It is doubtful if one can ever on clinical grounds determine which migrainous patients have an intracerebral aneurysm. The most suspicious features, short of the all too obvious hæmorrhage, seem to be the same site for the pain in each attack and fixed signs of cerebral deficit.

SUMMARY

Six cases of minor hemiplegic migraine are presented. The symptoms and nature of the syndrome are compared with the symptomatology of cerebral angiomas.

When the vasospasm responsible for the aura of migraine extends from the visual pathways to the Rolandic cortex, there is no clinical indication that the disease is other than migraine.

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RÉSUMÉ

L'hémicranie classique comporte l'aura, la douleur et les symptômes associés. Parmi ceux-ci la nausée, les vomissements, la diplopie, la photophobie, la sialorrhée, l'assoupissement ou le sommeil incoercible sont bien connus. L'aura peut assumer la forme d'une aire de picotements, d'engourdissement et de faiblesse musculaire. Cette faiblesse peut s'étendre aux membres. Même a-t-on déjà rapporté des reliquats permanents d'atteinte cérébrale (thrombose, entre autres) à la suite d'attaques de migraine. Ces formes de migraines dites accompagnées entraînent une paralysie transitoire d'une durée de deux à trois jours. On a proposé le terme de *migraine hémiplégique majeure* pour décrire l'affection retracée chez deux ou trois générations successives, par opposition à la forme non familiale ou *mineure* dont l'auteur rapporte six cas dans le présent article. L'aura et la douleur seraient produites par des mécanismes séparés et intéresseraient des régions différentes, L'aura viendrait de la constriction des vaisseaux du système de la carotide interne, alors que la douleur découlerait de la dilatation et de la perméabilité des vaisseaux appartenant surtout au système carotidien externe. Que le spasme vasculaire incriminé dans l'aura s'étende des structures de la perception visuelle à la zone rolandique ne forme pas pour autant une indication clinique que la maladie soit autre que la migraine.

TULARÆMIA IN BRITISH COLUMBIA

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THE RECENT OCCURRENCE of a case of tularæmia originating near Kamloops has aroused our interest in this disease and stimulated us to record the cases known to have occurred in our province.

HISTORICAL[1]

McCoy described a "plague-like disease in rodents" in California in 1911. The causative organism was isolated by McCoy and Chapin in 1912 and given the name of *Pasteurella tularensis* because it had first been isolated in Tulare County, California. The disease is found in many different rodents including rabbits, hares, ground squirrels, and water rats. Animals other than rodents vary in their susceptibility, and domestic animals, except domestic rabbits and sheep, are largely resistant. Outbreaks of epizootic proportions have been reported in range sheep in the western United States and Alberta.[2] Transmission from animal to animal is chiefly through the medium of bloodsucking insects, particularly ticks.

The disease in man was given the name of tularæmia by Francis in 1919. Man may contract the disease by several different routes. The most common mode of infection is for the organism to enter an abrasion on the hand of a person engaged in handling or skinning an infected animal. Pelts of infected animals may harbour the organism for considerable periods. The infection may be transmitted to man by the bite of an infected insect or through crushing an infected insect on the skin. The infection easily enters the conjunctiva, to which it is presumably carried by contaminated fingers. It is well established that *P. tularensis* is able to penetrate the intact skin when rubbed on it. The infection may be transmitted by the ingestion of the insufficiently cooked flesh of an infected animal, and instances of the transmission of the organism in drinking water have been recorded.

Cases of tularæmia in humans have been reported from all 48 states of the United States and from Canada, Japan, Russia, Norway, Sweden and other countries. California and the northwest United States have reported the greatest number of cases, and many have occurred in Russia from the pelting of water rats. In the years from 1922 up to the introduction of streptomycin in 1945, the United States Public Health Service reported 19,208 cases, and 1432 deaths were attributed to the disease. This would give a case mortality of about 7.4% before streptomycin became available as a specific treatment.

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TULARÆMIA IN CANADA

The first case of tularæmia diagnosed in Canada was reported by McNabb[3] from Timmins, Ontario, in 1930. We have made no attempt to ascertain the number of cases which have occurred in Canada. Enquiries made by the Dominion Entomological Laboratory, Kamloops, in 1939 revealed that the number of cases reported from 1932 to 1939 included: Nova Scotia two, Quebec two, Ontario six, Alberta 16 and British Columbia two; none were reported in Prince Edward Island, New Brunswick, Manitoba and Saskatchewan.

TULARÆMIA IN BRITISH COLUMBIA

Including a case which occurred near Kamloops in 1956, we have been able to find two British Columbia cases which have been reported in the literature and four cases which have not been reported previously, making a total of six known cases in British Columbia from 1934 to 1956. We feel sure that many additional cases have occurred. Two other highly suspicious cases have come to our notice but the diagnoses were not definitely confirmed and they are not included in our compilation.

It should be noted that Ootmar[4] reported a case in 1931 under the title of "B.C. Tularæmia Case Report". This was an account of a retrospective serological diagnosis of a case in a man who had been bitten by a deerfly in August 1929. Further follow-up by the Dominion Entomological Laboratory definitely established that this case did not originate in British Columbia, as the patient suffered his bite in Banff, Alberta.

TABLE I.—CASES OF TULARÆMIA IN BRITISH COLUMBIA, 1934 - 1956

Date	*Sex*	*Age*	*Location*	*Mode of infection*	*Agglutination titre*	*Outcome*
1934	Female	12	Cherry Creek	Scratch from a cat.	1:320	Recovered
1935	Male	18	Williams Lake	Tick bite.	——	Recovered
1944	Male	45	Williams Lake	Skinning a coyote.	1:640	Died
1953	Male	56	70 Mile House	Skinning hare.	1:1280	Recovered
1954	Male	43	Kamloops	Laboratory infection (conjunctival).	1:200	Recovered
1956	Male	29	Louis Creek	Skinning a rabbit.	1:640	Recovered

REPORT OF A RECENT CASE

T.C., a 29-year-old Italian-born caterpillar tractor operator, presented himself on October 13, 1956, with chills and fever and pain in the right elbow and right axilla of about 48 hours' duration. He had a small healing abrasion on the right middle finger.

He reported that one week before (October 6) he suffered a slight abrasion on his right middle finger. The same day at Louis Creek, 35 miles north of Kamloops, he noticed a rabbit on the edge of the field where he was working. The rabbit was not very active and stayed in approximately the same location for several hours. He shot it with a .22 rifle, took it home, skinned it and ate some of the meat after it had been boiled and roasted.

His symptoms continued and increased over a two-week period in spite of satisfactory healing of the abrasion and in spite of treatment for a week with Sulfadine and for another week with Achromycin. He was admitted to the Royal Inland Hospital, Kamloops, on October 27 with greatly enlarged and very tender right epitrochlear and axillary glands. The finger abrasion was covered by a dry scab and there was no surrounding inflammation. His temperature was 104.6° F. There were no other relevant physical findings, but he was flushed and agitated and complained of considerable pain over the enlarged right epitrochlear gland, which was firm and tender and showed extensive surrounding induration.

The laboratory findings were: hæmoglobin level 83.4% (12.1 g. %), red cell count 4,320,000 with normal red cell morphology. White cell count 12,300 with neutrophils 27%, band cells 18%, lymphocytes 34%, monocytes 13%, metamyelocytes 3%, myelocytes 1%, promyelocytes 4%. Sedimentation rate (modified Westergren) 10 mm. in one hour. Urine negative. A heavy growth of *Staphylococcus aureus* was obtained from the abrasion on the finger.

He was given large doses of penicillin on admission. Blood agglutination against *P. tularensis* was reported positive in a dilution of 1:160 on November 1, 26 days after infection. Treatment was then changed to 0.5 g. streptomycin twice daily. Four days later the agglutination titre had risen to 1:640. His temperature became normal on November 6 (tenth hospital day) and remained normal. Enlargement and tenderness of glands gradually subsided. There have been no relapses to date and recovery appears to have been complete.

The incubation period was four days and the duration of the illness 31 days.

PREVIOUSLY REPORTED CASES

October 1934.—A case originating at Cherry Creek, 12 miles west of Kamloops, was reported by Mr. T. K. Moilliet.[5] The patient was a 12-year-old girl, infected by a scratch from a cat which had been fed on a dead rabbit. She was under the care of the late Dr. R. W. Irving of Kamloops and made a good recovery after prolonged illness. The agglutination titre was 1:320.

September 1944.—Dr. K. K. Pump[6] reported a case occurring at Williams Lake in a 45-year-old man, who became ill after skinning a coyote. As coyotes are resistant to tularæmia, the presumption is that the coyote had been feeding on infected rabbits. The case showed an agglutination titre of 1:640 and ended fatally on the 20th day of illness.

CASES NOT PREVIOUSLY REPORTED

August 1935.—An 18-year-old boy was bitten by a tick and developed the symptoms of tularæmia. He

Canad. M. A. J.
Jan. 1, 1958, vol. 78

was treated by Dr. C. E. McRae of Williams Lake and Dr. H. A. DesBrisay of Vancouver and recovered. Though no record of the agglutination titre is available, the case would appear to have been well established.[7]

September 1953.—A 56-year-old man contracted the infection through a prick on his finger while skinning a hare at 70 Mile House. He was treated by Dr. L. C. Capling[8] of Haney, B.C. After an initial illness of six days he recovered on treatment with aureomycin but suffered three relapses in the succeeding three months, finally making a good recovery. The agglutination titre reached 1:1280.

May 1954.—A 43-year-old laboratory worker from the Kamloops Branch of the Laboratory of Hygiene was treated by Dr. J. H. Harland.[9] The patient himself suggested the possibility of tularæmia as he had been handling cultures of the organism. He had an inflammation of the conjunctiva but no lymphadenopathy. The agglutination titre was 1:200 and he recovered promptly under treatment with Achromycin and streptomycin.

October 1956.—This case in a 29-year-old man who contracted his infection at Louis Creek is reported in detail above.

Discussion

Tularæmia is not a common disease in British Columbia, but it is one which should always be kept in mind. Humphreys and Campbell[10] report that *P. tularensis* has been recovered from ticks collected from widely scattered areas of southern British Columbia. Both the common wood tick (*Dermacentor andersoni* Stiles) and the rabbit tick (*Hæmophysalis leporis-palustra* Packard) have been found to harbour the organism. A reservoir of infection is present in British Columbia, and all persons should be aware of this and should exercise great care in the handling of wild rabbits, especially rabbits which show signs of sickness or lack of normal agility.

The incubation period of tularæmia in man is from one to 10 days with an average of about 3½ days. The onset is sudden with headache, chills, body pains, vomiting and fever. The primary lesion on the skin or conjunctiva usually proceeds to ulceration. The regional lymph glands are usually enlarged and inflamed and may form abscesses or ulcerate. The fever, when not treated by streptomycin, usually persists for three or four weeks and convalescence is slow.

Diagnosis is usually confirmed by agglutination tests but it is also often possible to recover the organism from the skin lesion or the glands either directly or after passage through a guinea-pig.

Streptomycin administration is a specific treatment and usually leads to prompt recovery.

Summary

A recent case of tularæmia occurring in British Columbia is reported. A further five cases known to have occurred in this province are reviewed and are summarized in the accompanying table (Table I).

We would like to express our appreciation of the assistance extended to us by Mr. J. D. Gregson and his staff at the Veterinary Entomology Section, Science Service, Entomology Division, Canada Department of Agriculture, which is referred to above as the Dominion Entomological Laboratory, Kamloops.

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Résumé

Les voies d'infection humaine de la tularémie sont soit une solution de continuité des téguments, soit la piqûre d'un arthropode infectieux, ou même l'écrasement et le frottement de cet insecte sur une peau intacte, le contact de la conjonctive par des doigts infectés, ou l'ingestion d'eau ou de nourriture contaminées par des rongeurs malades. Avant l'ère de la streptomycine la mortalité dans cette affection était de 7.4%. Le premier cas rapporté au Canada fut découvert en 1930.

Les faits cliniques d'un opérateur de niveleuse qui avait mangé de la viande de lièvre malade sont donnés dans le texte. En plus d'une fièvre de 104.6° F. (40.3° C.), cet homme présentait une adénopathie douloureuse épitrochléenne et axillaire droite qui faillit masquer le diagnostic à cause d'une éraflure suppurée du médius droit de laquelle on isola du staphylocoque blanc. Le séro-diagnostic pour *Pasteurella tularensis*, cependant, montra un taux d'agglutinines de 1:160, qui quatre jours plus tard atteignit 1:640. L'instauration du traitement à la streptomycine (0.5 g. deux fois par jour) marqua le tournant de la maladie, dont l'incubation avait été de quatre jours et la durée totale de 31 jours. Cinq autres cas sont présentés de façon succincte. Même si la tularémie ne s'observe que rarement au Canada, il faut se rappeler qu'il en existe un réservoir en Colombie canadienne.

EFFECT OF X-RAY THERAPY ON GASTRIC ACIDITY AND 17-HYDROXYCORTICOID AND UROPEPSIN EXCRETION

Rider and his colleagues (*Ann. Int. Med.*, 47: 651, 1957) report that in 14 patients, x-irradiation of the stomach for the treatment of peptic ulcer resulted in a clinical remission in 12 and in a reduction in gastric acidity in all. Uropepsin excretion was consistently and significantly increased during or immediately after x-ray therapy and subsequently fell approximately to control levels. This rise was much greater in patients in whom the secretion of hydrochloric acid was markedly reduced. An occasional patient showed a marked persistent reduction. The rise in uropepsin excretion after treatment suggests that radiation causes an alteration in the function or permeability of the chief cells, leading to a change in the direction of diffusion of pepsinogen in favour of the blood stream. The 17-hydroxycorticoid excretion rose during and after x-irradiation and subsequently fell towards control levels, although a few patients maintained a persistent elevation. This effect may be part of a general stress phenomenon. An early marked rise in uropepsin, followed by a marked fall in gastric acidity, may enable one to predict early which patients will respond best to x-ray therapy.