INTRODUCTION

Cysticercosis is the somatic infestation or the systemic form of infection by *Cysticercus cellulosae*, the larval stage of *Taenia solium*. The invaded organs in order of frequency are the subcutaneous tissue, brain, eye, muscles, heart, liver, lung and peritoneum (Marsden, 1971).

Cerebral cysticercosis has a world wide distribution and many cases have been reported from Mexico (Obrador, 1948), South America (Arana Iniquez and Asenjo, 1945), India (Bajpai and Bhattacharya, 1974), Africa (Gelfand and Jeffrey, 1973), Rumania (Arseni and Samitca, 1957). The disease is now encountered with greater frequency in the United States (White *et al.*, 1957) and Great Britain (Wyburn-Mason and Shaikh, 1973). The prevalence of cysticercosis cerebri is relatively high in certain countries of the Far East because of the greater frequency of intestinal infestation with *Taenia solium* (Dent, 1957).

A few cases of cysticercosis have been reported in Indonesia (Hausman *et al.*, 1950; Soebroto *et al.*, 1960; Adnjana and Djojoprakoso, 1961) and in most of these cases the cysticerci were located in the subcutaneous tissue, muscles and tongue.

Neurological symptoms were also present in some cases, such as in the case reported by Lie *et al.*, (1955). In their case cerebral cysticercosis was confirmed by histopathological examination. This paper presents another case of cerebral cysticercosis from Indonesia, the diagnosis of which was based on autopsy findings.

CASE REPORT

A 35-year-old Indonesian male from Irian Jaya was admitted to the hospital for severe burns on his left foot. He had a past history of fits of 4 months duration. One night while sleeping in front of an open fire, a child saw that the patient’s foot was on fire and pulled it away. The patient was unconscious and had no memory of the accident on waking up a few hours later.

General physical examination revealed an athletic, well-nourished man. No subcutaneous nodules were detected. A third to fourth degree burn was noted on the lower half of his left leg. There were no signs of other neurological disturbances.

Laboratory data: Haemoglobin 12 gm %; leucocyte count 8,800 per c.mm; polymorphs 60%; lymphocytes 36%; eosinophils 2%; monocytes 2%.

Urinalysis was normal and *Ascaris* eggs were found in his stool sample. A below knee amputation was performed. A few hours after recovery from anesthesia, the patient became dyspneic associated with tremor of his legs and hands. Aspiration pneumonia was not present. Despite medications his condition became worse and he died 18 hours after operation.

Autopsy findings: An extensive search of the skin and musculature for evidence of cysticercosis was negative. The heart, lungs and aorta were unremarkable. The liver and spleen were not enlarged and the kidneys appeared normal. The intestines showed no
significant macroscopical changes, and no tapeworm was present in the bowel contents. The eyes were not examined. On removal of the calvaria, no abnormality of bone tissue or of the epidural space was seen.

The dura of the convexity and base, the falx and tentorium were smooth and white. The dural sinuses were free of thrombi or constriction. The subdural space contained no accumulations of fluid, blood or exudate. The brain had been removed in its entirety and weighed 1,300 grams.

The cerebral and cerebellar hemispheres were symmetrical, without shifting of the inter-hemispheric fissure. On the upper part of the right frontal lobe, the lower part of the left frontal lobe and the lateral aspect of the right parietal lobe, 5 small cystic nodules were visible, measuring from 2 to 4 mm in diameter. The pia-arachnoid over the surface of some of these nodules was opaque. The cerebral vasculature was free of arteriosclerosis, thrombosis or aneurysm. There were no malformation of the circle of Willis. The surfaces of the cerebellum, brainstem and cranial nerves were not remarkable. The cut surfaces of the cerebrum revealed multiple and scattered cystic nodules involving all lobes of both cerebral hemispheres, measuring from 2 to 7 mm in diameter (Fig. 1).

These cysts contained clear fluid and were lined by thin, shiny grey membranes. A firm whitish mural nodule projecting into the lumen of the cyst was seen. In all over 20 nodules were counted throughout the brain, the majority being located in the cortical layer. Other locations were the left basal ganglia, near the left lateral ventricle, in the white matter of the right temporal lobe. A cyst measuring 8 mm in diameter was present in the subarachnoid space of the right inferior cerebral fissure (Fig. 1). No cysts were present in the brainstem and cerebellum. The ventricles were of normal size and symmetrical.

Microscopic findings: Sections through many of these cystic nodules revealed various histological pictures. The wall of the cysts in some places consisted only of compressed cerebral tissue with slight lymphocytic infiltration and minimal glial response showing reactive astrocytes (Fig. 2). In others, the walls showed marked fibrosis and intense granulomatous inflammatory reaction with infiltration of lymphocytes, plasma cells, macrophages, epithelioid cells, and giant cells of both foreign body and Langhans' type (Fig. 3). Eosinophils were not seen. Perivascular cuffing of round cells was present in the
Cysticercosis Cerebri in Irian Jaya, Indonesia

Fig. 3—The wall of the cyst showing fibrosis, granulomatous inflammatory reaction with epithelioid and giant cells. The subarachnoid space (on the left) with chronic inflammatory cells.

Fig. 4—Photomicrograph of the parasite (in the centre) and minute foci of calcification.

Fig. 5—Section through a cystic nodule revealing a scolex with suckers and highly refractile hooklets.

Discussed

Cysticercosis is rarely encountered in Indonesia. Between 1950 and 1971, only 5 cases have been reported (Hadidjaja et al., 1971), all of which were from people living in Java and Bali. During a 6-month period in 1972-1973, 13 cases of cysticercosis were diagnosed by Tumada and Margono (1973 a) at the Enaratatali Hospital, Irian Jaya. Stool examinations on 170 hospitalized patients revealed Taenia eggs in 9% (Tumada and Margono, 1973 b).

In this area people are accustomed to place the pigs on a hot stone and eat the partially cooked meat. However, devouring measly, half-cooked pork does not necessarily cause cysticercosis. Food contaminated with Taenia eggs is probably an important factor in causing cysticercosis, as personal cleanliness and hygiene habits are still far below standard in this region. In this case no tapeworm was recovered, therefore the disease was probably contracted due to hetero-infection.

The brain is the most common site of encystment besides the subcutaneous tissues and muscles (Obrador, 1948; Reddy, 1951; Greenspan and Stevens, 1961). Arseni and Samitea (1957) found 90% of their cases the cysticerci were limited to the brain; 4% was found in the muscles, 4% in the subcutaneous tissues and 2% in the retina. Arana Iniguez and Asenjo (1945) reported intracranial cysts in 40% to 82% of human cysticercosis with a lesser likelihood of localization in the muscles and subcutaneous tissue. In our case no subcutaneous nodules were found and...
were only detected in the cerebrum which was similar with the cases of Lie et al.,(1955) and Tumada and Subianto (unpublished data, 1976). During autopsy it was found that most of the cysticerci were located in the cortical layer, involving all lobes of both cerebral hemispheres. Histopathological examination showed that the tissue reaction around the cysticerci consisted of marked fibrosis and intense granulomatous inflammatory reaction with edema which was similar to the findings of Bhaskaran (1973) and was responsible for the epileptic fits. The spotty calcification and the minimal tissue reaction surrounding many of the cysticerci suggested that the infection had taken place comparatively recently. At the time of infestation and during the stage of calcification, the larger amount of metabolic products elaborated is responsible for the epileptic seizures (Arseni and Samitca, 1957).

Cysticercosis cerebri as a causative factor of epilepsy has been stressed by many authors (Obrador, 1948; Gelfand and Jeffrey, 1973; Bajpai and Bhattacharya, 1974). So well-known is this relationship, that the diagnosis of cysticercosis cerebri is unlikely to escape consideration in a patient suffering from convulsions who has lived or served in India or the Far East (Bickerstaff, 1955); in this aspect Irian Jaya, Indonesia should be included.

Other clinical manifestations of cysticercosis cerebri besides epilepsy are increased intracranial pressure, mental disturbances, loss of vision and signs and symptoms resembling brain tumor (Reddy, 1951; White et al., 1957; Dent, 1957). Except for the epileptic seizures none of those manifestations were noticed in our case.

SUMMARY

A case is presented in which epilepsy was caused by diffuse involvement of cysticerci in the cortical layer of both cerebral hemispheres. The diagnosis was confirmed by histopathological examination. In the area of Irian Jaya cerebral cysticercosis should always be taken into consideration as the possible cause of epilepsy.

ACKNOWLEDGEMENTS

The authors wish to thank Dr. Alex Tandian of the Department of Anatomic Pathology, Faculty of Medicine, University of Indonesia for making the photomicrographs, and to Dr. Sadikin Darmawan for his valuable suggestions in preparation of this manuscript.

REFERENCES


Cysticercosis Cerebri in Irian Jaya, Indonesia


