CASE REPORTS

GENERALIZED CYSTICERCOSIS WITH CEREBRAL INFESTATION*

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Taenia Solium now rarely occurs in Canada, and in 1954 only four cases in pigs were reported from Western Canada by the Veterinary Director General.

Human cysticercosis presents therefore no problem, but with the influx of immigrants from South America, Mexico, Eastern and Southern Europe and India, the possibility of cases of human cysticercosis is to be borne in mind. The following case histories are an example of the above.

Case 1.

Mrs. G.P., a 33-year-old Italian woman, came to Canada two years ago. She was born in a small village in Southern Italy, in a poor agricultural district with very inadequate hygienic measures, where humans and animals intermingle freely, sharing to a great extent the already poor amenities of housing. She had scanty schooling and an unsettled family life. Married at 17, she had three children, now aged 9, 12 and 15 years. She was well, apart from minor childhood illness, an occasional brief febrile episode, until 1947. At that time she awoke one morning feeling ill, with general malaise, abdominal pain, tinnitus, vertigo and violent vomiting of large quantities of bile. She became restless, developed twitching movements of her limbs, and became unable to speak and finally lost consciousness. She remained so for three days, with recurring vomiting and convulsions.

On recovery she had daily seizures of various types. She was investigated in two hospitals in Italy and was told she had gallbladder disease. Since then she has continued to have several types of seizures, the descriptions of which vary greatly. She usually has some warning, with blurring of vision and a feeling that something terrible is going to happen. She has definite grand mal seizures involving chiefly twitching of the left face, with mild jerking of the hands and feet, lasting about 10 minutes. About once every 10-14 days she has an attack of nausea and vomiting, with tinnitus and vertigo, followed by a more severe grand mal seizure with incontinence and unconsciousness which may last for a day or so. She also has attacks in which she complains of paralysis, starting at her feet and spreading up to involve her trunk and arms, with eventual loss of consciousness, lasting for 20-30 minutes. In other attacks she becomes "rigid all over" for about 10 minutes. She has been observed in attacks in which, while talking, she suddenly stares into space for a matter of a few minutes. Her husband states that at times when talking normally she suddenly goes to sleep and remains so for 2-3 hours. At other times, when laughing heartily, she suddenly becomes paralyzed and falls to the floor unconscious for a minute or so.

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The physical examination of the patient in April 1955 did not reveal a great deal. She looked well and was well-nourished; the skin, subcutaneous tissues and muscles were everywhere normal. Head and neck were normal. The chest was within normal limits. The heart was normal—B.P. 120/74, pulse 72, regular. The abdomen was normally soft, no masses were encountered, the colon was contracted throughout and tender on pressure. The liver was palpable, the spleen not palpable. The joints and bones appeared normal. There was no impairment in the central nervous system. The fundi were normal. While the patient was undergoing examination, she had a petit mal seizure lasting approximately 5 minutes. The urine was normal. The blood count: red cell count 4,000,000, Hb 82%, white cell count 9,450 with 2% eosinophils, and 78 eosinophils per c.mm. on a direct count. The sedimentation rate was 14 mm. in one hour. Cephalin cholesterol flocculation and bromsulphalein retention tests were normal. Stools were negative for parasitic ova. The skull radiograph...
was normal, showing a well-calciﬁed pineal gland without shift. Radiographs of the thighs (Fig. 1) showed the soft tissues to be ﬁlled with numerous calciﬁed densities, some bigger, some smaller, some linear, some in the form of capsules representing calciﬁed larvae of Cysticercus cellulosa. Other muscles, i.e. the glutei and phenylhydantoin), Mysoline (pyrimidone) and barbiturates with considerable reduction of the paroxysms in intensity and frequency. Regular control visits are made.

**Case 2.**

Mr. J.S., 46 years old, was born in the Ukraine in a small village in a poor farming district with very rudimentary hygiene, where domestic animals and humans intermingled freely indoors and outdoors. During the First World War he lived for two years in a refugee camp in Russia in a barn shared by many women, children and domestic animals. In 1916-1918 he lived with his mother in an orphanage in Russia where he contracted typhus. He came to Canada in 1929. He was well until 1934, when he had a gastrointestinal haemorrhage with black stools but no haematenaemia and was treated conservatively in the Toronto General Hospital. In 1937 he was admitted to the Toronto General Hospital complaining of dizziness and loss of speech twice in four weeks and loss of comprehension of words for a few minutes, right-sided frontal headache for 3-4 days, fatigue and loss of weight, general weakness in arms and legs, more on the right side, and aching pain in the lumbar region. He was discharged with a final diagnosis of duodenal ulcer and petit mal. In November 1937, he came to the Medical Clinic with numbness and pain over the right eye, on one occasion associated with inability to speak. In 1939 he had two episodes in bed consisting of inarticulate noise, heavy breathing, stiffening of the body but no biting of tongue, no clonic convulsions or loss of sphincter control. These attacks have been described by family members. The attacks were followed by right frontal headaches, fatigue and inertia. He seemed to have had several of such attacks since his first gastrointestinal haemorrhage. In 1939, a diagnosis was made in the Neurological Clinic, T.G.H., of epilepy or cerebral spasm. In January 1942, he was treated again conservatively in the Toronto General Hospital for haemorrhage and duodenal ulcer.

In December 1943, he was admitted to the Mayo Clinic, Rochester, Minnesota, for partial gastrectomy of posterior Polya type. A skull radiograph showed a small area of calciﬁcation in the meninges over the right frontal lobe and a contiguous meningioma was suspected. He was reasonably well until 1946 and worked as a truck driver, but at this time started to have frequent right-sided headaches, increasing fatigue, lack of energy and difﬁculty in remaining awake. He was seen to have another seizure while driving, with an

![Fig. 3. Case 1.—The electroencephalogram showed a diffusely disorganized record with an abnormal focus in the right fronto-temporal region, with paroxysmal features suggesting epilepsy.](image)

![Figs. 4a and 4b. Case 1. (G.P.).—The air encephalogram showed asymmetry in the lateral ventricles, the lateral right being larger than the left (2 views).](image)
outcry of inarticulate sounds, heavy breathing, foaming and convulsions followed by prolonged sleep and right frontal headache. He began to take amphetamine and Bromo-Seltzer and large quantities of APC tablets to combat his fatigue and headaches. In 1950 he was in Toronto East General Hospital with abdominal pain. At that time an appendectomy was performed and his old gastrectomy scar was opened, but nothing abnormal was found.

From 1951 till the present time he had no permanent form of employment. He worked at various jobs, but became very fatigued, unable to concentrate, very irritable and dismayed. When taking Bromo-Seltzer, Dexedrine (d-amphetamine) and APC he felt better for short periods. In September 1955, he was admitted to the Toronto Psychiatric Hospital, where he remained until November 1955, being discharged with a diagnosis of a drug addiction on the basis of inadequate personality. He was found to have sulphonamolbinaemia and anaemia (60% Hb), and was referred to the Toronto General Hospital for further investigations. These showed no abnormalities on physical examination except for a marked pallor of the skin and mucous membranes, with no stomatitis or glossitis.

The urine was normal. Haemoglobin 7.5 g.%, red cell count 4.8 million, white cell count 9,200 with normal differential, haematoctit 32%. The blood smear revealed hypochromia but little microcytosis. The sedimentation rate was 21 mm. in one hour. Blood serology was negative. Serum calcium level was 8.9 and 8.7 mg. %. Serum phosphorus level was 2.8 mg. %. Alkaline phosphatase was 8.6 King-Armstrong units. Serum electrolytes were normal. The non-protein nitrogen was 30 mg. %. The stools were negative for occult blood, fat globules or meat fibres, ova, cysts or parasites. Fasting gastric analysis revealed a histamine-fast achlorhydria. A xyleose tolerance test for gastrointestinal absorption was within normal limits. A barium enema showed no abnormalities. An upper G.I. series revealed rapid transit from the stomach to the small intestine; there was no evidence of stomal ulceration. A small bowel follow-through examination revealed a completely normal small bowel. Skull radiographs showed two small areas of calcification in the right frontal region compatible with an encysted larva of *Cysticercus*. In the lateral view a linear calcific density in a tongue of the parotid gland, representing a calcified cysticercus, was found. X-ray of the thighs showed the presence of multiple fusiform calcifications in the soft tissues ranging from several mm. to several cm. in length. An electroencephalogram was within normal limits. On repeating the latter two months later, epileptic paroxysms were seen during hyperventilation, and evidence of a localized abnormality near the occipital pole and in the right temporal region was obtained. The patient improved on oral iron therapy, proper diet and a small amount of barbiturate at night and is being followed up regularly.

**Final Diagnosis:** (1) Hypochromic anaemia after subtotal gastrectomy for duodenal ulcer; (2) generalized cysticercosis with cerebral calcification and personality pattern disturbance.

**Pathological Anatomy**

If man ingests eggs of *Taenia solium* contained in human intestinal content or if mature proglottids are forced into the stomach by reverse peristalsis, the embryos are liberated by the action of gastric juice. The tapeworm larvae, known as *Cysticercus cellulosae*, invade the intestine, enter the blood and are distributed to all tissues of the body. Full development takes place in the subcutaneous tissue, brain, orbit, musculature, heart, liver, lungs and peritoneum, with the formation of a cyst. Each cyst consists of a clear, translucent membrane with an opaque white spot at one point, representing the worm. Microscopically a surrounding fibrous capsule is seen, infiltrated with eosinophils and polymorphonuclear leukocytes. After several months or years many of the worms die and there is a conspicuous reaction with necrosis of the worm and some of the surrounding tissue, infiltration with eosinophils and polymorphonuclear leukocytes, formation of giant cells and eventually calcification.

The cysts produce symptoms almost entirely because of the pressure on surrounding structures. Many instances of epilepsy are believed to be caused by cysticercosis of the brain.

Cysticercosis is found in all parts of the world where *Taenia solium* is found, but especially in those regions where personal hygiene is at a low level. Bruns' describes a symptom complex consisting of a sudden onset of violent headache, vomiting, tinnitus and vertigo, passing on in the more severe attacks to deep coma and even death, and often precipitated by sudden movements of the head. It was considered to be diagnostic of a solitary cysticercus in the fourth ventricle with acute intermittent obstructive
hydrocephalus. The solitary intraventricular cysticercus is, however, not the only cause of acute obstructive hydrocephalus.

Bickerstaff et al. describe a racemose form of cysticercosis, which is apt to form at the base of the brain and, being a mobile structure sometimes with a long pedicle, is capable of intermittent blocking of the roof foramina of the fourth ventricle. Disturbance of equilibrium and spasmodic ataxia may accompany attacks of obstructive hydrocephalus; more progressive disturbance of equilibrium without marked ataxia of the limbs when supine accompanies posterior fossa racemose cysticercosis, simulating a tumour of the vermis of the cerebellum.

Lesions in the ventricular system or in the posterior fossa, followed by long-standing raised intracranial pressure, may lead to impairment of vision and consecutive atrophy of the optic disc. Involuntary movements of different localization and grand mal seizures have been described as consequences of cerebral cysticercosis. Changes in personality in patients suffering from cerebral cysticercosis have been noted.

**CONCLUSION**

Two cases of generalized cysticercosis with cerebral infestation are reported in immigrants to Canada. In one, evidence of myocardial infestation was obtained; in the other a personality pattern disturbance was brought to light. The importance of an accurate personal history in regard to soil and seed of the infestation and the manifold symptoms is considered. The diagnostic value of radiography of the thighs, a selective site of calcific densities of cysticercosis, is pointed out. The need for awareness of the possibility of cerebral cysticercosis in immigrants from South America, Mexico, Eastern and Southern Europe and India is stressed.

**REFERENCES**


**RESIDUAL PULMONARY COCCIDIOIDAL GRANULOMA**

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Coccidiodomycosis is an acute or chronic pulmonary infection caused by the fungus *Coccidioides immitis*. It is endemic in parts of the southwestern United States and Central and South America. Most cases which occur elsewhere can be traced to residence or travel in the endemic regions, as in the case reported here, or to contact with materials from these regions, as in the Canadian case reported by McLetchie et al. That coccidiodomycosis may occur in an unapparent or asymptomatic form and leave residual pulmonary granulomata has recently become better appreciated. However, in areas where coccidiodomycosis is not ordinarily encountered, the coccidiodial residual may be overlooked in the differential diagnosis of so-called "coin lesions". Attention to a history of residence or travel in endemic areas and application of the coccidiodin skin test may supply leads.

The etiological diagnosis of chronic granulomata in surgical specimens by routine histological techniques often presents considerable difficulty. Simple techniques for the identification of possible etiological agents are, therefore, of considerable interest. In the present case, the diagnostic spherules of *Coccidioides* were demonstrated in a pulmonary granuloma by a concentration method hitherto not applied for this purpose.

Mr. E.O., a 35-year-old farmer, was referred to the University of Alberta Hospital on June 19, 1955, because of a single discrete chest lesion discovered on a film taken by the mobile X-ray unit on April 18, 1955. His only complaint was of a productive morning cough which had been present for years and had not changed.

He had worked in Arizona from February 1951 to February 1952, and stated that "valley fever" was prevalent in this region. He could recall no fever or illness during this period.

His temperature and sedimentation rate were normal. His chest was clinically clear. Chest X-rays and tomograms confirmed the presence of a single discrete opacity about 2 cm. in diameter in the apical segment of the left lower lobe (Fig. 1). Sputum smears were negative.

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