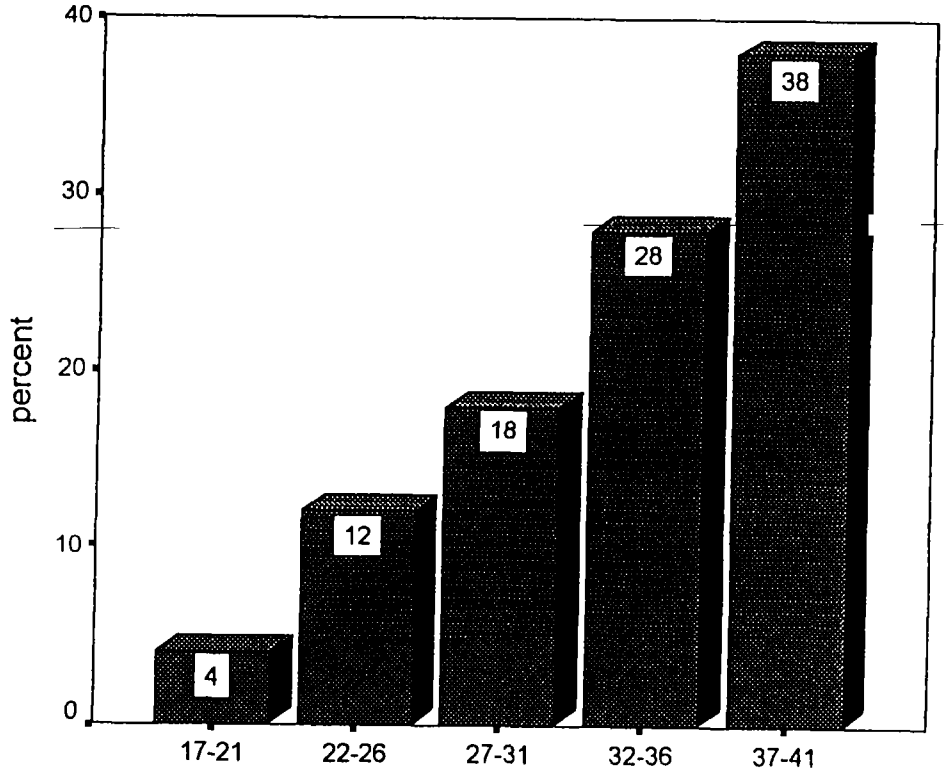

Fig.(2): Distribution of patients according to Parity

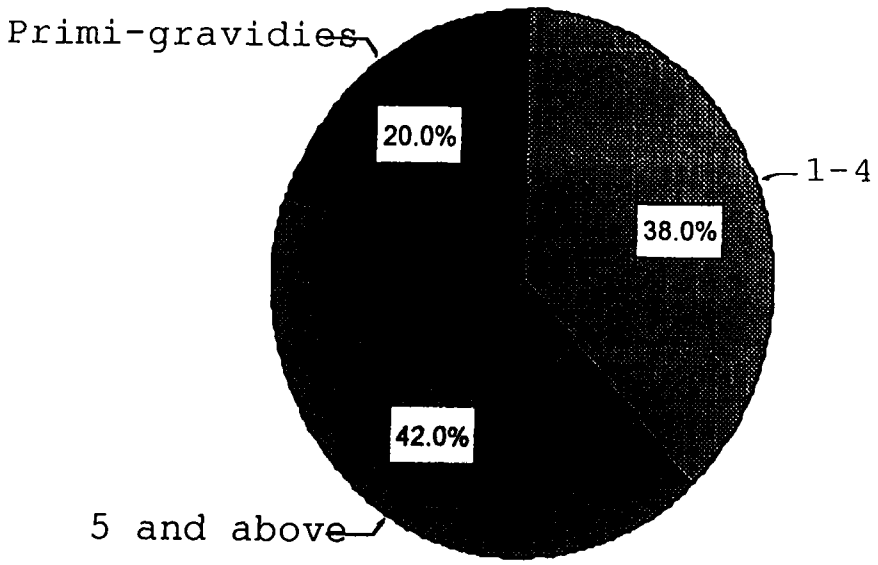

Fig.(3): Distribution of patients according to past obstetrical Histroy of stillbriths

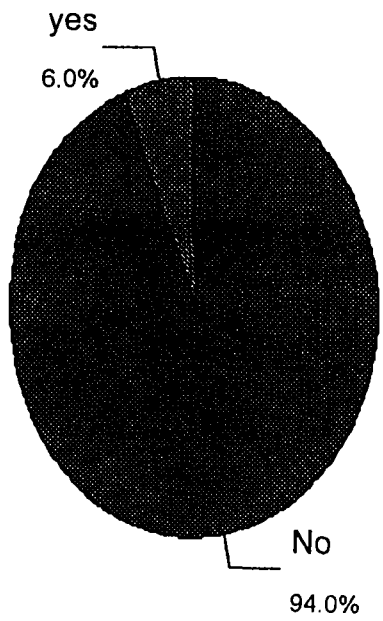

Fig.(4): Distribution of patients according to Past obstetrical history of neonatal deaths

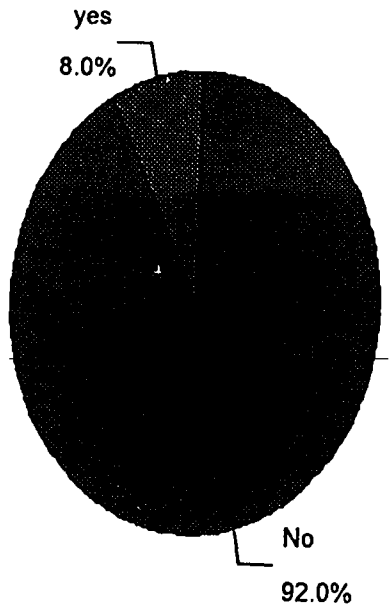

Fig.(5): Distribution of patients according to Past obstetrical history of abortion

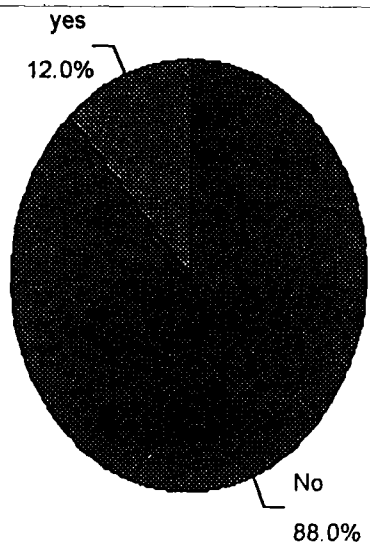

Fig.(6): Distribution of patients according to gestational ages at diagnosis in weeks

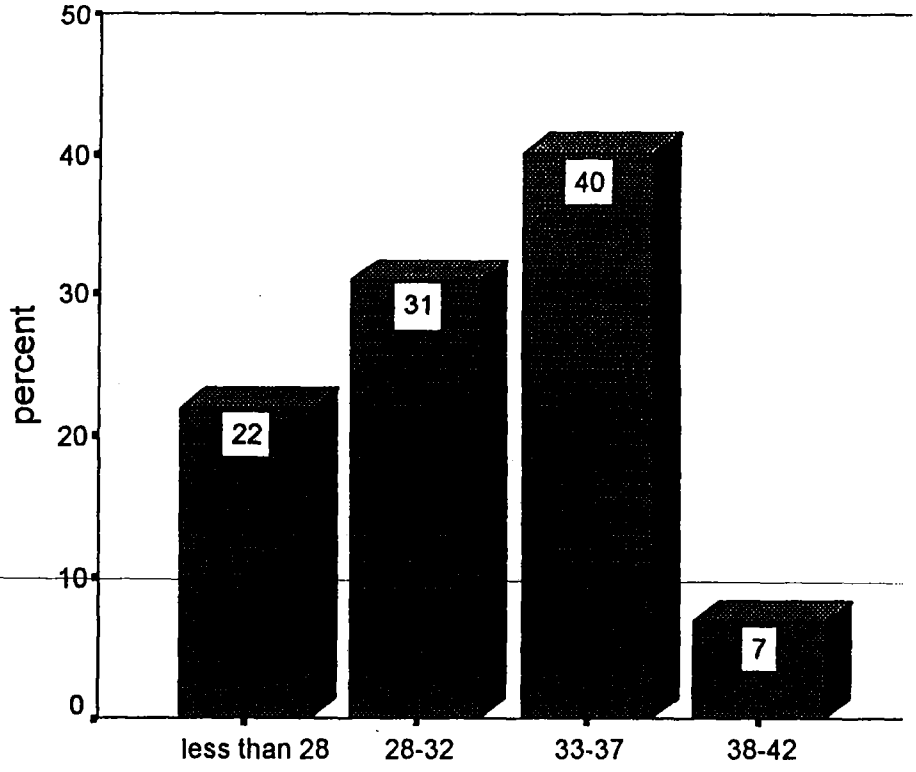

Table (1): Percentage distribution of patients according to past hi personal risk factors

RISK FACTOR	FREQUENCY	PERCI
Hypertionsion	3	3
Diabetes	16	1(
Multiple Pregnancy	3	3
Congenital malformation	2	2
Polyhydramnios	2	2
No risk factor	74	7.
Total	**100**	**10**

Table (2): Percentage Distribution of Patients according to fast ris

FAMILY RISK FACTOR	FREQUENCY	PERC
Hypertionsion	10	1(
Diabetes	14	1.
HPT & DM	7	7
Multiple Pregnancy	4	4
Congenital malformation	1	1
No relevant family history	64	6.
Total	**100**	**10**

Table(3): Percentage distribution of patients according to Rhesus

RH- FACTOR	FREQUENCY	PERCE
positive	88	88.0
negative	12	12.0
Total	**100**	**100.0**

Fig.(7):Dstribution of patients according to Deepest pool of amniotic fluid volume by ultrasound

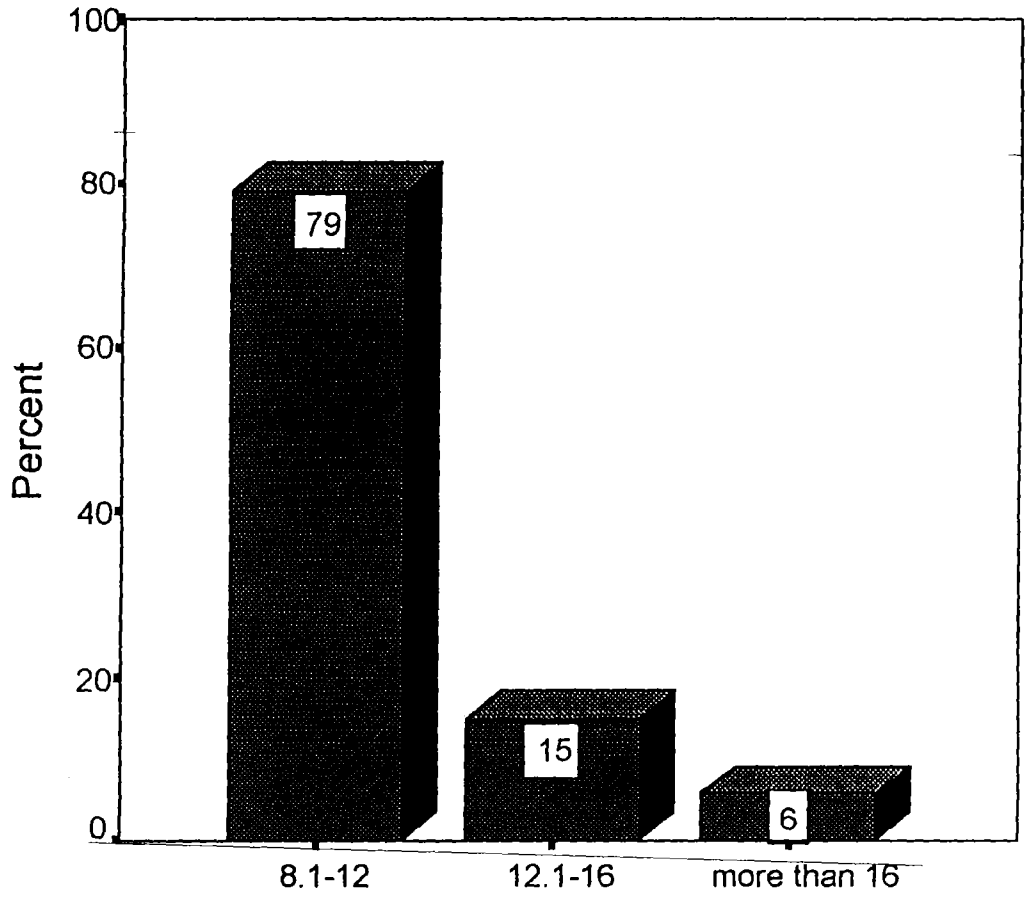

Fig.(8): Distribution of patients according to presence of associated risk factors

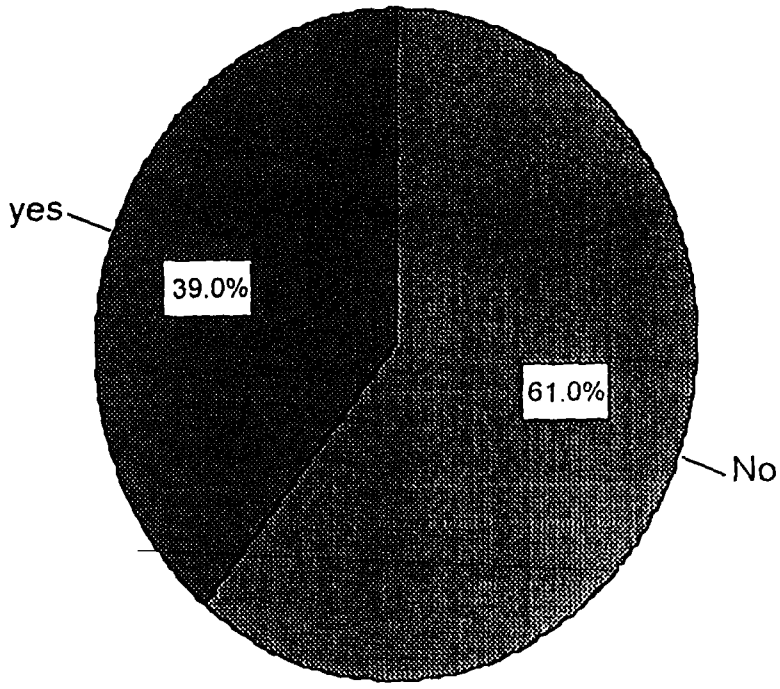

Table (4): Percentage distribution of patients according to different risk factors

RISK FACTOR	PERCENT%
Neural tube defect	20.5
Gastro-intestinal obstraction	10.3
Achondroplasia	7.6
Multiple pregnancy	10.3
Diabetes	41.0
Hydrops fetalis	10.3
Total	**100**

Table (5): Percentage distribution of patients according to current complication

COMPLICATION	FREQUENCY	PERCENT%
Preterm labor	11	11
Acute Abdominal pain	6	6
Hydrorrahae gravidarum	2	2
Premature Rupture of membranes	12	12
Intrauterine fetal deaths	4	4
No complication	65	65
Total	**100**	**100**

Table (6): Percentage distribution of patients according to different antenatal provided management

ANTENATAL MANAGEMENT	FREQUENCY	PERCENT%
Control of diabetes	16	16
Amnioreducation	4	4
Indomethacin	13	13
Amnioreducation & Indomethacin	1	1
Amnioreducation & Control of diabetes	1	1
No Treatment	65	65
Total	**100**	**100**

Fig.(9): Dstribution of patients according to gestational age at delivery in weeks

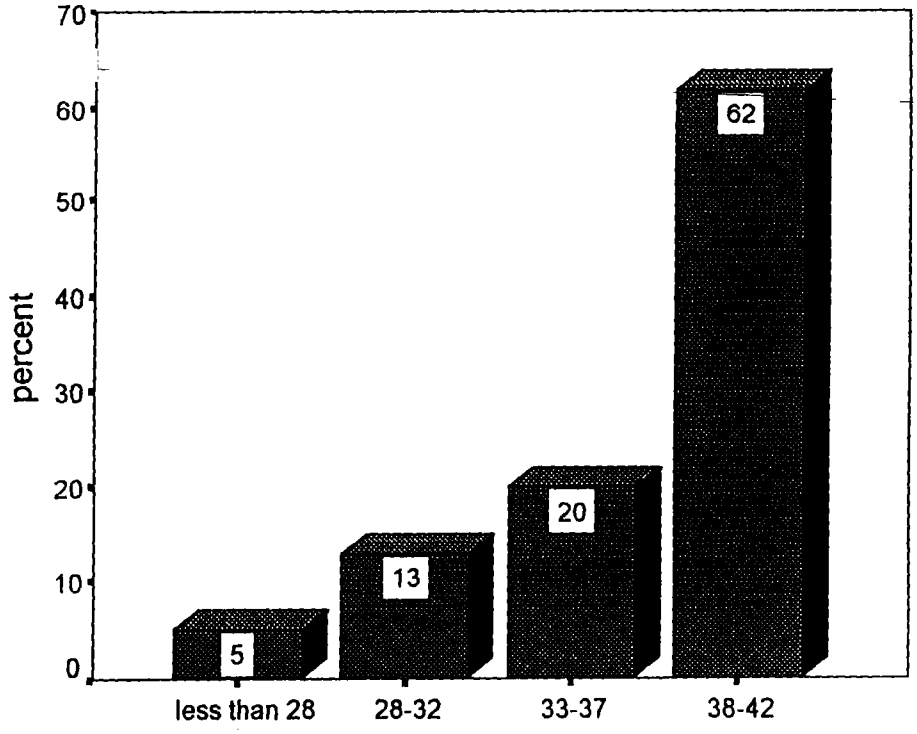

Fig.(10): Dstribution of patients according to Mode of delivery

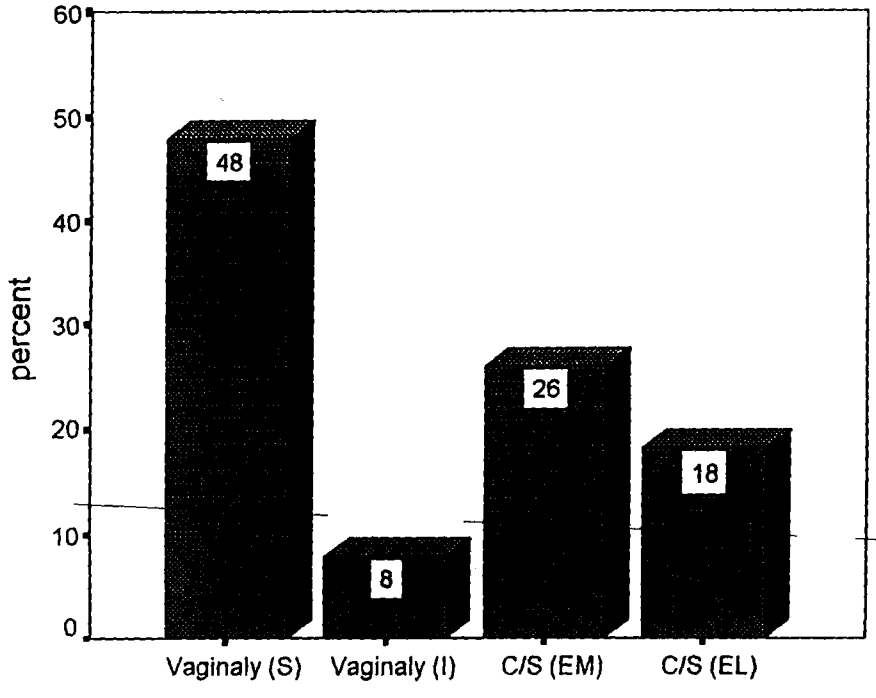

Table (7): Percentage distribution of patients according to indications for induction of labour

INDICATION FOR INDUCTION	PERCENT
Intrauterine fetal deaths	50
Rh-isoimmuniation	25
An encephaly	25
Total	**100**

Table (8): Percentage distribution of patients according to indications for emergency ceasarean section

INDICATION FOR EM/CS	PERCENT
Footling breech	19
Cord prolapse	19
Abrupyio placentae	8
Diabetes + macresomia	15
Twins first was breech	8
Fetal compromises	15
Other (malpresentation, triplet, previous scars)	16
Total	**100**

Table (9): Percentage distribution of patients according to indications for elective caesaren section

INDICATION FOR EL\CS	PERCENT
Diabetes + macrosomia	50
Hydocephalus	17
Reported caesarean section	28
Twin first was breech	5
Total	**100**

Table (10): Percentage distribution of status of outcome

OUTCOME	PERCENT
Alive	80
Frech stillbirths	2
Macerated stillbirths	4
Neonatal deaths	15
Total	**100**

Fig.(11): Dstribution of outcomes according to their sex

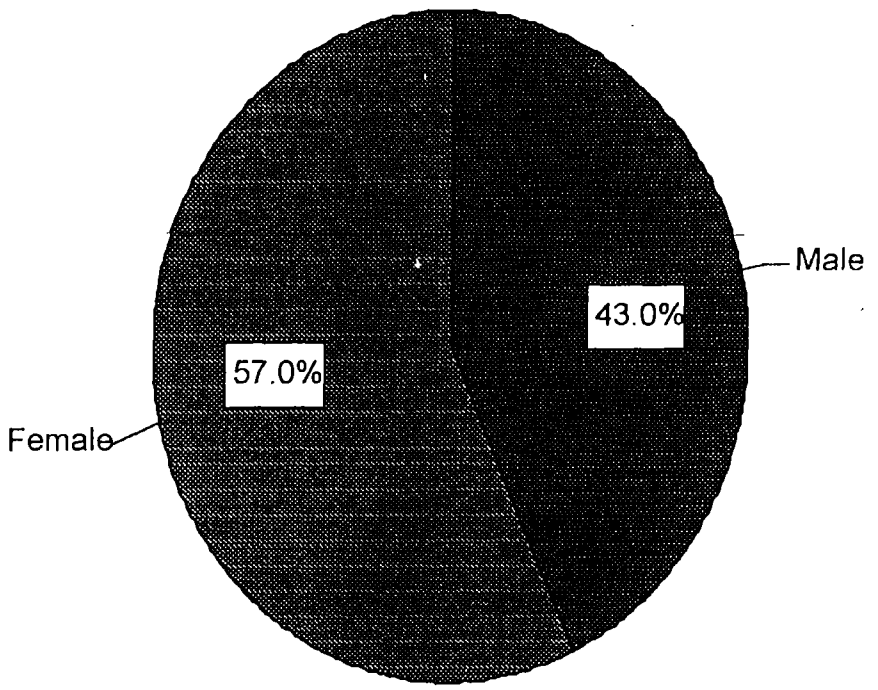

Fig.(12): Distribution of outcome according to weight

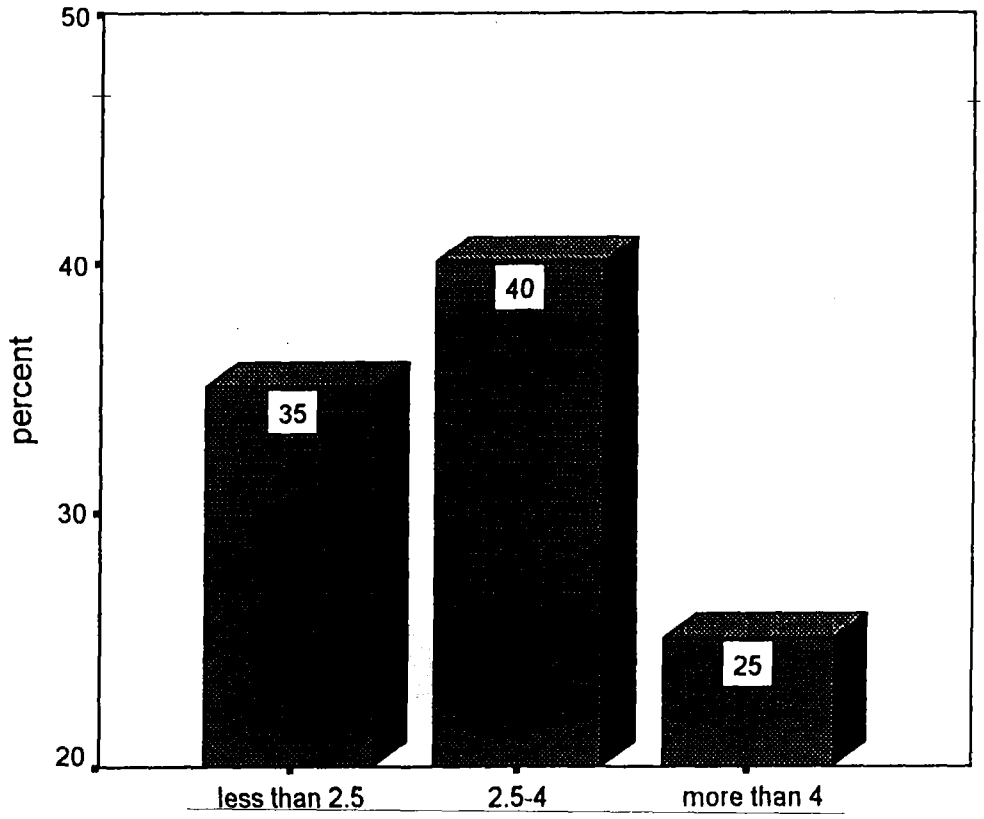

Fig.(13): Distribution of outcome according to presence of apparent malformation

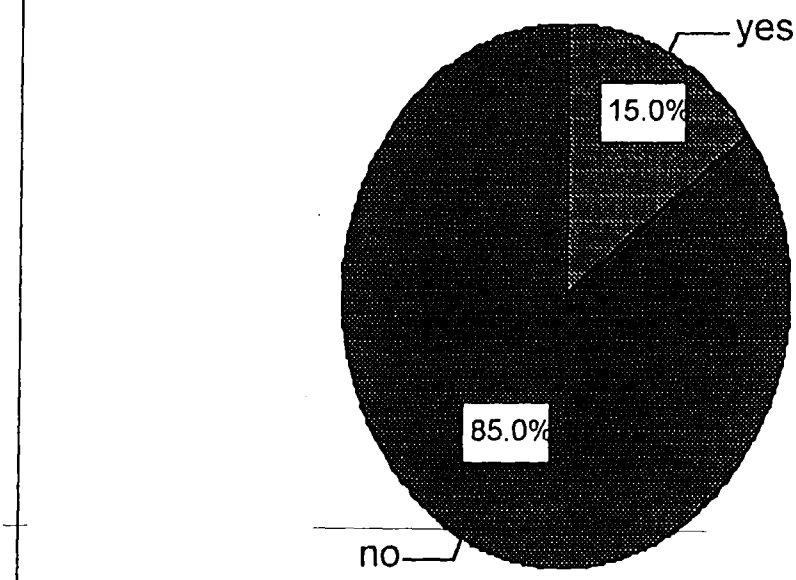

Table (11): Percentage distribution of outcome according to different apparent malformation

APPARENT MALFORMATION	PERCENT
Anencephalus	26.6
Hydrocephalus	21.1
Encephalocel	6.6
Hydrops fetalis	26.6
Achondroplasia	20.1
Total	**100**

Fig.(14): Distribution of newborns according to Special management provided

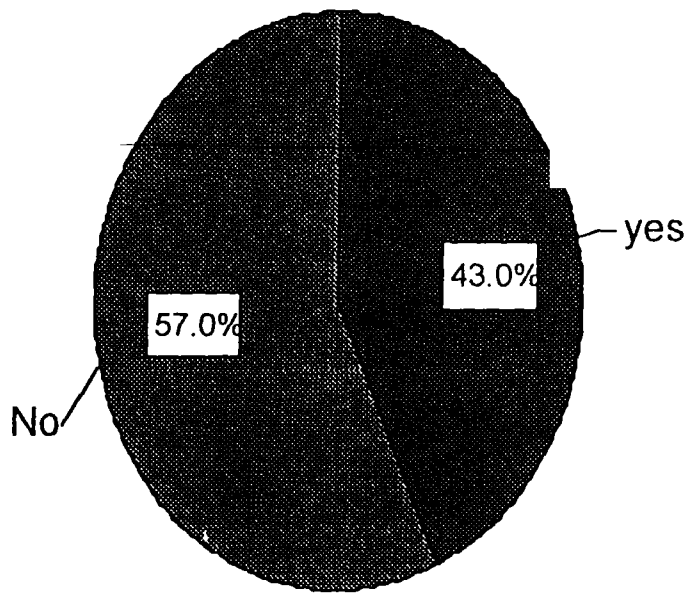

Table (12): Percentage distribution of newborns according to types of special management provided

TO TYPES OF SPECIAL MANAGEMENT	PERCENT
Incubatered	9.3
Correction of Hypoglycemia	28.0
Surgical correction	14.0
intubation	11.5
Blood exchanges	4.7
Observation in the Nursary	32.0
Total	**100**

Deepest pool of Amniotic Fluid volume by U\S

DEEPEST POOL OF AMNIOTIC FLUID VOLUME BY U\S	WEIGHT OF THE OUTCOME/KG			TOTAL
	less than 2.5	2.5-4	More than 4	
8.1-12	26.6%	46.8%	26.6%	100.0%
12.1-16	73.3%	13.3%	13.3%	100.0%
More than 16	50.0%	16.7%	33.3%	100.0%
Total	**35.0%**	**40.0%**		**100.0%**

Chi-Square test

	VALUE	D.F	SIG
Person chi-square	13.751	4	0.008
N of valid cases	100		

CHAPTER

FOUR

DISCUSSION

Polyhydramnios is one of the alarming signs of pregnancy which neccesitates a proper assessment in specialised center as it is associated with high perinatal mortality and morbidity.

This is a prospective discriptive study of patients with polyhydramnios who had been clinically suspected and referred to the fetomaternal unit in Soba university hospital in the period from 1/4/2001-1/ 6 /2002. The total number of patients in this study was (100), all of them were confimed cases of polyhydramnios by ultrasound.

In this study(38%) of the patients were in the age group 37-41 years, this is can be explained by the increased risk of congenital malformation, diabetes and multiple pregnancy with the advanced maternal age.

In relation to parity, most of the patients(42%) had more than 5 -childern and this is related to the association of high parity and age as risk factors as had been mentioned in the literature [5].

In this study patients who had past obstetrical history of still births were(6%), neonetal deaths were (2%), abortion were (12%), and (2%) had a previous polyhydramnios. This showed the

association between bad obstetrical history and polyhydramnios, this is also had been reported in study done in Paris [3].

In relation to gestational age at diagnosis in weeks most of the patiens (78%), were diagnosed after 28 weeks and only(22%) were diagnosed before that, this indicates that polyhydramnios accumalates gradually and only noticed after 30 weeks while in few cases polyhydramnios accumalates quickly and diagnsed earlier as mentioned in the literature [5].

Regarding the personal past history and family history of possible associated risk factors, no such risk factors were found in (74%) and(64%) of the cases respectively this can be explained by the fact that, most of the cases were idiopathic as reported in two studies from Paris and Switzerland 3,4. Also the same had been mentioned in literature [2].

Eight percent of the patients in this study were Rhesus negative, half of them (4%) were complicated by hydrops fetalis, this showed the high prevalence of isoimmunization.This result is higher than that obtained from the study in sweizerland (1.3%) [4].

Seventy nine percent of the patients had mild ployhydramnios and this is explained by that, most of the cases were idiopathic which is usually mild.

In the current pregnancy it was found that(39%) of the patients had associated risk factors, (16%) of the total cases were diabetic, (15%) had structural congenital malformation, these results are simillar to that from the study done in sweizerland [4].

Regarding the complication arising during the current pregnancy it was found that (65%) of the patients had no complication and this can be explained by that, most of the cases were idiopathic and mild.

Preterm labour represented the most frequent complication (38%).

Regarding the management, (65%) of the patients did not require antenatal treatment, and this can be explained by that most of the patients were without complication.

In relation to the mode of delivery (44%) of patients delivered by caesarean section. and this high rate of caesarean section could be explaind by the fetal macrosomia in diabetes, and possible complication that might arise such as footling breech, cord prolapse, abruptio placentae and larger for gestational age babies even in absence of identifiable cause, as reported in study from United States [21].

In relation to the outcome, in our study (6%) were stillbirths this is almost like that obtained in Paris where stillbirths were (7.5%)

Regarding the weight of outcome (25%) were had birth weight more than 4 kg, in study done in United States, the macrosomic babies account for (36.3%) and this can be explained by the association between macrosomia and polyhydramnios.

Most of the outcome (85%) had no apparent congenital malformation and this is because most of the patients had no risk factors in their current pregnancy.

Of the (15%) with congenital malformation, neural tube defect accounted for (53%), this concides with that had been mentioned in the literature [7].

Fourty three percent of the newborns required special management.

the perinatal mortality was found to be (19.2%) and this confirmed the high association between polyhydramnios and perinatal mortality.

Conclusion

The conclusion of this prospective discreptive study which is aiming to find out the associated risk factors and the outcome in cases of polyhydramnios is as follows:-

- The occurance of polyhydramnios is directly related to advanced maternal age.
- polyhydramnios is more common in grandmultiparae.
- There is association between bad obstetrical history and polyhydramnios.
- Most of the cases had been diagnosed after the gestational age of 28 weeks, and most of them had idiopathic and mild polyhydramnios.
- Diabetes and congenital malformation were the most frequent associated risk factors of polyhydramnios.
- Preterm labour represented the most frequent compliaction of polyhydramnios.
- Caesarean section as a mode of delivery was found to be higher in cases of polyhydramnios than the international rate.

- There is a high incidence of fetal macrosomia in cases of polyhydramnios even in non diabetic mothers.
- Neural tube defect and intestinal obstruction are the commonest congenital malformation associated with polyhydramnios.
- polyhydramnios is associated with high perinatal mortality and morbidity rate.

should be controlled preconceptionally and during the antenatal period.

- Rhesus isoimmunization shoud be prevented by admistering prophylactic AntD to all RhD negative women during pregnancy when there is an increased risk of fetomaternal bleeding and within the first 72 hours postnataly to women who give birth to RhD positive babies.
- In mothers with polyhydramnios associated with fetal congenital malformation which needs immediate surgical interference, delivery should be attended by peadiatric surgeon.
- Amnioreduction should be carried out in a specialised unit under aseptic condition and ultrasound guidence.

- prophylactic steriods should be administered as preterm labor is a possible major complication of polyhydramnios.
- Use of Indomethacin for all cases with previous preterm deliveres.

- All patients presented with unexplaned hydramnios must be investgated for TORCH (toxoplasmosis, other viruses, rubella, cytomegalovirus, herpes).

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Questionnaire

Polyhydramnios in Sudanese Women, Associated risk factors and outcome

Serial number.....Date.....

1- Name: .....

2- Age: .....

3- Parit:

a)Nullipra ☐ b) I-4 ☐ c) 5 and above ☐

4- Obstetrical History of previous outcome.

a) alive ☐ b) Stillbirth ☐

c) Neonatal death ☐ d) Abortion ☐

5- Gestational age at diagnosis in weeks:

a) less than 28 ☐ b) 28-32 ☐

c) 33-37 ☐ d) 38-42 ☐

6- Personal past history:

a) hypertension ☐ b)diabetes ☐ c) multiple prgnancy ☐

d) congenital malformation ☐ e) others ☐ f) not applicable ☐

7) Family history:

a) hypertension ☐ b)diabetes ☐ c) multiple prgnancy ☐

d) congenital malformation ☐ e) others ☐ f) not applicable ☐

8- Rh-factor:

a) Positive ☐ b) negative ☐

9- Deebest pool of Amniotic fluid volume by ultrasuond.

a) 8.1-12 ☐ b) 12.1-16 ☐ c) 16 and obve ☐

10- Posible causes of polyhydramnios in current pregnancy:

a) Diabetes b) Rh-isoimmunization c) congenital malformation

d) multiple pregnancy e) others f) no posible cause

11- Antenatal complication of polyhydramnios:

a) preterm labor ☐ b) PROM ☐ C) acute pain ☐

d) IFUD ☐ e) others ☐ f) not complication ☐

12- Antenatal management of polyhydramnios:

a) control of diabetes ☐ b) amnioreduction ☐

c) Indometazin ☐ d) others ☐ e) No-tretment ☐

13- Gestational age at delivery:

a) less than 28 ☐ b) 28-32 ☐ c) 33-37 ☐ d) 38-42 ☐

14- Mode of delivery:

a) Vaginaly ☐ b) EM/CS ☐ C) EL/CS ☐

15- Indication of different modes of deliveries: .....

16- Status of outcome.

a) alive ☐ b) FSB ☐ C) MSB ☐ D) END ☐

17- Sex of outcome.

a) Male ☐ b) Female ☐

18- Weight/kg of outcome.

a) less than 2.5 ☐ b) 2.5-4 ☐ c) more than 4 ☐

19- Any apparent malformation of outcome.

a) yes ☐ b) No ☐

20- If yes mentioned: .....

21- Any special management for the outcome:

a) yes ☐ b) No ☐

22- If yes mentioned: .....