Sparganosis of the spinal cord

Case report

C. F. Fung, F.R.C.S.(Edin), F.R.C.S.(Glas), Thomas H. K. Ng, M.R.C.Path.(UK), and W. T. Wong, Ph.D.(HK), F.R.C.P.A.

Division of Surgical Neurology (Department of Surgery) and Departments of Pathology and Microbiology, University of Hong Kong, Hong Kong

Sparganosis of the spinal cord is a rare disease: only two cases have previously been described. A third case of spinal sparganosis is reported in which a sparganum was removed from the central canal of the patient’s spinal cord. The clinical presentation and pathology are described. A brief review of the life cycle of the Spirometra is given and the possible mode of infestation is discussed.

KEY WORDS sparganosis • spinal cord • spinal granuloma • Diphyllobothrium mansonioides • Spirometra

HUMAN infestation with sparganum, which is the larval stage of the genus Spirometra, was first reported by Patrick Manson in 1882.7 Sparganosis of the brain has been reported on many occasions; however, sparganosis involving the spinal cord is a rare condition. Only two cases have previously been reported.9,10

Case Report

This 22-year-old Chinese man grew up in a family of boat people. He had a history of eating raw fresh-water fish in hot congee since he was a boy. In 1985, at the age of 19 years, he started to have repeated attacks of fever, chills, and rigidity. Three months after the onset of these symptoms, he suffered a sudden attack of back pain at the upper lumbar region. This was followed by incontinence of urine a few hours later. During the next few months, he experienced progressive numbness and weakness of both lower limbs.

First Operation. In 1985, the patient was investigated in Macau, a small city near Hong Kong, and a T7-8 laminectomy was performed. No pathology was found at that level. Laminectomy was also carried out at the upper thoracic level for an unknown reason. There was no improvement after the operation. The patient then went to China, where he was again examined; magnetic resonance imaging of the spine demonstrated widening of the spinal cord at T-9.

Second Operation. The patient presented to our department in April, 1988, with further deterioration of lower limb strength. On examination, he exhibited loss of sensitivity to pain and temperature and proprioceptive sensation below T-8. Muscle power of the lower limbs was Grade 3, with significant clonus and brisk reflexes. The white blood cell (WBC) count on May 10, 1988, was 5.6 x 10⁹/liter. The differential count was: neutrophils 60%, lymphocytes 33%, monocytes 3%, and eosinophils 4%. A myelogram and a computerized tomography (CT) scan demonstrated widening of the cord, centered at the T-9 level.

On May 17, 1988, a laminectomy was performed from T-8 to T-10. The spinal cord at that level was found to be swollen. A T8–9 myelotomy was carried out at the midline, and a firm well-encapsulated solid mass was found in the center of the cord and removed with blunt dissection. The mass measured 1 x 0.5 cm, and contained a small amount of pus-like fluid in the center. At the upper end of the myelotomy a small worm was retrieved from the central canal.

Postoperative Study. The patient showed marked improvement in muscle power of the lower limbs after operation. A CT scan of the brain performed shortly after the operation was normal. On May 17, 1988, the WBC count was 12 x 10⁹/liter, and the differential count was: neutrophils 84%, lymphocytes 14%, monocytes 1%, and eosinophils 0%.
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Pathological Study. The worm was white, flat, and ribbon-like, measuring 40 mm in length and 1 to 2 mm in width (Fig. 1). Its club-shaped head had a vertical groove (Fig. 2) and the body showed irregular pseudosegmentation. There were no scolex or internal organs. Microscopically, the body wall consisted of a tegument with microvilli, smooth muscle, and tegumental cells. The parenchyma was composed of loose stroma, calcareous bodies, mesenchymal fibers, and smooth muscle. The worm was identified as a *Spirometra* species.

The operative specimen consisted of four biopsy fragments each measuring 4 to 5 mm in size. They were pinkish, with small foci of yellowish material. Histologically, fibrinoid necrotic areas were evident surrounded by granulomatous reaction (Fig. 3). There was marked mononuclear inflammatory infiltration, mostly of plasma cells and lymphocytes. Eosinophils were present but not prominent.

Discussion

Sparganosis was first described in China by Patrick Manson in 1882.7 Most commonly, infestation involves subcutaneous soft tissue and superficial muscle fascia of the human body.3 Sparganosis of the central nervous system is uncommon and spinal involvement is extremely rare.2 To date, only two cases of intradural spinal sparganosis have been reported,5,10 both of them caused by *Sparganum proliferum*. Two cases of extradural spinal sparganosis were reported in Korea.8,11

Sparganosis is endemic in China, Japan, Korea, southeast Asia, and the United States.1,3,7,12 The majority of human infestations are caused by larvae of the genus *Spirometra*. The life cycle of *Spirometra* has been well documented.5,7,12 The definitive hosts of the adult worm are dogs and cats. Eggs passed in the feces develop into coracidia in fresh water. After being eaten by Cyclops, the coracidium develops into a procercoid larva. The second intermediate hosts are fish, snakes, and amphibians. When the infected Cyclops is eaten, the procercoid penetrates the intestine wall and migrates to various organs and tissue where it develops into a plerocercoid larva, the sparganum. The sparganum will develop into an adult worm when a dog or cat eats the flesh of the second intermediate host.

Human infestation can result from drinking water contaminated with infected Cyclops or from eating the flesh of a second intermediate host containing the sparganum. Once absorbed, the parasite will burrow through the intestinal wall and finally encyst again as sparganum in human tissue without undergoing any further development.7 The most likely source of infestation in the present patient was the raw fresh-water fish in hot congee, a delicacy for Chinese.

This patient had chills, rigidity, and back pain immediately before the onset of paraplegia. This may suggest that the larva induced a strong systemic reaction when it entered the spinal cord and subsequently lodged in the central canal. The patient's progressive deterioration after the episode is obviously due to an inflammatory reaction induced by the worm, with granuloma formation.

Differentiation of this pathology from intramedullary spinal tumor is difficult before operation. The only hint to the diagnosis is a slight increase in the eosinophil
count, a finding that was also reported in another case. Treatment of the condition is surgical excision. This patient showed significant improvement after surgical removal of the worm and granuloma.

References

Manuscript received August 26, 1988.

Address reprint requests to: C. F. Fung, F.R.C.S., Division of Surgical Neurology, Department of Surgery, University of Hong Kong, Queen Mary Hospital, Hong Kong.