ECHINOCOCCOSIS IN NORTH AMERICAN INDIANS AND ESKIMOS*

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HYDATID DISEASE, a form of helminthiasis, was described by Hippocrates, and now is known to be widely distributed throughout the world. It is prevalent in South America, Iceland, Australia, and New Zealand, and frequently encountered in the regions along the Mediterranean, and in Central Europe, Russia and South Africa. Native infection in the United States and Canada has been considered very rare. Recent observations indicating the presence of endemic echinococcosis in North America, and the increasing number of immigrants admitted to the United States and Canada from hydatid-infested countries, make imperative the recognition of the disease.

Osler in 1882[1] collected 61 cases occurring in Canada and the United States. Somner in 1895[2] increased the total number to 110.

Lyon in 1902[3] was able to present 135 new cases, all in natives of the United States and 2 in natives of Canada. Magath in 1921[4] summarized 334 cases. He added 4 more cases in the native-born to the previous observations. Phillips in 1930[5] reviewed 36 cases of pulmonary cyst published in the North American literature; only 2 of them occurred in natives. Magath in 1937[6] was able to find 31 cases of echinococcosis originating in natives of the United States and 4 in natives of Canada. Haight and Alexander in 1940[7] increased the total number of pulmonary hydatid cysts reported to 44 and added 2 of their own; only 5 of the patients were born in America and only 2 of them had never left the continent. In 1944 Davidson[8] reported 4 additional pulmonary hydatid cysts in natives, most likely contracted outside of the country. A complete review of the distribution of echinococcosis in North America was made by Magath in 1941[9] and in 1950.[10] By then 596 cases had been published, and 36 of them originated in natives of Canada and the United States. Since then 1 native case had been reported each from California,[11] Virginia,[12] Florida,[13] Mississippi,[14] four cases from Western United States,[15] 6 in Indians from Northern Manitoba[16] and 3 in Metis from Northern Alberta.[17] New York State registered 34 hydatid cases from 1947-52 without specifying the place of birth of the patients.[18] Rausch and Schiller in 1951[19] observed an unusual number of nodular livers and 2 surgically proved hydatid cysts in Alaska. Sweatman in 1952[20] reported 21 unpublished cases from Canada. Miller in 1953[21] increased this number to 3 Eskimos, 136 Indians and 2 white patients. This number likely included the previously published Canadian cases and Harrison's[22] 11 patients. The cases presented here include all the hydatid cysts originating from Alberta, the Yukon and Northwest Territories, some of them already reported in Sweatman's and Miller's publications.

MORPHOLOGY

The disease in man is produced by the larval form of the dog tapeworm, *Echinococcus granulosus*. The adult worm, 2.5-6 mm. long and 0.5 mm. broad, is composed of a head, neck and 2 segments or proglottides. The head has four suckers topped by a double row of from 28-50 hooklets. The terminal segment is gravid and contains several thousand eggs. The ova are globular, 30 microns in diameter. The adult worm lives parasitically in the digestive tract, mostly the terminal jejunum of the definitive host. The intermediate host becomes infested by ingesting the eggs fallen to the ground with the excreta of the definitive host.

The classical domestic life cycle of *Echinococcus granulosus* is between the dog and the sheep and cattle. The importance of a sylvatic life cycle as a reservoir for the infection was stressed by Magath.[23] He differentiated three variants. The typical one for Canada includes wolf-moose-wolf, with caribou and deer substituting for moose on occasions. The second variant occurs in certain states of the United States and involves on the one hand the fox and occasionally the dog and wolf and on the other hogs, sheep and cattle. The third life cycle is occurring in the St. Lawrence Island of Alaska, where foxes eat voles, the intermediate hosts to the disease. Rausch[24] found that the St. Lawrence cestodes may represent a separate species of *Echinococcus*.

The list of definitive hosts in Canada, besides the dog, include the wolf, the coyote, the fisher

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and the arctic fox. The intermediate hosts are moose, white-tailed deer, coast deer, reindeer, elk, wapiti, caribou and bison. The parasitic infection kept alive in wild animals will be mediated by dogs to humans, who act as aberrant hosts.

GEOGRAPHIC DISTRIBUTION AND INCIDENCE

The Foothills Region is a geographic unit made up of Alberta, the Yukon and Northwest Territories. The Indians of this region can be divided roughly into two groups. The plains natives are farmers and ranchers. Only rare isolated cases of hydatid cyst infestation have been discovered among them. The 7,000 Indians and Eskimos living above the 58th parallel are

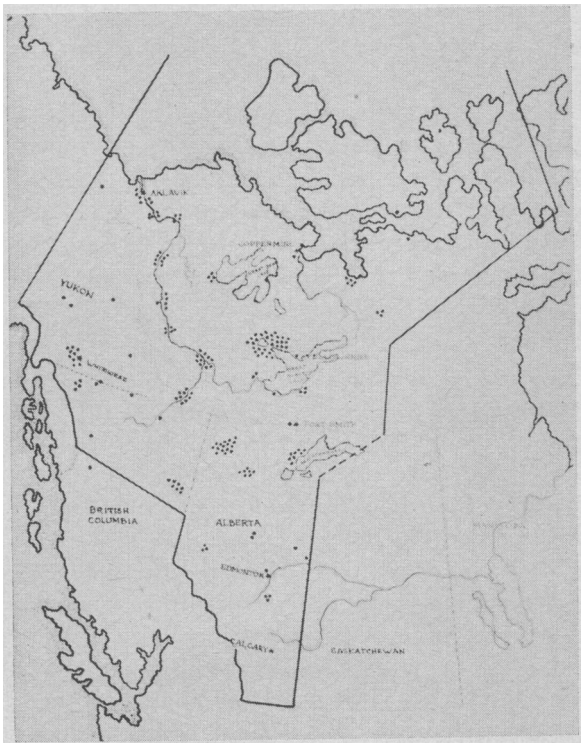

Fig. 1.—Map indicating the distribution of cases in the surveyed territories.

hunters and trappers who eat the products of their hunting and fishing, often without proper cooking. They live a nomadic life, closely associated with numerous dogs which are essential beasts of burden. The offal is fed raw to the dogs. This is a variant from the typical life cycle of the parasite, because the carnivore acquires the disease directly by being fed the viscera of the affected animals. The herbivorous game animals harbour the cyst most frequently in the lungs.

We have collected up to the present time 180 cases of hydatid cysts in the liver and/or the lungs, which represents a 2.7% incidence. The infestation can be assumed to be more prevalent on the basis of Miller's[21] survey in B.C. and N.W.T. In 1953 he obtained 15% positive and 12% doubtful reactions by performing the Casoni type of intradermal test with Australian antigen (sheep hydatid fluid). Wolfgang[25] in a different region, the Southern Yukon, tested 294 persons in the summer of 1954 with Aklavik-type antigen (reindeer hydatid fluid) and found 45% positive reactions, not counting 50 doubtful results.

MORBID ANATOMY

The human infestation is initiated by the ingestion of grass, vegetables or stagnant water where *Echinococcus* ova have been deposited. The parasites may also be directly acquired by close contact with affected dogs or pelts of wild animals. Once the ova are ingested by man, they lodge in the stomach and the gastric juice digests the external coat, thus freeing the embryo. These burrow through the gastric mucosa and enter the capillaries of the portal circulation and are carried to the liver. The majority are retained there but some of them reach the lungs. Once the embryo comes to rest, it grows to a vesicle and then into a hydatid cyst. A mature cyst is composed of an endocyst or germinal membrane and an ectocyst or laminated hyaline membrane. From the former wall, masses of cells which become vacuolated protrude into the vesicle's cavity and form scolices. These in turn vacuolate and develop into daughter cysts. The spilling of any part of the germinal layer or scolices may result in secondary *Echinococcus* cyst formation. The free scolices sink to the bottom of the hydatid fluid and form the hydatid sand. The cyst fluid is clear, limpid and alkaline, having a specific gravity of 1.008-1.015, and contains 0.6% NaCl. The finding of any component of the hydatid cyst is the only conclusive evidence of the disease. The host's reaction to the cyst is a non-specific foreign-body reaction, forming a pericystic layer or adventitia composed of polymorphonuclear, lymphocyte and plasma-cell infiltration and degenerated as well as normal cells of the affected organs. The continuous pressure of the cyst will produce necrosis of the neighbouring cells, with subsequent occurrence of second-

TABLE I.

DISTRIBUTION OF HEPATIC AND PULMONARY HYDATID CYSTS IN DIFFERENT COUNTRIES

Country	*Liver %*	*Lungs %*
Lebanon[26]	56.0	30.0
Turkey[27]	51.7	29.9
Greece[28]	....	30.0
South America[29]	70.0	15.0
Australia[30]	76.6	9.4
France[31]	75.0	8.5
United States and Canada[10]	72.0	9.0

ary fibrosis and hyaline degeneration. The pericyst formation is more intense in the lungs than in the liver.

The distribution of the cysts in the body varies from country to country (Table I). In the liver 85% of the cysts are in the right lobe,[32] and in the lungs the right to left ratio is 60 to 40.[30] Hydatid cysts may also be located in voluntary muscles, kidneys, spleen, heart, bones, brain, and spinal cord. They occur in these organs in about equal frequency and are the result of systemic spread of the ova.

The ultimate fate of the cysts is variable. If the growth becomes excessive, the danger of rupture is imminent. Liver cysts may rupture transdiaphragmatically into the pleural space, bronchi, bile ducts or the free abdominal cavity. The rupture of large pulmonary cysts is the rule, with emptying of the cyst content into the bronchi or pleural space. Apical, parabronchial cysts are most likely to end in early rupture. If all the contents are eliminated, spontaneous cure may result. This is considered to occur in 60-90% of the cases.[30, 31, 33-36] The cyst content may also become infected or hæmorrhagic, which will kill the living parasite in the liver. Secondary calcification follows.

TABLE II.

AGE DISTRIBUTION OF CYSTS IN LIVER AND LUNGS

Age	*Liver*	*Lungs*	*Total*
Under 25	1	63	64
25-50	16	35	51
Over 50	54	11	65
Total	71	109	180

In our series of 180 cases, liver cysts were found in 71 patients and lung cysts in 109. The sex incidence was not unusual; 93 were in females and 87 in males. Table II shows the age distribution and indicates that lung cysts were found mainly in children and young adults, while liver cysts were seen in older persons. Of the pulmonary cysts 27 were found in children under 10 years of age; the youngest child was 3 years old. The relatively small number of pulmonary hydatid cysts in older persons suggests that most of the cysts terminate in spontaneous cure by rupture into a bronchus.

The cysts were found almost equally in the two lungs, 56 in the right and 53 in the left. Analysis of the position of cysts in the hospitalized group reveals a marked tendency to posterior location. On the right four were in the posterior apical segment and 9 in the posterior basilar segment, the remaining four being in the anterior apical, middle lobe, medial basilar and dorsal segments. On the left side three were in the posterior apical segment, three were in the dorsal segment of the lower lobe, and the other six were scattered in the posterior basilar, anterior apical, pectoral and lateral basilar segments.

No cysts were found outside the liver and lungs in our experience.

CLINICAL SIGNS

The infection is most frequently acquired in childhood but the clinical manifestations appear only later in life when the slowly growing cyst makes itself evident by pressure on some important structure or by some complication. The uncomplicated hydatid infection is a silent condition. The symptoms of pulmonary cysts are variable and include cough, slight hæmoptysis due to bronchial irritation and vascular congestion. Pain is exceptional, unless the cyst is in contact with pleura.[37] Cardiac displacement, even with large cysts, is unusual. Large masses in the liver may produce pain by pressure or perihepatitis, or colic from extrinsic compression of the bile ducts. The possible ways for liver cyst to rupture were outlined in the section on morbid anatomy, and the symptoms are self-evident. The pressure erosion of bronchi by pulmonary cysts may result "in cure or morbidity or disaster".[38] Susman states that spontaneous rupture has a mortality rate of 1-2% by asphyxia from drowning by the hydatid fluid or impaction

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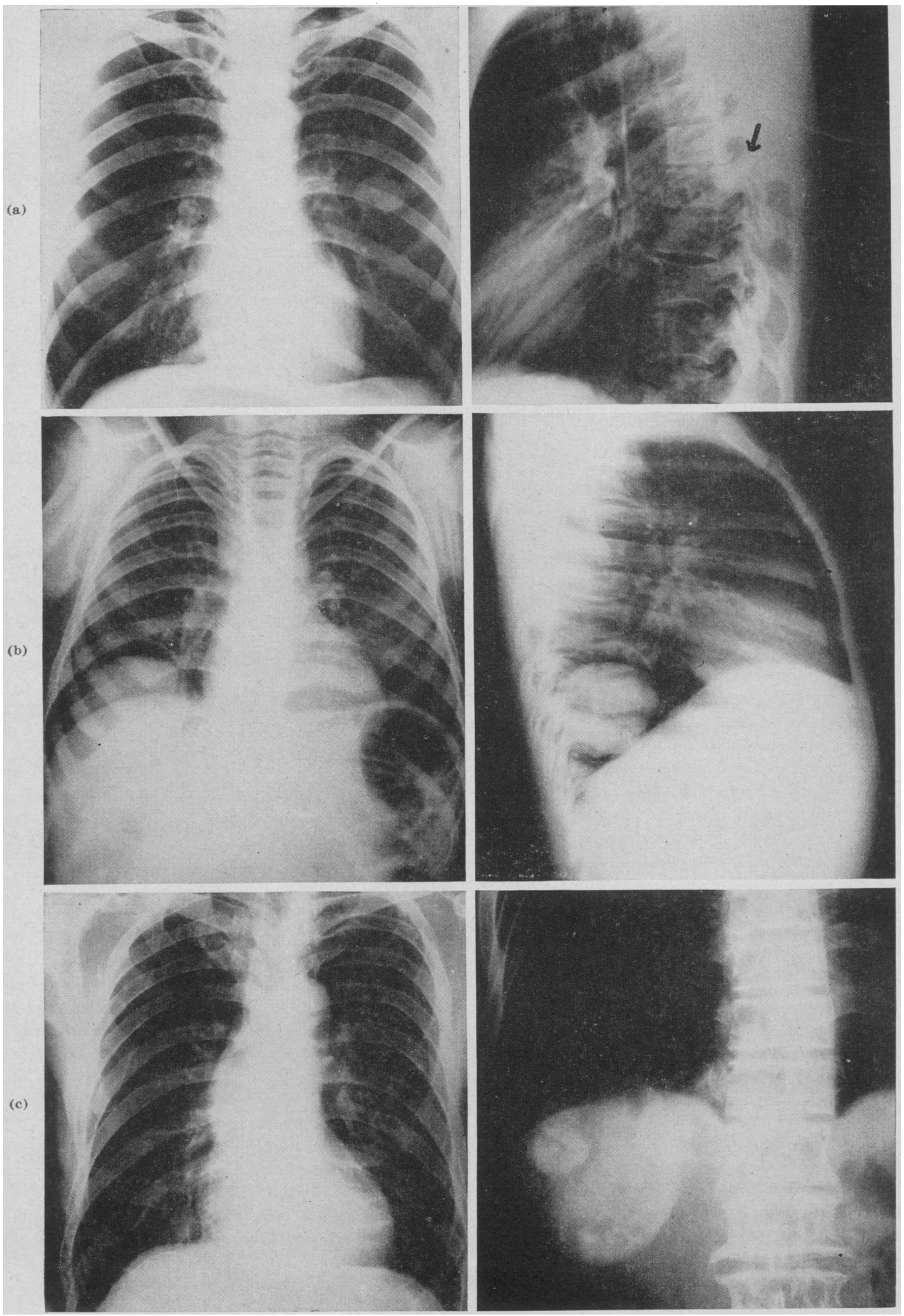

Fig. 2.—X-ray appearance of (a) simple hydatid cyst; (b) ruptured hydatid cyst (double arch formation); (c) calcified liver cysts as seen on films of the chest and the hepatic region.

of the membrane in the glottis, anaphylactic shock or severe hæmorrhage. The hydatid vomica is characterized by a peculiar sputum, formed of mucus, frothy red blood, hydatid sand and laminated membrane. The sputum is tenacious, tastes salty and causes violent paroxysms of cough. Less serious symptoms or even absence of symptoms may accompany the rupture. There is no way of forecasting the event. The other possible outcomes of the cyst are listed as follows by Barrett and Thomas:[39] (1) Sealing of the rupture with continuous growth of the cyst; (2) Elimination of the fluid but retention of the membranes with secondary infection and eventual abscess formation or daughter cyst production; (3) rupture into the pleural space with possibility of pneumothorax, pyothorax or daughter cyst formation; (4) rupture into the vascular system with spread of the disease by the systemic circulation.

In our series 35 patients were admitted to hospital, 29 with lung cysts and 6 with liver cysts; the others were observed in the field. Three liver cysts caused sufficient pain to require operation; the others were accidental findings on routine investigation. Of the 109 lung cysts, 54 have shown spontaneous rupture. The process seems to be a gradual enlargement over a period of about five years, reaching a diameter of approximately 8-10 cm. Two patients with ruptured cysts required hospitalization because of the severity of their hæmoptysis. Two patients ruptured their cysts while in the hospital and the event was only discovered on subsequent x-ray examination. One patient had an unexplained fluid collection in the abdomen with fever and pain, and was later operated upon for a left basal pulmonary hydatid cyst. Another patient had a pleural fluid collection which cleared spontaneously in two weeks after the rupture. Another patient had developed a chronic empyema following intrapleural rupture of a pulmonary hydatid cyst. In all the other cases the rupture was uneventful or presented itself as a mild illness evidenced by chilly sensation and a low-grade fever for a few days without obliging the patient to request medical aid. Daughter cysts were not seen in the five-year period of follow-up studies.

RADIOLOGICAL AND LABORATORY SIGNS

Simple pulmonary hydatid cysts are usually detected by radiography. The uncomplicated pulmonary cyst gives a regular, well-defined, rounded or oval shadow of uniform density which cannot be differentiated from that of a tumour.[40] The differential diagnosis has to be made from carcinoma, sarcoma, abscess, interlobar empyema, actinomycosis, tuberculoma, infarct, dermoid cyst and intrathoracic struma. The ruptured cyst will give a much more typical, even pathognomonic x-ray appearance. Three different gradations are described: (1) perivesicular air collection produced by the entrance of air between the adventitia and hydatid vesicle. (2) Double arch formation with air infiltrating around the cyst as well as inside the cyst (Cumbo's sign). (3) Complete disintegration of the cyst with floating of the retained membrane on the residual cyst fluid, giving the Camelot or water-lily sign. At this stage the radiological picture may simulate that of a tuberculous focus, abscess or carcinoma (Fig. 2).

Liver involvement may be suspected by the elevation of the right leaf of the diaphragm, with disturbance of its normal contour. Diaphragmatic excursion is decreased on fluoroscopy. The tennis-ball elevation of the dome is considered to be typical of *Echinococcus* cyst, and the presence of one or more dense, circular, calcified shadows in the liver parenchyma is pathognonomic for hydatid cysts (Fig. 2). In our series, only the calcified liver masses were included and disfiguration of the diaphragm alone was not considered as sufficient proof of the disease, though the phenomenon was observed frequently.

The laboratory diagnostic methods consist of intradermal, precipitin and complement-fixation tests as well as the detection of eosinophilia in the peripheral blood. Casoni's intradermal test is usually done by the injection of 0.1-0.2 ml. of sterilized hydatid fluid; a positive reaction will result in a central wheal with peripheral erythema within 10-30 minutes. The presence of pseudopodia is characteristic of the reaction. Delayed reaction may occur after 24 hours but is less frequently positive than the immediate reaction. The accuracy of the test varies from 53.8-100% with different authors, and it is considered to be the most reliable single test for hyatidosis. It is very likely that the reaction, once acquired, remains positive for life, even when the cyst is dead or surgically removed. There is some controversy on the changes in

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the positivity of the reaction in the case of rupture. Negative reaction practically excludes complicated cyst for Susman,[38] but for Dew[41] and Oberhofer[42] specificity will decrease with rupture. The precipitin test gives 65% and the complement-fixation test 52.4% positive results in cases of proved disease. The complement-fixation test remains positive only as long as a living cyst is present in the body. Godfrey[43] disagrees with these observations and considers the precipitin test as the most reliable, and Bensted and Atkinson[44] had better results with the complement-fixation test. No false positive results are described.

Of our 27 histologically proven cases, 15 had positive and 7 negative reactions to Casoni reagent; in 5 cases no test was done. All the negative results occurred with the Australian type of antigen. The increased specificity of endogenous antigen made available by Professor T. W. M. Cameron of the Institute of Parasitology, McGill University, is illustrated in Fig. 3. Since

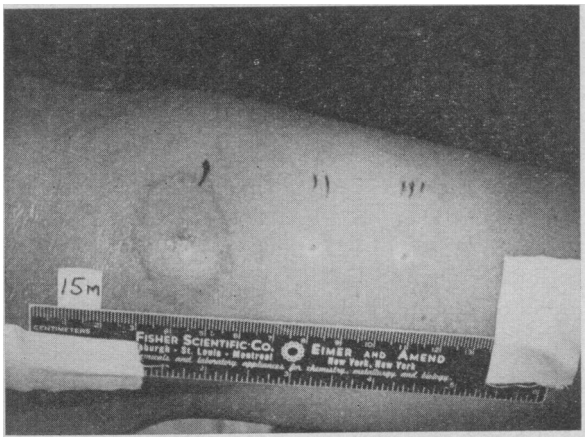

Fig. 3.—Casoni's intradermal test 15 minutes after injection of 0.2 ml. of: I. Aklavik type antigen; II. Normal saline; III. Australian type antigen.

the introduction of this type of antigen, all but one of our radiologically and surgically proven cases reacted positively. A considerable number of patients had a positive Casoni reaction without evidence of pulmonary cysts, and therefore were supposed to harbour the disease in the liver or elsewhere or to have ruptured their cysts before investigation. In positive Casoni reactors with proven hydatid disease, six complement-fixation tests were positive and two were negative. Three positive precipitin tests were obtained, one in the presence of positive and two in the presence of negative intradermal tests. In two other cases, both the Casoni reaction and precipitin tests were negative. An eosinophilia over 4% is considered to be present in 50% of cases. In our patients the eosinophilia varied from 0-46% and was consistently elevated in four patients only, occasionally over 4% in 11 patients, and the eosinophil level was never higher than 4% in the remaining 12 patients.

A triad of negative Casoni test, negative complement fixation and absence of eosinophilia does not exclude the diagnosis in the presence of a typical radiological picture. The biological and x-ray investigatory techniques together will give the correct diagnosis in 90% of cases.

In 20 cases of pulmonary cyst the average vital capacity was 84% of normal. There was evidence of right axis shift with inverted T waves in lead 3 in the majority of electrocardiographic tracings done preoperatively for pulmonary hydatid cysts. In seven cases bronchographic studies did not aid in establishing the diagnosis; no dye entered the cyst cavity.

TREATMENT

Medical treatment is of no value in hydatid disease. The only type of medical management had been described by Castex and Capdehourat,[45] consisting of transthoracic intrapulmonary injections of sulfonamides and antibiotics. The induction of artificial pneumothorax with the intention of producing pressure on the cysts and secondary rupture was advised only for deeply located cysts and in the presence of multiple cysts,[46] but is mentioned only to be condemned.

The majority of publications dealing with echinococcosis are surgical, and for many authors[18, 38, 39, 42] the removal of every pulmonary cyst is the only treatment. The rationale of early surgical approach is to avoid the higher mortality and morbidity after complications begin.

The indications for operation in our series were the rapid growth of the cyst, secondary infection of the ruptured cyst cavity, empyema, and uncertain diagnosis. Because of the high incidence of spontaneous cures by rupture without known mortality and limited morbidity, the symptomless pulmonary cysts were followed up by constant radiological observation. As mentioned before, no daughter cysts have been observed to this date. The average period of observation before operation was 23 months with a range varying from 3 to 60 months.

The principles underlying surgical treatment are:[47] (1) removal of the parasite; (2) avoidance of contamination; (3) elimination of the residual space. In uncomplicated cysts the two-stage operation creating pleural adhesions at the first stage has been abandoned, and open thoracotomy is favoured,[48, 49] with the protection of the pleural space from contamination. Because there is never a direct fusion between the cyst and the lung, the hydatid cyst may be completely detached from the pericyst either by sudden inflation of the lung which will push out the whole cyst or by suction of the cyst. While there is a general agreement that no diagnostic (transthoracic) puncture of the cyst should be attempted, most surgeons favour operative aspiration of the cyst before hydatid delivery.[38, 42, 50, 51] This manœuvre technically facilitates the procedure and lessens the risk of accidental rupture. Barrett and Thomas stress that enucleation should not be preceded by aspiration. The empty pericystic sac may be sutured, drained or left alone. Some advocate postoperative drainage of the pleural cavity.[26] Additional marsupialization with ether or 2% of 40% formaldehyde is advocated for infected cysts, with subsequent drainage. These techniques avoid unnecessary removal of healthy lung structure. The resection of pericyst as an additional procedure is also advised for complicated cysts, though more radical resection is the treatment of choice in these cases. Segmental resection or lobectomy is advisable in the presence of severe hæmorrhage from the cyst, residual bronchiectasis, giant cyst, uncertain diagnosis and empty, infected or hæmorrhagic cysts.[48] Four pneumonectomies were reported in the literature reviewed. The mortality rate in surgical removal of cysts varied from 0.6% for simple to 10% for complicated cysts,[42] with an over-all mortality of 2.6%.[51]

In our series 23 pulmonary cysts were removed at operation. In the earliest cases the absence of a positive diagnosis resulted in a more radical type of treatment. Of the first 10 operated upon, 5 had lobectomy, 4 wedge resection and 1 segmental resection. Of the last 13 patients subjected to surgery, 12 had simple cyst removal, referred to as cystectomy, and the remaining one required a pleural decortication for chronic empyema following rupture of a basal cyst into the pleural cavity.

Cystectomy is very simple and very effective. On opening the pleural space the cyst has always been very easily identified. A fine needle is introduced and the cyst content aspirated, resulting in marked deflation. At the margin of the cyst an incision is made through the visceral pleura and by using suction the hydatid vesicle is removed easily and without any pleural leakage. The residual space is opened widely, and deeply placed catgut sutures are inserted to obliterate the space. Finally the visceral pleura is loosely closed with running catgut suture. The pleural space is drained by an underwater seal drainage to remove any blood or air accumulation.

In our entire series no complications have been encountered. Within two weeks the area of cyst removal has reverted to a normal appearance with a minimal linear x-ray shadow, which completely resolves in six weeks.

Liver cysts were surgically treated when they produced symptoms. Usually, simple enucleation without drainage of the residual cavity is adequate treatment. For subdiaphragmatic cysts the transthoracic approach is more convenient.

SUMMARY

The North American literature of echinococcosis is reviewed. The morphology, geographical distribution, morbid anatomy, clinical, radiological, and laboratory signs and the treatment of the disease are discussed. One hundred and eighty cases are reported from Alberta, the Yukon and Northwest Territories, where the disease is endemic in Indians and Eskimos.

REFERENCES

1. OSLER, W.: *Am. J. M. Sc.*, **84**: 475, 1882.
2. SOMNER, H. O.: *New York M. J.*, **62**: 656, 1895.
3. LYON, I. P.: *Am. J. M. Sc.*, **123**: 124, 1902.
4. MAGATH, T. B.: *M. Clin. North America*, **5**: 549, 1921.
5. PHILLIPS, E. W.: *Arch. Surg.*, **21**: 1324, 1930.
6. MAGATH, T. B.: *Am. J. Hyg.*, **25**: 107, 1937.
7. HAIGHT, C. AND ALEXANDER, J.: *Arch. Int. Med.*, **65**: 510, 1940.
8. DAVIDSON, L. R.: *J. Thoracic Surg.*, **13**: 471, 1944.
9. MAGATH, T. B.: *Pennsylvania M. J.*, **44**: 813, 1941.
10. *Idem: Arch. internac. hidatid.*, **11**: 193, 1950.
11. ATTWOOD, C. J., SARGENT, W. H. AND TAYLOR, F.: *Ann. Int. Med.*, **15**: 1109, 1941.
12. WILLIAMS, C., JR.: *South. M. J.*, **46**: 1104, 1953.
13. ZIZMOR, J. AND SZUCS, M.: *Am. J. Roentgenol.*, **53**: 15, 1945.
14. JOHNSTON, J. H., JR. AND TWENTE, G. E.: *Ann. Surg.*, **135**: 305, 1952.
15. CARLQUIST, J. H. AND DOWELL, R. J.: *Rocky Mountain M. J.*, **46**: 773, 1951.
16. PERRIN, M. B.: *Winnipeg Clin. Quart.*, **8**: 3, 1955.
17. STEPHENS, H. H.: Personal communication.
18. DAVIDSON, L. R.: *Quart. Bull. Sea View Hosp.*, **14**: 75, 1953.
19. RAUSCH, R. AND SCHILLER, E. L.: *Science*, **113**: 57, 1951.
20. SWEATMAN, G. K.: *Canad. J. Pub. Health*, **43**: 480, 1952.
21. MILLER, M. J.: *Canad. M. A. J.*, **68**: 423, 1953.
22. HARRISON, E.: *J. Thoracic Surg.*, **26**: 129, 1953.
23. MAGATH, T. B.: *J. Am. Vet. M. A.*, **125**: 411, 1954.
24. RAUSCH, R.: *J. Infect. Dis.*, **94**: 178, 1954.
25. WOLFGANG, R. W.: Personal communication.
26. JIDEJIAN, Y.: *Surgery*, **34**: 155, 1953.
27. KURBAN, A. K. *et al.*: *Am. Heart J.*, **46**: 764, 1953.

28. TOOLE, H., PROPATORIDIS, J. AND PANGALOS, N.: *Thorax*, **8**: 274, 1953.
29. ARCE, J.: *Arch Surg.*, **42**: 1, 1941.
30. DEW, H. R.: *Surgery*, **2**: 363, 1937.
31. DEVE, F.: *Compt. rend. Soc. de biol.*, **79**: 911, 1916.
32. ARCE, J.: *Arch. Surg.*, **42**: 973, 1941.
33. BAKISH, M.: *Treat. Serv. Bull.*, **9**: 1, 1954.
34. MORQUIO, L.: *Arch. de méd. enf.*, **29**: 72, 1926.
35. LEPICARD, S.: Thèse de Paris, No. 290, 1912. BELLARD, L.: Thèse de Paris, No. 105, 1924. as quoted by Godfrey (43).
36. WADDLE, N.: *Australian & New Zealand J. Surg.*, **19**: 273, 1950.
37. SUSMAN, M. P.: *J. Thoracic Surg.*, **26**: 111, 1953.
38. ARCE, J.: *Arch. Surg.*, **43**: 789, 1941.
39. BARRETT, N. R. AND THOMAS, D.: *Brit. J. Surg.*, **40**: 222, 1952.
40. EVANS, W. A., JR.: *Radiology*, **40**: 362, 1943.
41. DEW, H. R.: Hydatid disease, Australasian Med. Publ. Co. Ltd., Sydney, 1928.
42. OBERHOFER, B.: *J. A. M. A.*, **157**: 1331, 1955.
43. GODFREY, M. F.: *Arch. Int. Med.*, **60**: 783, 1937.
44. BENSTED, H. J. AND ATKINSON, J. D.: *Lancet*, **1**: 265, 1953.
45. CASTEX, M. R. AND CAPDEHOURAT, E. L.: *Brit. M. J.*, **2**: 604, 1950.
46. BARRETT, N. R.: *Lancet*, **2**: 234, 1949.
47. LECOS, E.: *Quart. Bull. Sea View Hosp.*, **14**: 72, 1953.
48. SUSMAN, M. P.: *Thorax*, **3**: 71, 1948.
49. BENDANDI, G.: *J. Internat. Coll. Surg.*, **18**: 22, 1952.
50. BETTS, H. R. AND THOMAS, T.: *Dis. Chest*, **26**: 584, 1954.
51. TAIANA, J. A., SCHIEPPATI, E. AND ZORRAQUIN, V. A.: *Dis. Chest*, **26**: 686, 1954.

ADDENDUM

Seven more hydatid cysts have been removed from the lung since this paper was written. All cases were uncomplicated.

Case Reports

THE SIMULATION OF CARCINOMA OF THE COLON BY SEGMENTAL COLITIS

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Two CASES are reported in which a preoperative diagnosis of carcinoma of the colon was made partly on the basis of a localized obstructing lesion, but in which subsequent pathological examination disclosed segmental colitis. Segmental (regional) colitis is an idiopathic inflammatory disease involving one or more segments of large bowel, exclusive of rectum and rectosigmoid, either as an extensive continuous lesion, or a single segmental lesion, or as multiple discontinuous segments. Pathologically the disease is considered by some to be akin to regional enteritis (Crohn's disease), by others as an atypical form of idiopathic ulcerative colitis, while occasionally its clinical simulation of carcinoma and diverticulitis of the colon has presented diagnostic problems of considerable magnitude. The following two cases are illustrative of this problem.

CASE 1. A 30-year-old logger began to develop periodic gnawing periumbilical pain maximal on the right side two months before hospitalization. The pain was not related to food or activity; it was relieved, however, by defæcation and by lying on the left side. During these two months, there was intensifying constipation which required increasing amounts of laxative for relief.

Three weeks before hospitalization, the patient's appetite decreased markedly, and because of nausea only fluids were tolerated. He lost 15 lb. in weight. On the day of hospitalization, crampy periumbilical pain was felt and he vomited sour, green fluid repeatedly.

Examination revealed a firm, slightly tender intra-abdominal mass in the right upper quadrant, which was fixed, irregular and about 3 inches (7.5 cm.) in diameter. Sigmoidoscopic examination was negative. The stools were free of occult blood. The white cell count was 13,000 with 83% neutrophils and 17% lymphocytes; the hæmoglobin level was 100% and urinalysis was negative.

An upper gastrointestinal barium series revealed moderate distension, without displacement, of the descending duodenal loop; the stomach and small bowel were normal while barium enema outlined a pronounced narrowing of the lumen and distortion of the mucosal pattern in a 1½ inch segment of the transverse colon just distal to the hepatic flexure (Fig. 1). The preoperative diagnosis

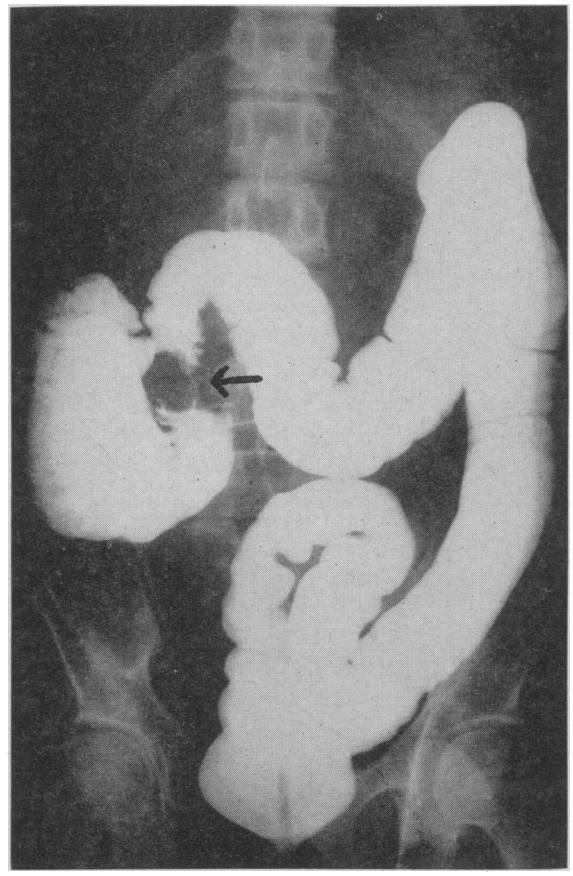

Fig. 1.—Barium enema showing linear filling defect in the transverse colon.