CHAPTER 23

Schistosomiasis Japonica — Clinical Features

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1 Introduction

Schistosomiasis japonica is widely distributed in Asia, particularly in China, the Philippines, Japan, Laos, Vietnam, South Celebes, Thailand, etc., and the estimated infected population is about 100 million [1,2]. In the central region of the People’s Republic of China along the Yangtze River Basin, this disease was most rampant because of the numerous water channels, small rivers and lakes where snails proliferated and people used to wash their chamber pots. During the Mao regime, the government campaigned with all-out efforts to eradicate the disease [2,3], and at one time a complete eradication was declared. In reality, however, such a goal has not yet been reached in this country. In Japan, this disease was put under control with the technical aid from the U.S.A. [4], and it is now all but extinct. There are still several thousand old people who have chronic Schistosoma (S.) japonicum infection in the previously endemic regions, and the number of symptomatic cases is perhaps several hundred.

The clinical features of schistosomiasis japonica largely depend on the degree of exposure to cercariae. A female S. japonicum produces 3500 eggs a day, 12 times the number laid by S. mansoni, and the disease it causes is generally more severe than the other types of schistosomiasis [1,5]. Mild repeated exposures result in chronic schistosomiasis without an obvious acute stage, whereas a severe acute episode will develop following heavy infection. Although this chapter is divided into acute and chronic schistosomiasis, many patients with chronic disease have never experienced typical acute schistosomiasis. In fact, acute schistosomiasis has not been seen after 1965 at Kurume University Hospital which is located in an endemic area where one of the authors (K.O.) used to see such patients; the situation is similar in other parts of Japan.

2 Acute schistosomiasis japonica

From one to several weeks prior to the onset of acute disease, the patient develops a skin lesion with a severe itch which is sometimes called “swimming itch” [1]. The lesions are characterized by miliary-to-soybean-sized red papules in the skin areas that were exposed to waters infected by cercariae [6]. Scratching changes the lesions to vesicles and pustules which are then ulcerated and are surrounded by edema. Untouched, they disappear spontaneously within a week. Young people seem to develop more severe skin lesions compared to adults whose response is milder perhaps due to certain immunologic tolerance [1].

The first presentation varies considerably from case to case, but the early toxemic signs are thought to be due to reactions to the destroyed cercariae and metacercariae within the body. In a typical case of heavy infection, the early acute toxemic stage closely mimics typhoid fever or malaria [6], characterized by chills and high remittent fever, and hepatosplenomegaly. Without serological and other tests, it may be mistaken for typhoid or paratyphoid fever. The patient complains of headache, anorexia, vertigo, cough, and severe prostration. The liver may be enlarged, extending to the umbilical level, but splenomegaly at this stage is not very marked. Fever slowly comes down within one to several weeks, the duration of the febrile period also
depending on the severity of infection. The toxemic phase is soon followed or superimposed by the intestinal phase which is caused by egg production and in which enterocolitis-like features dominate, such as abdominal pain, watery diarrhea, bloody stools mixed with mucus, and tenesmus. Clinically, the picture is very similar to that of dysentery. Unlike bacterial dysentery, however, anorexia is mild and the patient can eat. More patients present with the intestinal symptoms without experiencing the toxemic stage.

At this stage, the feces contain a large number of eggs and diagnosis is readily made by fecal examination. In severe cases, loss of blood and hypoalbuminemia induce dependent edema, or even anasarca with severe anemia, and the clinical picture is similar to that of nephrotic syndrome with anemia [6]. Leukocytosis and eosinophilia are the prominent hematological findings; the net count of eosinophils may go up to 60,000/mm³, constituting more than 95% of the peripheral leukocytes. Endoscopy of the rectum and sigmoid colon demonstrates hyperemia with dilation of superficial venules and multiple small elevated nodules of 1–3 mm in diameter occurring in small clusters [7,8]. Biopsy of such nodules reveals immature ova. These nodules turn to ulcers. Liver biopsy likewise demonstrates immature ova in the portal tract. Liver function test abnormalities such as plasma BSP retention, decrease in serum albumin, increase in γ-globulin, etc., are related to the degree of infection as determined from the number of ova excreted in feces [9]. There may be a slight increase in serum bilirubin [7].

### 3 Chronic schistosomiasis

The transition from acute to chronic schistosomiasis is insidious and there is no clear definition of the time lapse from the acute infection. The major pathology is seen in the liver where reactions to the deposited ova lead to portal fibrosis as discussed in Chapter 22. Portal venous pressure is elevated as hepatic fibrosis increases with time, producing portal outflow block in the portal tract not involving the hepatic venules (presinusoidal portal hypertension). There is a considerable gradient between wedged hepatic venous pressure and portal venous pressure as shown in Table 23.1 in which the results of such measurements in chronic schistosomiasis are given. In this study conducted in the Kofu area of Japan [10], portal venous pressure was measured intraoperatively in 65 patients who underwent laparotomy for various surgical problems. With increasing portal venous pressure, collateral veins and esophageal varices develop as in liver cirrhosis. There seems to be no difference between schistosomiasis and cirrhosis in the site of collateral route formation. In another study in Kofu in which 910 subjects positive for the schistosome skin test using an acidic soluble protein antigen [11] were studied by barium swallow examination, esophageal varices were found in 167 (18.3%); the frequency of varices was 25% in patients with no hepatic fibrosis, 44% in those with fibrosis and 59% in those with cirrhotic changes [12]. In a further study on 1524 subjects positive for the skin test, the spleen was palpable.