the homogenous spherical enlargement of adrenals, invasion of adjacent structures, non-homogenous attenuation and a thick irregular enhancing rim. The involvement of adrenals in lymphoma is invariably associated with involvement of other abdominal organs. Solid calcified pheochromocytomas may mimmick TB, but they show marked contrast enhancement. Associated urinary catecholamine and elevated vannilyl-mandelic acid (VMA) levels will confirm the diagnosis.

It is important to diagnose the cases of tubercular adrenalitis in their early stage of enlargement as institution of specific therapy at this stage make it possible to affect complete recovery of adrenals and obviate the need for a life-long hormonal supplementation.

REFERENCES

1. Tarvinder B, Rakesh B, Sujatha, Shrishti P, Sushant M, Mathew D. CT in adrenal enlargement due to tuberculosis. A review of literature with five new cases. Clin Imaging 1992, 16: 102-108.
2. Wilms GE, Baert AL, Kint EJ, Pringot JH, Goddeeris PG. Computed tomographic findings in bilateral adreneal tuberculosis. Radiology 1983, 146: 729-730.
3. Eason RJ, Croxon MS, Perry MC, Somerfield SD. Addison's disease–Adrenal autoantibodies and computerized adrenal tomography. NZ Med J 1982, 95: 569-573.
4. Perry MC, Eason RJ, Croxson MS. Computed tomography in tuberculous Addison's disease–A case report. Australas Radiol 1983, 27: 33-36.
5. Schultz CH. CT and MR of the adrenal glands. Semi Ultrasound CT MR 1986, 7: 219-233.
6. Yee ACN, Gopinath N, Ho CS, Tao LC. Fine needle aspiration biopsy of adrenal TB. J Can Assoc Radiol 1986, 37: 287-289.

Acute Schistosomiasis in the Indian Subcontinent

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Schistosomiasis is a group of diseases caused by various species of blood flukes of the genus *Schistosoma*. About 200 million people in 73 countries of the world are affected(1). The disease is unknown in the Indian subcontinent except for a small endemic focus of *S. hematobium* in Gimvi village of Maharashtra(2). Acute schistosomiasis or Katayama fever is a clinical syndrome that occurs within three to six weeks after infection with *S. mansoni* or *S. japonicum*. Though the acute stage is self limiting, it is important to recognize this condition because if left untreated, the patient presents with complications of chronic schistosomiasis years later (*e.g.*, decompensated liver either as periportal fibrosis (Symmer's fibrosis) leading on to cirrhosis and portal hypertension or malignancy of the liver.

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Case Report

A 7-year-old girl, resident of Karachi, Pakistan was admitted with complaints of high grade remittent fever of 2 months duration and pain in right upper abdomen for 6 weeks. There was associated malaise, weakness, weight loss and anorexia. There was no history of cough, breathlessness, convulsions, bowel or bladder problems, jaundice, bleeding or passage of worms in the stool. The patient was a non-vegetarian, but had no pets at home, or had travelled to any other region in the recent past.

On examination the child was undernourished, pale and febrile. There was no jaundice, edema or subcutaneous nodules. The liver was palpable 7 cm below the costal margin and was firm and tender. The spleen was enlarged 1 cm below the costal margin. There was no evidence of free fluid in the abdomen. Other systemic examination was normal.

Investigations showed the level of hemoglobin of 7 g/dl, total leucocyte count being 10,500/cu mm with polymorphs 30%, lymphocytes 25%, monocytes 5% and eosinophils 40%. The red blood cells were normocytic normochromic, absolute eosinophil count was 2,750/cu mm and ESR 45 mm at the end of first hour. The levels of blood urea, sugar, creatinine, amylase and electrolytes were normal. Liver function tests showed blood levels of bilirubin of 0.9 mg/dl, SGOT 28 IU/L, SGPT 24 IU/L, and alkaline phosphates 1257 IU/L (normal 80-280 IU/L). Prothrombin time was prolonged to 29 seconds. The urine, stool examination and *X*-ray chest were normal. The blood culture was sterile and Widal test, Mantoux test, Hepatitis B surface antigen, serological tests for amebiasis and blood smear examination for microfilaria were negative. The ultrasound examination of the abdomen showed diffuse hepatomegaly with a normal echotexture. Bone marrow aspiration revealed preponderance of eosionphil precursors. The liver biopsy showed mild dilatation of sinusoids, with a foreign body granulomatous reaction in the lobules. The granuloma comprised of eosinophils, neutrophils and a few mononuclear cells around the eggs of *Schistosoma mansoni* which were oval shaped with a prominent lateral spine. The rheumatoid and antinuclear factors were negative. The blood levels of IgG (3218.8 mg/dl) and IgA (90.9 mg/dl) were all elevated.

The child improved slowly, with abdominal pain disappearing on the 5th day. She became afebrile about 2 weeks after the start of therapy when the hepatomegaly had regressed considerably with a general improvement in appetite and weight.

Discussion

Schistosomia mansoni infection is common in Africa, Saudi Arabia, South America and the Carribean Islands. Man is the definitive host while snail is the intermediate host. Human infection occurs when cercariae which have been shed into the fresh water by the snail penetrate the human skin. There may be a transient dermatitis (Swimmer's itch) at the local site.

Acute schistosomiasis or Katayama fever is seen in non-immune hosts and occurs when egg production commences. During this stage, eggs get deposited in the liver with formation of acute granuloma comprising of eosinophils and neutrophils(3,4). The syndrome of Katayama fever usually develops three to six weeks after initial infection and may last upto 3-4 months. The common manifestations are spiking fever, profuse sweating, abdominal pain, loss of weight and appetite, malaise, drycough with splenomegaly and tender hepatomegaly. Istre

et al. (5) described an outbreak of acute schistosomiasis among 6 cases of a group of 11 members who took part in a rafting expedition in Ethiopia. Fever, eosinophilia and tender hepatomegaly were present in most patients. Stuiver(6) described a similar clinical profile in three Dutch patients on a holiday in West Africa.

Diagnosis of acute schistosomiasis depends on the identification of eggs in stool or in liver or rectal biopsy. However, light infections and delayed onset of egg excretion usually prevent detection of eggs in stool. Eosinophilia is a conspicuous and constant feature. Normal liver function tests except for elevated alkaline phosphatase and hypergammaglobulinemia is usual. Serological tests including ELISA, CFT, IFAT and circumoyal precipitin test are useful for diagnosis(7,8). Farid *et al.* (9) have suggested that counterimmunoelectrophoresis (CIEP) using crude adult *S. mansoni* antigen is a simple rapid test for detecting the infection when stool contains no detectable eggs.

Acute toxemia is rapidly controlled with a short course of steroids after which praziquantel in a dosage of 40 mg/kg as a single dose or oxamniquine(8) should be given. Side effects of praziquantel are generally mild, including abdominal pain, nausea, diarrhea and headache. Oxamniquine has been tried in a in-hospital study for 5 years(8) in children as well as in adults in uncomplicated and also those cases with complications. All patients were given oxamniquine (20 mg/kg/day) for 5 days. There were no major side effects, only 38% of the group had a febrile reaction which lasted for 3 days.

From this study authors have reported a low cure rate (55%) in children - though since this drug is very well tolerated, it is the ideal drug in patients with decompensated liver functions.

REFERENCES

1. Iarotski LS, Davis A. The schistosomiasis problem in the world. Bull WHO 1991, 99: 115-117.
2. Gaitonde BB, Sathe BD, Mukerji S. Studies on schistosomiasis in village Gimvi of Maharashtra. Indian J Med Res 1991, 74: 352-357.
3. Rocklin RE, Brown AP, Warren KS. Factors that modify the cellular immunity response in patients infected with *Schistosoma mansoni*. J Immunol 1980, 125: 1916-1923.
4. Mahmoud AA. The ecology of eosinophils in schistosomiasis. J Infect Dis 1982, 145: 613-622.
5. Istre GR, Fontaine EF, Tarr J, Hopkins RS. Acute schistosomiasis among Americans rafting the Omo river, Ethiopia. J Am Med Assoc 1984, 251: 508-510.
6. Stuiver PC. Acute schistosomiasis (Katayama Fever) Br Med J 1984, 288: 221-222.
7. Neils de Jonge, Fillie Y, Hilberath GW, *et al.* Presence of schistosome circulating antigen (CAA) in urine of patients with *schistosoma mansoni* or *S. hematobium* infections. Am J Trop Med Hygiene 1989, 41: 563-569.
8. Andre M Deelder, Neils de Jong, Fillie Y, *et al.* Quantitative determination of circulating antigens in human schistosomiasis mansoni using an indirect haemaglutination assay. Am J Trop Med Hyg 1989, 40: 50-54.
9. Farid Z, Mansour N, Kamal K, Girgis M, Woody J, Kamal M. The diagnosis and treatment of acute toxemic schistosomiasis in children. Trans R Soc Trop Med Hyg 1987, 81: 959-963.
10. Kilpatrick ME, Farid Z, Bassily S. Treatment of *S. mansoni* with oxamniquine–five year's experience. Am J Trop Med Hyg 1981, 30: 1219-1221.