HEPATIC DISTOMATOSIS AND INFECTIOUS CANINE HEPATITIS IN NORTHERN MANITOBA

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A REPORT received in November 1959 from a Game Branch Officer in Cranberry Portage, pointed out a distemper-like disease was quite prevalent among dogs and also probably among foxes, since the population of the latter had decreased greatly from that of preceding years. Towards the end of December 1959, and the beginning of January 1960, rumours were heard that sleigh dogs were dying in great numbers in Northern Manitoba (Figure 1). These rumours were investigated. However, as we could find nothing definite, no action was taken at that time. On January 29, 1960, the Superintendent of the Department of Indian

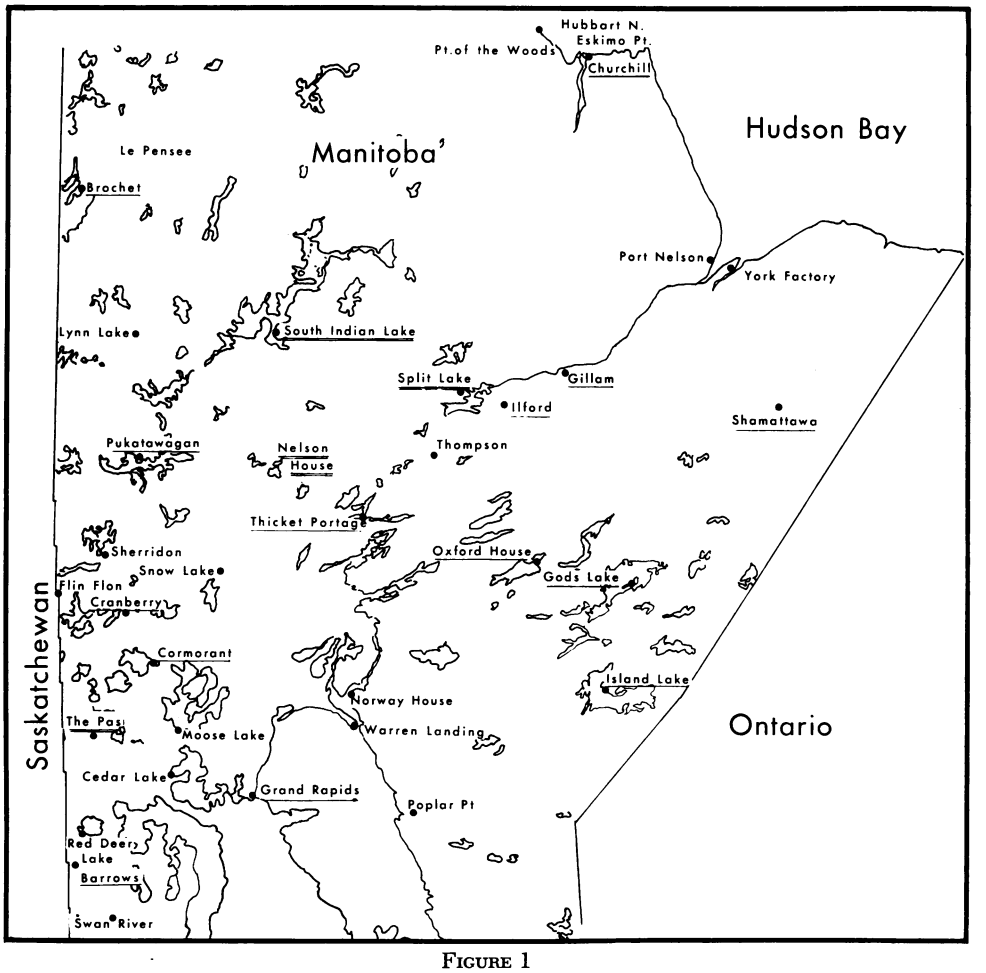

FIGURE 1
Map of Northern Manitoba. Affected settlements are underlined.

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Affairs in The Pas, reported the seriousness of an outbreak of a distemper-like but unknown disease, and requested our assistance to diagnose and eventually treat or in other ways deal with this unknown disease affecting the dogs in Northern Manitoba. Accompanied by the Superintendent of the Department of Indian Affairs, I flew to Nelson House and South Indian Lake, where the situation appeared to be the most serious, and was causing economic hardship to the Indians. Dogs are the sole means of transportation and the Indians depend upon trapping and hunting, which involves a great deal of travelling, for their livelihood; so one can understand why the Indians were deeply concerned with the death of their dogs.

As soon as we arrived we informed the Indians of the object of our visit, and we were shown dogs in varying stages of the disease and carcasses of some that had died in January. The owners reported that the dogs which had died had been sick for a period of from three to four days to as long as one month. The first thing they noticed was that the dogs were listless, a few hours or days later they had diarrhea, stopped eating, became thinner and thinner, and then died. Some cases had improved suddenly for two or three days, but death promptly followed this apparent recovery.

A great number of dogs were examined and the following symptoms noted: temperature 91.5–106.5°F, muscular tremors especially of the hind quarters, partial to complete paralysis, conjunctivitis, diarrhea; nervous symptoms varied from twitching of face muscles to epileptiform convulsions which were noted in five dogs only. A tentative diagnosis of infectious canine hepatitis and/or distemper was made pending the completion of laboratory examination of postmortem specimens. I destroyed several dogs that were moribund and conducted autopsies. The most striking lesions were found in the liver, which in most cases was greatly enlarged and covered with little abscesses. The incised abscesses and bile ducts were filled with small parasites that were tentatively identified as the trematode Metorchis conjunctus, causative agent of hepatic distomatosis or liver fluke disease (Figure 2).

Suitable specimens of the different organs and of the parasite were secured and forwarded to the Animal Disease Research Institute, Hull, Quebec. The pathologist's report on a nine-year-old dog indicated that the liver was greatly enlarged and had a hobnailed appearance. The bile ducts were greatly enlarged, with thickened walls, and contained large numbers of flukes, identified as M. conjunctus. The pancreatic ducts were also similarly enlarged and plugged with flukes. The stomach contained only what appeared to be deer hair, spruce twigs and branches. The intestines appeared normal but contained a large number of

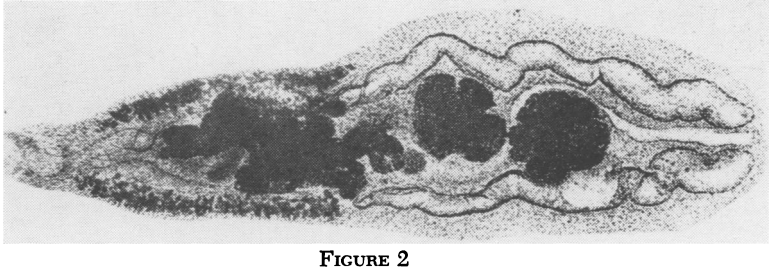

FIGURE 2
Photomicrograph of a stained specimen of Metorchis conjunctus.

tapeworms. Except for calcification of the calyces the kidneys were normal, as were the heart and spleen.

Microscopic examination of the liver showed intranuclear inclusion bodies in the hepatic cells, similar to those seen in infectious canine hepatitis. Extensive fibrosis and proliferation of the epithelium was noted in the bile and pancreatic ducts. Distortion of the architecture of the liver as a result of fibrosis was striking. The lungs were congested. Calcification of the renal calyces appeared to be long standing and was not accompanied by any inflammatory reaction. Inclusion bodies of canine distemper could not be demonstrated in the bronchial epithelium or in the epithelium of the renal pelvis. The diagnosis was infectious canine hepatitis plus a heavy infestation of Metorchis conjunctus.

Three other specimens showed similar lesions. All were infested with Metorchis conjunctus, one extremely heavily. Two animals had intranuclear inclusion bodies indicative of infectious canine hepatitis in the hepatic cells. The other dog had acute lobar pneumonia as well as the parasites. All were negative for Negri bodies; experimental animals were inoculated with brain material.

Another specimen consisted of the complete thorax and abdomen of a dog. Gross examination was essentially similar to that of the first dog. In addition the abdominal cavity contained peritoneal fluid in excess of 950 cc. Microscopic examination failed to reveal the presence of inclusion bodies of either infectious canine hepatitis or canine distemper but the liver damage due to the parasitic infestation was extreme in this instance.

Diagnosis

Infectious canine hepatitis and extremely heavy infestation with Metorchis conjunctus.

Comments

It was felt that possibly all dogs may have suffered from infectious canine hepatitis but because of the heavy infestation of the liver parasite Metorchis conjunctus, the dogs may have succumbed before the development of inclusion bodies.

When the field diagnosis was confirmed by the laboratory, the Department of Indian Affairs' officials were advised that a vaccination program for distemper and ICH would be beneficial. They were further advised that if they wished to purchase a supply of distemper and ICH vaccine, the Health of Animals Division would be pleased to co-operate with them by administering part of the vaccine and by training officials of the R.C.M.P. and the Department of Indian Affairs in the technique of vaccination. In the more southern and larger settlements, such as The Pas, Manitoba, where many of the dogs are not fed on fish, there have been some very serious outbreaks of infectious canine hepatitis during the past winter. I have conducted approximately 100 postmortems on dogs, throughout the north during last winter and would estimate that about 90 per cent showed typical lesions of infectious canine hepatitis.

Disscussion

It is interesting to note that Metorchis conjunctus was first reported in Manitoba in 1934 by Allen & Wardle, who suggested the name Parametorchis manitobensis N. Sp. since it represented a previously undescribed species (1).

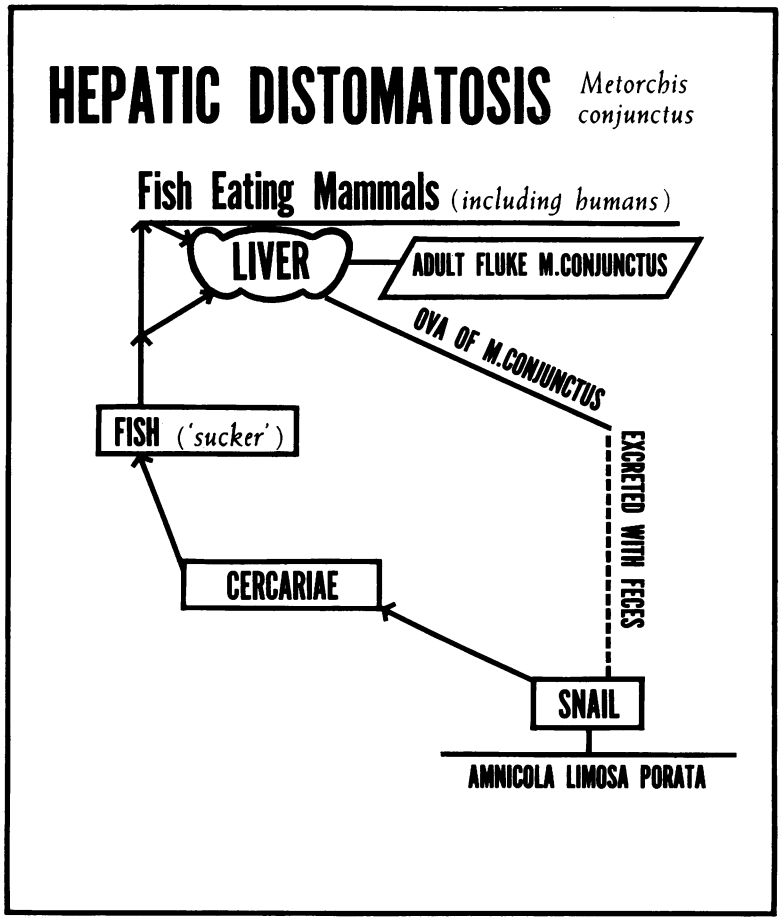

FIGURE 3
Diagram of the life cycle of Metorchis conjunctus.

From time to time during the past winter I received reports of acute canine disease with symptoms similar to those described above. These have come from Game Guardians, Department of Indian Affairs personnel, Royal Canadian Mounted Police and others, in the northern settlements. I have had an opportunity to investigate only a few of these outbreaks personally, but by the reports received, I am of the opinion that most of them were caused by infectious canine hepatitis. I am quite certain, however, that in some cases the virus infections are complicated by M. conjunctus which probably caused debilitation and rendered the animals susceptible to virus infection, or one could theorize that M. conjunctus is a transport host of ICH virus, acting in a way similar to Troglotrema salmincola and Neorickettsia helmintheca in Salmon poisoning disease (Figure 3).

At this point, I would like to quote the Director of the Commonwealth Bureau of Helminthology, Mr. J. M. Watson, D.Sc., A.R.C.S. who wrote recently:

> There is, of course, the possibility that the etiology of infectious canine hepatitis is similar to that of black disease in sheep. In the latter, immature Fasciola or Dicrocoelium larvae migrating through the liver parenchyma are thought to stimulate dormant spores of Clostridium oedematiens, already present in the

liver, into active reproduction with the resulting pathological effects. One might assume that the virus of ICH is stimulated in a similar way by M. conjunctus. In other words the invasion by the trematode triggers off a latent infection and causes an exacerbation of symptoms which otherwise would have remained subclinical.

To conclude this report, I would like to point out a very interesting observation made by Dr. Colburn, Director of Indian and Northern Health Services and myself: in settlements where dogs were seriously affected with M. conjunctus and ICH, there was a large number of cases of influenza (unfortunately it was impossible to classify it) and of infectious hepatitis affecting the human population, while in the other settlements where dogs were not sick, there was almost no disease in humans. Mere coincidence, or relationship between the organisms?

Résumé

Dès novembre 1959, de vagues rapports commencèrent à arriver au sujet de chiens de traineaux, mourant d'une étrange maladie dans le nord du Manitoba. Manquant de précisions quant à l'endroit et étant encore plus vagues dans leur description, ces rapports ne permettaient aucune action de la part de la Division de la Santé des Animaux. Enfin le 29 janvier 1960, le Surintendant de la Section des Affaires Indiennes du Ministère de la Citoyenneté de l'Immigration, rapportait la gravité d'une maladie inconnue, affectant les chiens dans le nord, et demandait l'assistance de la Division, afin de diagnostiquer, traiter ou de toute autre façon, enrayer une épidémie qui semblait des plus sérieuses. Accompagné du Surintendant, nous avons volé de Le Pas, à Nelson House et South Indian Lake où la situation semblait la pîre. Les Indiens étaient très alarmés parce que les chiens sont leur unique moyen de transport. Aussitôt que le but du voyage fut connu des Indiens, ils s'empressèrent de nous amener des chiens malades a différent degré et quelques-uns morts en janvier. En général les chiens mouraient 3 ou 4 jours après l'apparition des premiers symptômes, quelques rares exceptions furent malades environ un mois avant de mourir. J'ai examiné plusieurs chiens, et d'après les symptômes notés: apathie, innapétence, diarrhée, symptômes nerveux (allant de tremblements musculaires à la paralysie partielle ou complète de l'arrière-train) T°. 91.5–106.5; un diagnostic des plus réservés d'hépatite infectieuse accompagnée ou non de distemper fut posé. J'ai pratiqué de nombreuses nécropsies pour retrouver les mêmes lésions à chaque fois. L'état du foie surtout était frappant: de deux ou trois fois son volume normal, l'organe était couvert de petits abcès. A l'incision les abcès ainsi que les canaux biliaires l'aissaient s'échapper des milliers de petits parasites classifiés cliniquement comme étant des douves hépatiques du nom de Metorchis conjonctus, agent de la distomastose hépatique. Des spécimens convenables furent prélevés et envoyés à ADRI Hull, Québec où le diagnostic clinique fut confirmé.

Notons en passant que M. conjonctus fut décrit pour la première fois par Allen and Wardle de l'Université du Manitoba en 1933–34. Leurs travaux furent publiés dans le *Canadian Journal of Research*. Presque régulièrement tout l'hiver, de nouveaux cas furent rapportés pour finalement affecter presque tous les postes. Dans les postes les plus au Sud, où les chiens ne mangent pas de poisson, l'hépatite fut incriminée.

Suivant les commentaires concluant le rapport de Hull, les chiens ont tous souffert d'hépatite, en même temps que du parasite. Peut-être y a-t-il possibilité que M. conjonctus, soit vecteur du virus ICH tout comme Troglotrema salmincola l'est pour Neorickettsia helmintheca dans "Salmon poisoning".

Ici, j'aimerais citer le Directeur du Commonwealth Bureau of Helminthology, M. J. M. Watson D.Sc. A.R.C.S. qui, dans une récente lettre m'a suggéré ce qui suit:

> Il existe évidement des possibilités pour que l'étiologie de l'hépatite infectieuse soit semblable à celle de "Black Disease" chez le mouton. . . . On peut supposer que le virus de l'hépatite est stimulé de la même façon par M. conjonctus. En d'autres termes, l'invasion par le trématode déclenche l'infection et cause une exacerbation des symptômes qui autrement seraient demeurés latents.

Pour conclure cette partie du rapport, voici une observation des plus intéressantes. Dans tous les postes où les chiens étaient atteints par le parasite et le virus, les humains etaient atteint d'influenza (qu'il fut malheureusement impossible de classer) et d'Hépatite infectieuse, tandis que dans les quelques postes où les chiens ne furent pas affectés, les humains ne subirent aucune épidémie. Simple coincidence ou relation possible entre les agents pathogènes?

Reference

1. ALLEN, J. A. and WARDLE, R. A. Fluke disease in Northern Manitoba sledge dogs. Canadian Jour. Res. 10: 404–408, 1934.

RARE CAUSE FOR INCONTINENCE IN A BITCH

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Subject and History

A seven-and-one-half-month-old female Beagle was presented to us with incontinence. She had been spayed two months earlier by another veterinarian. Her incontinence had been treated unsuccessfully by him with diethylstilbestrol.

The incontinence developed a few weeks after spaying. This led us to suspect that it might be due to fibrous adhesions or a chronic productive peritonitis involving the bladder subsequent to the use of non-absorbable sutures such as silk, in ligating the stump of the uterus.

Treatment

A possible cause of incontinence is a remnant of ovarian tissue (1).

We performed an exploratory laparatomy and found only an abnormally long uterine stump. We removed as much uterine tissue as possible and within a week of the operation the patient was asymptomatic.

Reference

1. ARCHIBALD, J. Questions and Answers: Surgery for urinary incontinence in a bitch. Modern Veterinary Practice 41: 52. 1960.

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