SCHISTOSOMIASIS OF THE BRAIN*

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Although there is much to be found in the literature concerning intestinal and hepatic involvement by Schistosoma japonicum, the reports on cerebral involvement are infrequent. Because of the extreme gravity of complications from this disease, we feel that the members of American medical profession should be aware of it as a neurological differential diagnostic problem in anyone presenting a bizarre neurological picture who has been in an endemic area. That the disease may well assume the roles played by typhoid fever in the Civil War, by influenza in World War I and by malaria in World War II was suggested at a recent meeting of the Society of American Bacteriologists.

HISTORICAL REVIEW

Fujii38 first mentioned this disease entity in 1847, but it was 42 years later before any cerebral lesions were associated with the disease65 and not until 1904 was the etiological agent discovered and named by Katsurada.37 The first autopsy reports on cerebral findings were made in 1905.58 Presumptive evidence that abnormal neurological reactions in infected patients were attributable to lesions caused by eggs or worms in the central nervous system was initially presented by Houghton43 in 1910, but not until 1935 was the first report of surgical verification of a cerebral lesion made by Shimidzu,49 which was followed by similar reports by Edgar19 in 1936, and Greenfield and Pritchard29 in 1937. No further cerebral granulomas were verified surgically until Swanson55 reported the fourth case in 1946. Since that time, 11 instances of operative and microscopic verification of such lesions have appeared in the literature.14,19,29,31,47,48,49,51,55,62 although others may have been unreported, as Chang and his co-workers14 referred to 2 cases surgically verified by Sanders, and Watson and his co-workers62 mentioned 1 case verified by Maltby and Schmidt and 1 by Chasnoff. Hunt and his co-workers34 reported 1 operative case which, however, was not verified microscopically. Faust32 mentioned 1 case verified by Palmer.

Many observers have reported on presumptive ectopic cerebral lesions.10,12,15,17,20,26,36,32,56,57 Eight authors3,4,5,23,56,61,65 reported 11 postmortem examinations on

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patients who had exhibited neurological manifestations of the disease and among these only 1 cerebellar and 4 cerebral granulomas were microscopically verified as containing Schistosoma japonicum ova. Perhaps significantly the number of surgical verifications since 1941 overshadows the number of autopsy verifications.

ENDEMIC AREAS

The disease is apparently limited to areas containing a certain species of amphibious fresh water snail known variously as Oncomelania, Katayama or Schistosomophora which presently is the only known intermediate host suitable for the larval development of Schistosoma japonicum in the field.

Known endemic areas include Japan (Honshu and Kyushu Islands), China (especially southern and central), Formosa, Korea, the Celebes and the Philippine Islands of Leyte, Samar, Mindoro and Mindanao,10,16,24,40,41,43,44,50,54,59,64

Abbott,1,2 and Berry and Rue9 have reported a species of fresh water snail having a wide range of distribution in the United States which is capable of serving as the intermediate host under laboratory conditions. Their medical significance is presently unknown, but one must recall that after World War I Australia was faced with the previously unknown problem of endemic Schistosoma hematobium due to the adaptability of a certain species of one of their native fresh water snails.

ROUTE OF CEREBRAL INFECTION

The route of larval migration was reported by Miyagawa42 in 1912, and restudied by Faust and Melney25 in 1924 whose description of the stage-by-stage development in mammals is classical. However, this deals only with the normal portocaval development and does not embrace the far distant sites of ectopic lesions.

The largest number of proven or presumptive sites of ectopic lesions in schistosomiasis japonica is in the cerebrum. However, the route of cerebral infestation is not clearly understood. Five routes have been theorized and include (a) the valveless vertebral veins or “Batson’s circulation”;7,8 (b) migration of the worms outside their normal habitat against the venous blood flow and into collateral vessels with egg deposition on reaching the end venules.23,29,45,51 It has been shown that worms do migrate out of their normal habitat and the nature of most of the verified cerebral lesions supports this hypothesis, but no worker has explained how the worms reach the cerebral venous sinuses; (c) normal development outside the hepatic-caval system;23,29 this has been disproven by the studies of Faust and Melney25 and Waelch;62 (d) the presence of a patent foramen ovale which, although a rational explanation, does not occur with enough frequency to serve as an explanation; none of the 9 autopsy reports on cerebral ectopic lesions mentions the presence of such an anomaly; (e) eggs deposited in the usual sites filtering through the capillary barriers of the liver and lungs into