

ECHINOSTOMIASIS—A SNAIL-BORNE INTESTINAL TREMATODE ZONOSIS*

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Abstract. Numerous echinostome trematodes are found in the intestines of birds and mammals throughout the world, and echinostomiasis in humans has been attributed to approximately 16 different species. In humans it is usually regarded as a rare intestinal parasite of little clinical importance except in heavy infections. Diagnosis of echinostomiasis is made by identification of eggs during fecal examination; however, speciation of echinostomes requires morphological study of adult worms following anthelmintic treatment. The complex life cycles of echinostomes are all linked to freshwater habitats. A mammalian or avian definitive host, one or two molluscan hosts, and one or two freshwater stages are usually required to complete the life cycle. In addition, amphibians and fish have been implicated in the transmission of some species. Prevention of human cases is dependent on eating habits, since raw or insufficiently cooked molluscs, and to a lesser extent fish and amphibians, are sources of infection for humans. Human cases have been effectively, albeit accidentally, controlled by the introduction of fish which prey on the larval stages of the essential molluscan hosts.

INTRODUCTION

Echinostomes, next to schistosomes, have probably been studied more than any other group of trematodes. In the past 10 years there were more than 500 papers on echinostomes published in journals indexed by Biological Abstracts. The intense interest in echinostomes is not generated by the morbidity they cause in either animals or humans; rather, they are ubiquitous trematodes whose life cycles are relatively easy to establish and maintain in a laboratory. Huffman and Fried (1990) reviewed the Genus *Echinostoma* and echinostomiasis and provided a summary of information on the biology, life history, infectivity, immunology, pathology, epidemiology, physiology and biochemistry of this group of echinostomes.

Echinostomes are common intestinal parasites of aquatic birds, rodents and to a lesser extent other mammals such as dogs, cats and pigs

that phylogenetically are included in the Family Echinostomatidae. They are small elongated flukes, 2–6 mm in length by 1.0–1.5 mm in width, with a large ventral sucker and with one or two rows of large spines surrounding the oral sucker; hence the name “spiny mouth”. The number and arrangement of the circumoral spines and the topography of the genital systems are used to differentiate species. There is considerable taxonomic confusion; however, the systematics of some 37-collar-spined *Echinostoma* group, commonly used in experimental research, was thoroughly revised on the basis of morphological, biometrical, isoenzymatical and biological characteristics (Kanev, 1985 as summarized by Huffman and Fried, 1990). Undoubtedly, the application of the same analytical methods to the rest of this trematode family would result in considerable lumping and splitting of current species.

LIFE CYCLE

The potential life cycle paths of an echinostome are varied. There is always a sylvatic cycle, in many cases a zoonotic cycle and in some cases a human cycle. Echinostomes

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require a vertebrate definitive host, a molluscan intermediate host and at least one aquatic free living stage. Echinostomes, however, usually require two separate molluscan hosts to complete their life cycle and in some cases utilize alternate second intermediate hosts such as fish or amphibians. Sources of infection for humans usually can be traced to ingestion of raw or insufficiently cooked molluscs, fish or amphibians. *Echinostoma cinetorchis* illustrates the variety of paths echinostomes can utilize. Rats are the primary definitive hosts and freshwater molluscs serve as both first and second intermediate hosts; in addition, several kinds of frog and salamander larvae, and freshwater fish serve as second intermediate hosts (Lee *et al*, 1990). Human cases are attributed to ingestion of uncooked freshwater fish.

EPIDEMIOLOGY

Human cases, although worldwide in distribution, are most frequently seen in Asia where at least 16 species of echinostomes have been recovered from human infections (Rim, 1982; Huffman and Fried, 1990; Radomyos *et al*, 1985). Human cases occur focally in areas where freshwater molluscs, fish or amphibians are eaten raw or insufficiently cooked.

In Korea, at least three echinostomes have been recovered from human infections: *E. cinetorchis* (Lee *et al*, 1990; Seo *et al*, 1980) and *Echinostoma hortense* and *Echinostoma japonicus* (Hong *et al*, 1989). Human cases in Korea are usually attributed to eating raw or insufficiently cooked fish.

In Indonesia, five species of echinostomes have been reported from humans (Carney *et al*, 1980). They are: *Echinostoma ilocanum*, *Echinostoma malayanum*, *Echinostoma revolutum*, *Echinostoma echinatum* (= *E. lindoense*) and *Echinoparyphium recurvatum*. Infection rates are usually low (1%); however, there was a highly endemic focus of human cases in the Lindu Valley of Central Sulawesi where infection rates ranged from 24% to 96%. In Indonesia human cases are attributed to eating freshwater snails and clams.

In the Philippines, two echinostomes have been reported from man: *E. ilocanum* and *E.*

malayanum (Monzon and Kitikoon, 1989). In surveys of rural populations, echinostome-like eggs were detected in most areas surveyed but generally low rates (1%) (Cross and Basaca-Sevilla, 1986). An exception was encountered in Northern Luzon, where *E. ilocanum* eggs were found in the feces of over 10% of persons examined (Cross *et al*, 1970). In Northern Luzon the large rice field snail, *Pila luzonica*, is a second intermediate host and, apparently, a preferred delicacy for the inhabitants.

In Malaysia and Singapore, only *E. malayanum* has been reported from man (Rim, 1982).

In India, there are few case reports of human echinostomiasis. *Paryphostomum sufrartyfex* was reported from a child in 1915. Recent cases of undetermined echinostome species were reported in a tribal community near Calcutta (Bandyopadhyay and Nandy, 1986) and in coastal communities.

In Japan, there are numerous reports of human infections with echinostomes based on the recovery of eggs in human stool specimens (Rim, 1982). Eating raw freshwater fish is considered the primary source of most *E. cinetorchis*, *E. hortense* and *E. japonicus* cases.

In Taiwan, three species of echinostomes *E. recurvatum*, *E. revolutum* and *Echinostoma melis* have been recovered from humans (Lu, 1982). There, freshwater clams, eaten raw, half boiled or pickled over night, are considered the main source of human cases. The prevalence of echinostome eggs in stool surveys throughout Taiwan varied from 11 to 65%.

In Thailand, four echinostomes have been recovered from man in the northeast provinces: *E. malayanum*, *E. revolutum*, *E. ilocanum* and *Hypoderaeum conoideum* (Radomyos *et al*, 1982). In one region of northern Thailand, echinostomes were found in approximately 50% of the villages surveyed (Sornmani, 1969) where most infections were due to *Hypoderaeum conoideum*. Raw snails and tadpoles were identified as the source of human cases. *Episthmium caninum* was recently recovered from a Thai farmer from Roi Et Province (Radomyos *et al*, 1985). It was suspected that he was infected through consuming raw fish.

Although most human cases are reported from Asia, they do occur elsewhere (Rim, 1982). Recently an outbreak of parasitic gastroenteritis was reported among travelers who returned to the United States from Africa (Poland *et al*, 1985). Eighteen of 20 American tourists who traveled to Kenya and Tanzania were passing echinostome-like eggs in their stools.

DIAGNOSIS

The recent increase in immigrants from tropical to temperate countries and the growing popularity of international travel challenge the diagnostic talent of microbiologists and physicians. The diagnosis of echinostomiasis is still done the old fashion way: recovery and identification of eggs in the feces. In fresh fecal matter the eggs are unembryonated, operculate, ellipsoidal and yellow or yellowish brown and range considerably in size depending on the species. Echinostome eggs must be distinguished from unembryonated, operculated eggs of other trematodes such as *Fasciola hepatica* and *Paragonimus westermani*. Specific diagnosis requires anthelmintic treatment to recover adult worms for examination of morphological characters used to differentiate species.

DISEASE IN HUMANS

The clinical aspects of echinostomiasis are still meager even though there have been case reports since 1917 (Hillarrio and Wharton, 1917). Echinostomes are not very pathogenic. In light infections, significant symptoms are usually not found. Clinical manifestations are related to the number of worms. When the number is large, there may be eosinophilia, abdominal discomfort, soft stools, diarrhea and anorexia (Hong *et al*, 1989; Seo *et al*, 1980; Ryang *et al*, 1985; Lee *et al*, 1988; Bandyopadhyay, 1986; Sun, 1982). Pathologically, echinostomes damage the intestinal mucosa with inflammation and ulceration of the mucosa characterizing heavy infection. Harada *et al* (1983), using endoscopic examination of the upper jejunum in patients infected with *E. hortense* and *E. japonicus*, demonstrated graphically multiple intestinal erosions in association with the intestinal niche occupied by these flukes.

DISEASE IN ANIMALS

Light infections of echinostomes in animals do not cause significant pathology or symptoms, but heavy infections are associated with serious pathology, disease and even death. In fowl, hemorrhagic diarrhea, progressive emaciation, weakness in flight, and death have been associated with infections of hundreds to thousands of flukes (Rim, 1982). The flukes insert their anterior ends deep between the villi and appear to feed on the tissues with their oral suckers; this leads to desquamation of the epithelium and the villi, and there is a marked cellular reaction with associated edema and thickening of the mucosa (Soulsby, 1965 as cited in Rim, 1982). Histopathological changes in the intestines of rats infected with *E. hortense* showed villous atrophy, crypt hyperplasia and stromal inflammation (Noh, 1989 as cited in Hong *et al*, 1989).

TREATMENT

Recent comprehensive reference texts on the chemotherapy of tropical parasitic diseases provide meager guidance for the treatment of intestinal trematodes such as echinostomes (Campbell and Garcia, 1986; Gustafsson *et al*, 1987; Cross, 1985). The latter text indicates that drugs used for other intestinal trematodes are generally effective in the treatment of echinostomiasis. However, a number of anthelmintics have been demonstrated as effective for treatment of echinostomiasis in man. These include mebendazole (Notteghem *et al*, 1980; Cross and Basaca-Sevilla, 1986), albendazole (Pungpark *et al*, 1984), praziquantel (Seo *et al*, 1983, 1985; Lee *et al*, 1988; Radomyos *et al*, 1982, 1985); bithionol (Seo *et al*, 1980); hexylresorcinol crystoids (Rim, 1982) and nicolsamide (Rim, 1982). Campbell and Garcia (1986) note that tetrachloroethylene and carbon tetrachloride have been used successfully to treat intestinal trematodes of birds and mammals; however, they recommend less toxic and more effective compounds such as nicolsamide, oxyclozanide, rafoxanide or praziquantel.

PREVENTION

Preventive measures already exist; raw or undercooked molluscs, fish and amphibians

should not be consumed. Although this parasitosis could easily be eliminated by simply altering eating habits, long-standing traditions are difficult to change. Health education programs, marketing preventive measures, are essential to breaking the zoonotic or human cycles of transmission.

CONTROL

The minor nature of this parasitosis does not justify the establishment of control programs; however, echinostomiasis has been controlled, albeit unintentionally, in one region of Indonesia (Carney *et al*, 1980). The original description of *E. echinatum* (= *E. lindoense*) and the account of part of its life history and epidemiology in 1940 was of particular interest to parasitologists because prevalence rates in the human population of the Lindu Valley in Central Sulawesi were high, ranging from 24 to 96% (Sandground and Bonne, 1940). Surveys conducted from 1937 through 1956 reconfirmed the high rates of human cases from repeated exposures and subsequent re-infections.

Corbicula spp. were incriminated as the primary source of human infections. Mussel beds were plentiful along the shore of Lake Lindu (Bonne, 1939) and mussels were a substantial item in the local diet. Piles of mussel shells were found below each house in the village where 96% of the population were infected. Residents ate raw or insufficiently cooked mussels, became infected and established a human cycle of echinostomiasis in the Lindu Valley.

During the 1970s stool specimens all the residents of the Lindu Valley were examined but no echinostome eggs were found (Clark *et al*, 1974). *Corbicula*, previously incriminated as the primary source of human infections, had essentially been eliminated from the diet of residents. The *Corbicula* population and other freshwater molluscs, common along the shore line of Lake Lindu in the 1940s and 1950s, were no longer found (Carney *et al*, 1980). Only one small mussel bed was found at the outlet of the lake and this mussel bed was only accessible to humans during abnormal dry periods.

The reason for the decline in the *Corbicula* and other freshwater molluscs of Lake

Lindu was attributed to major changes in the ecosystem of lake following the introduction of an non-indigenous freshwater fish (Carney *et al*, 1980). In the 1950s the Mujair fish, *Tilapia mossambica*, was introduced; it thrived and in the 1970s it was the most common fish in the lake. During the same time frame there was a corresponding decline in the freshwater molluscan fauna, particularly the *Corbicula* population. *Corbicula* are phytoplankton feeders as are young omnivorous *T. mossambica*. The decline and near extinction of *Corbicula* was probably due to competition of the Mujair fish for phytoplankton and/or predation by the Mujair fish, known to be zooplankton feeders, on the veliger stage of *Corbicula*.

The disappearance of echinostomiasis from the human population of the Lindu Valley illustrates a successful interruption of a parasite's life cycle leading to the disappearance of a human disease. However, since naturally infected freshwater molluscs and mammals were found in all accessible areas surrounding the lake, the sylvatic cycle was intact and the zoonotic capability still existed; but, without a new channel to man, human echinostomiasis in the Lindu Valley will remain a disease that disappeared (Carney *et al*, 1980). The lesson learned in the Lindu Valley is simple. Any change which modifies eating habits of man can eliminate human cases of a food-borne parasitic disease just as the Mujair fish eliminated the *Corbicula* intermediate host that channeled the disease to the human population in the Lindu Valley. Health education is such a stimulus.

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