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IRRADIATION THERAPY OF INFLAMMATION

Inflammatory diseases of infectious etiology are today treated with increasing effectiveness by anti-biotics and chemical agents. A considerable variety of organisms is susceptible to their action, in varying degrees. For those infections in which the organism is more or less resistant to these agents, and for the many inflammatory processes of non-infectious origin, X-ray therapy is a valuable means of attack, alone or as adjuvant to surgical and drug therapy. There are no known contra-indications to the use of X-rays in infectious diseases, nor any incompatibilities with the chemotherapeutic and anti-biotic agents.

It may be well to point out the harmlessness of X-radiations when administered with due regard for the duration of the inflammatory disease, and with proper dosage, fractionation and spacing. The anti-inflammatory dosage is a very small fraction of the cancericidal dosage of X-radiation, and none of the occasionally unpleasant side-effects of the latter are, therefore, seen. As a rule, the more acute the process, the smaller is the therapeutic dose employed, and the more rapid is the response, with either resolution or early fluctuation. Other factors influencing the quantity of irradiation are the site of pathology, the type of surrounding tissues, ease of drainage, and the degree of regional vascularity. The quality of radiation is a matter of judgment by the radiologist, and must vary with the depth of the disease being treated.

The non-specificity of the X-rays in therapy is often an actual advantage, and is due to their mode of action. Their effect is to aid rapid localization and lysis of leukocytes in the inflamed area under treatment. An intense concentration of anti-bacterial enzymes,

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and possibly antibodies, is released thereby. The action in non-infectious inflammation is apparently due to stimulation of absorption of the inflammatory elements locally, probably due to favorable alterations in the vascular bed.

The wide variety of non-neoplastic conditions which can be affected favorably by irradiation is well attested in the literature, and borne out in the experience of the qualified physician who practices therapeutic radiology. The amenable diseases range from such acute infections as parotitis, mastitis, erysipelas, mastoiditis, carbuncles, and deeper-seated infections to the more chronic conditions of viral or of unknown origin, such as the spondylitis of Marie-Strumpell, herpes zoster, "virus" radiculitis, tuberculous adenitis (including the abdominal type), the diphtheria carrier state, etc. There is evidence to indicate that radiation therapy causes a definite decrease in length and severity of the morbidity in atypical pneumonia of presumed virus etiology. In gas gangrene, too, radiation therapy has been used with gratifying effect in reduction of morbidity and mortality, fortifying the effect of sulfonamides and penicillin. A recent report of the development of B.welchii infection at the site of long continued injection of penicillin may indicate an inherent or acquired resistance of at least some strains of the organism to that agent.

Another field of usefulness in radiation therapy is the group of nonspecific and post-traumatic arthropathies and peri-arthropathies, including such disabling affections as peritendinitis (with and without calcification), bursitis, and some of the arthritides of the spinal and peripheral joints. Complete relief may be afforded, or at least a degree of relief from pain and spasm to a point where orthopedic corrective measures may be effectively adopted.

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A claim for one-hundred percent effectiveness for radiation therapy in all inflammatory conditions can no more be made than for any other therapeutic agent. The proven merit of this means of treatment, its ease of application, its availability in skilled hands in most populated centers today, and its usefulness even as a supportive measure with other agents indicate that it be more widely employed, however.

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THE ORAL TRANSMISSION OF MURINE TYPHUS IN HUMANS*

Morris Pollard, D. J. Wilson, H. R. Livesay and J. C. Woodland

The transmission of murine typhus by arthropods has been demonstrated so frequently that one may consider this as the principal method of transmission from mammal to mammal. Many reports have demonstrated that this disease can be transmitted experimentally in mammals through infestation with infected fleas (Xenopsylla cheopis) and mites Liponyssus bacoti; and cases of the disease in humans are frequently associated with exposure to such arthropods. The disease has been induced in mammals through the respiratory tract, by Castenada with such success that the resulting infected lungs are considered suitable either for vaccine or for complement fixing antigen.

The possibility of acquiring typhus infection through the alimentary tract has caused some controversy among epidemiologists and typhus control personnel. Violle and Suntel offered experimental data on attempts to induce murine typhus infection in animals through their alimentary tracts. Nicolle et.al. report that eight monkeys were fed infected brain emulsions and that four of these were immune when challenged with typhus. It should be pointed out, however, that precautions were not taken against a possible inhalation of the agent, and that no actual case of the disease developed with the ingested material.

*From the Eighth Service Command Laboratory, Fort Sam Houston, Texas. Received for publication July 6, 1946.

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Since murine typhus has a reasonably low fatality rate in young humans, it was considered more direct and conclusive to attempt the oral transmission of this disease with human volunteers.

Experimental

Six young healthy male volunteers were selected for the feeding experiment. They were questioned at length concerning previous illnesses and none professed any history suggestive of an arthropod-borne disease. Their serums were examined prior to the experiment and all were found to be negative for typhus by the complement fixation test and negative for significant Proteus OX19 agglutination titers.

The infected food was in the form of chick embryo yolk sacs which were infected with the Wilmington strain of murine typhus. Cases I, II and III were fed infected yolk sac material which was collected on the same day (Pool 1010). Cases IV, V and VI were fed infected yolk sac material which had been stored in the dry ice chamber for four days (Pool 1005). The infectious material was transmitted in a dry ice chamber to an institution located two hundred miles from the laboratory.

Just prior to the feeding the infected yolk sacs were emulsified in saline and were transferred to gelatin capsules (no. 00) with a syringe and needle. The filled closed capsules were carried to an adjoining room and placed into the mouth of the volunteer. Four cc of the material was thus swallowed with

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tap water and the men were subsequently observed, to preclude the possibility of regurgitation. The feeding experiment was performed four hours following lunch and two hours prior to the following meal. All six of the volunteers were then isolated in a separate room of the institutional hospital and were observed at least three times daily during the succeeding four weeks. A serum specimen was collected from each volunteer at five day intervals. Each specimen was examined for typhus complement-fixing antibodies and for Proteus OX19 agglutinins.

After the volunteers had eaten the test material, 2.0 cc. of the yolk sac material from each lot was injected intraperitoneally into four guinea pigs and the material was also passaged into additional chick embryos. This material was also examined for bacterial organisms with thioglycollate broth and blood agar plates.

Results

Subsequent clinical and serological examinations of cases I, II, III, IV and V failed to reveal any evidence of typhus infection as a result of the feeding experiment. All individuals remained normal.

Eleven days after the feeding experiment Case VI suddenly developed a severe headache, generalized malaise, and insomnia. A generalized maculo-papular rash developed on the fourth day of illness and was evident for six days thereafter. A febrile temperature was recorded for eleven days and defervescence came

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by crisis (Chart I). Sedatives were administered early in the course of the disease and may explain the vacillations in temperature. No alteration in blood count could be detected during the course of the disease. This volunteer developed a typical clinical infection of typhus fever; and serological examinations demonstrated rising titers for Proteus OX19 agglutinins and for typhus complement-fixing antibodies, the latter attaining a peak titer of 1:2560 (Chart II).

The control guinea pigs developed typical febrile reactions and orchitis (Chart III). The short incubation period is observed frequently with yolk sac inoculums. All developed complement fixing antibodies for typhus.

Discussion

We realize that flea feces or rat urine would be a more probable source of natural human infection; however, we were interested primarily in having a sufficient quantity of virulent material for all phases of control inoculations for the experiment, therefore, we had to resort to infected chick embryo yolk sacs. The dosage administered to the volunteers was probably very large in comparison with possible natural dosage by mouth.

One man out of six developed typhus fever as a result of eating typhus contaminated food. This individual (Case VI) was questioned rather extensively regarding any gastro-intestinal symptoms which might be referable to a lesion through which the agent possibly gained admission to the body. No.

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evidence which might be adduced to such a lesion could be elicited. Whatever peculiarity this man had which might have made him susceptible, the fact remains that an individual, who was considered clinically normal, contracted the disease by way of the digestive tract. The institution at which this experiment was conducted was a newly constructed modern building in which there had been no history of typhus among the inmates. All of the volunteers had been in this institution at least three months prior to this experiment.

It may be possible that certain naturally-occurring cases of murine typhus develop as a result of consuming food contaminated either with infected urine or flea feces. This might explain certain cases of the disease where direct exposure to a focus of infection is uncertain.

Conclusion

One human volunteer of six acquired an action typhus fever infection by ingestion of typhus infected food.

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8. d. Chart I. Temperature, pulse and skin rash of experimental typhus fever volunteer number VI.

Chart II. The serological reaction for typhus fever as manifested by volunteer number VI.

Chart III. Febrile reactions of typhus fever inoculated control guinea pigs.

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