Sparganosis: A Brief Review

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INTRODUCTION

Sparganosis, first described by Manson in 1882, is a parasitic zoonosis caused by plerocercoid larvae of Pseudophyllidea tapeworms mainly recognized in Spirometra genus. Many species of Spirometra are recognized in clinical diseases including Spirometra mansonii, S. ranarum, S. mansonioides, S. erinacei, and the aberrant Sparganum proliferum. The genus identification can only be indicated in adult worms which complete their life cycle in definite host; dogs or cats. Humans are accidental intermediate hosts and worms develop into plerocercoid larvae which migrate into the tissue and cause local inflammation at the final sites.

Epidemiology

Although several cases have been reported around the world, Eastern and South East Asia are mainly affected. There were sporadic cases in North America and Europe. In Thailand, there were 34 cases reported by Wiwanitkit et al in 2005. Males were slightly less affected than females with the ratio of 15:19. The mean age was 34 ± 12.8 years (range 11-60 years). The risk factors could be identified in 14 cases which included drinking untreated water, and history of eating snake or frog.

Life cycle

As shown in Figure 1, mature Spirometra worms inhabit in the intestines of dogs and cats. Eggs are released into fresh water and hatch into the coracidia, which are ingested by copepod (crustaceans). Coracidia develop into procercoid larvae in the copepod; primary intermediate host. Infected copepods are ingested by secondary intermediate hosts including reptiles, amphibians, fish, and procercoid develop into plerocercoid. The plerocercoid develops to mature worms in definitive hosts; canine. Humans are intermediate hosts for Spirometra worms which develop sparganosis, and cannot serve for definite hosts.

Mode of transmission

Humans are accidental host by
1. Drinking untreated water containing infected copepods
2. Ingesting raw or inadequately cooked second intermediate hosts e.g. flesh of snakes or frogs infected with the Spirometra larvae
3. Applying the flesh of an infected intermediate host as a poultice on the open wounds, lesions, or eyes e.g. for medicinal or ritualistic reasons.

The first two are the major mode of transmission.
Clinical manifestations

Spargana cause various symptoms depending on the organs or tissue involvement. They could settle on subcutaneous tissue as nodule, visceral organs including lungs, abdominal viscera, and also vital organs such as the brain. The reported incubation period ranges from a few weeks to years.\(^9\) However the larvae can live in the tissue more than decades before symptoms happened.\(^9\)\(^\text{-10}\)

After ingesting the infected copepod from untreated water or uncooked intermediate host, the larvae will penetrate the intestinal wall into peritoneum, migrate painlessly systemic to any site of the human body for growing and elicit pain with local inflammation at the final site of invasion. Eyes and skin are the most affected final location.\(^3\)

Ocular manifestation was the first leading symptom in the series of Thai patients, followed by subcutaneous tissue and central nervous system which presented with progressive neurodeficit and had the worst prognosis.\(^6\)

Others unusual organ involvements e.g. bone, pulmonary, oral cavity and proliferative sparganosis have also been reported in Thailand.\(^11\)\(^\text{-13}\)

Sparganosis can be divided into 2 types as follows;

1. **Proliferating type** is caused by *S. proliferum*, mainly reported outside the United States including Japan, Venezuela, and Paraguay.\(^14\)\(^\text{-16}\) The larvae could proliferate as a parasite in humans, with more than one plerocercoid in one lesion, and spread to other part of organs.\(^3\)\(^\text{-5}\) Most cases were fatal.

2. **Nonproliferation type** is usually caused by remaining of species of Spirometra. They could not proliferate and find only one plerocercoid larvae in one lesion. *S. mansonoides* are the major acquired infection in United States.\(^3\)

Diagnosis

The definite diagnosis is usually made by direct visualization of parasite, and pathological examination of tissue biopsy. The length of larvae is a few
millimeters to a centimeter, and white, ribbon-like, wrinkles are the gross morphology. The recorded length in Thailand was more than 2 mm.6

The serological testing by monoclonal antibody IgG with enzyme-linked immunosorbent assay (ELISA) has been documented as optional for diagnosing cutaneous and cerebral sparganosis. Clonorchis and Paragonimus infections may cause positive result for this test.3,17

Imaging

Brain computer tomography and magnetic resonance imaging is a useful tool for guiding the diagnosis, determining severity and excluding other possibilities. Frontoparietal lobe, basal ganglion, and external capsule are the most common invasion sites. Although there are no specific signs for definite diagnosis, there are some suggestive findings as follows.

Computer tomography may show an extensive low density of white matter with adjacent ventricular dilatation, an irregular nodular enhanced lesion, and changing in the location of the enhancing nodule punctuate calcifications.18

Magnetic Resonance imaging may show widespread white matter degeneration, cortical atrophy, ipsilateral ventricular enlargement, decreased volume of the ipsilateral crus cerebri, and tunnel sign on postcontrast MRI or bead-shaped enhancement.19

Treatments and Preventions

Surgical resection and larvae removal are the best treatment.3,5 Further damage can be prevented when live larvae are removed from the vital organs such as brain. Removing the dead larvae and granuloma might not improve the neurological deficit in cerebral sparganosis cases.18 The antihelminth including mebendazole, albendazole, and praziquantel have not been shown to be beneficial. Direct ethanol injection and flash freezing may be another treatment option.3,5

References


