Total granulomatous infarction of testis due to *Schistosoma haematobium*

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**SYNOPSIS** This is an unusual case of total granulomatous infarction of testis due to *Schistosoma haematobium*, the first such case to be reported. This report reinforces the theory of extraportal maturation of worms in spermatic veins and capillaries as postulated earlier (Joshi, 1962).

**CASE REPORT**

A 10-year-old, well-developed but poorly nourished African boy (J.H.) was admitted to Connaught Hospital, Freetown, in January 1964 with a unilateral, slowly growing painless swelling of the right scrotum. There was no history of trauma, venereal disease, and haematuria. However, not much reliance could be placed on the history as both the child and his parents were completely illiterate. Examination of the right scrotum revealed a uniform, rather hard, painless swelling without any signs of hydrocele. The skin over the swelling was freely movable. The epididymis and spermatic cord were normal. There was no inguinal lymphadenopathy. The rest of the physical examination, apart from obvious malnutrition and anaemia, was essentially negative.

The laboratory examinations showed: haemoglobin 9 g.% and total white cell count 1,200 per cmm. (polymorphonuclears 53%, lymphocytes 30%, monocytes 2%, eosinophils 15%). The Kahn test was negative. Stool examination revealed moderate infestation with ova of *Schistosoma mansoni* and *Ankylostoma*. The urine examination showed an occasional egg of *Schistosoma haematobium* and a few pus cells and erythrocytes. X-ray studies of the chest and the skeleton were negative. A provisional diagnosis of 'seminoma' was made and orchidectomy was advised to be followed by appropriate treatment for schistosomiasis and ankylostomiasis. The post-operative course was uneventful, and after discharge we lost track of the patient.

**PATHOLOGY**

The entire specimen of testis, epididymis, and part of the spermatic cord weighed 80 g. and measured 8.5 × 4 × 2 cm. The external surface was shaggy and greyish white studded with tiny white nodules. The epididymis appeared to be incorporated in the mass (Fig. 1). The mass was very hard and the cut surface was greyish scattered with white nodules, without any recognizable areas of normal testis or frank necrosis. The tunica vaginalis was not thickened (Fig. 2). After making multiple cuts, the entire mass was decalcified, before processing for microsections.

**MICROSCOPIC EXAMINATION** Examination of multiple microsections from different areas revealed mostly scattered pseudotubercles in different stages of...
FIG. 2. The longitudinal cut surface of the specimen in Fig. 1 shows epididymis and spermatic cord incorporated in testis. The tunica vaginalis peeled off on the left side is not thickened, and the testis shows a homogeneous greyish-tan surface stippled with whitish granules.

FIG. 3. Low-power photomicrograph showing pseudotubercles in different stages of involution and evolution with central schistosoma ova. The pseudotubercles are rimmed by concentric fibrosis, which also spans the intertubercular zones. Haematoxylin and eosin × 50.

FIG. 4. Low-power photomicrograph in another area showing diffusely scattered S. ova with intervening fibrosis and chronic granulation tissue. Haematoxylin and eosin × 70.
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involution and evolution (Fig. 3) consisting of a central ovum of Schistosoma surrounded by a zone of epithelioid cells about which there was a ring of plasma cells, eosinophils, and an occasional polymorphonuclear cell. The Schistosoma ovum possessed a terminal spine which was either intact or partially or completely distorted by calcification. The pseudotubercles were variously separated by thick and thin bands of fibroblasts or mature fibro-collagenous tissue. In other areas the ova were diffusely scattered in chronic inflammatory cell reaction with degrees of fibrosis (Fig. 4). Most of the ova were in different stages of degeneration and calcification as seen from the size, shape, chitinous coating, and embryonic contents (Fig. 5). In none of the sections seminiferous tubules or Leydig cells were identified.

COMMENT

This patient came from the northern region which is known to be endemic for schistosomiasis. He was infected both by S. mansoni and S. haematobium. In spite of this the presence of S. haematobium ova in the capillaries of the testis must be explained on the basis of extraportal maturation of worms in the spermatic venous circulation, a theory propounded earlier (Joshi, 1962) after studying and reporting the first case in the literature of bilateral total infarction of the testis due to schistosomiasis of the spermatic cord. A biopsy of the testicular mass should be done when possible before orchidectomy. The routine microscopic examination of the testis and spermatic cords removed in cases from known endemic areas might throw further light on the true incidence of spermatico-testicular schistosomiasis.

I am grateful to the director and staff of the Armed Forces Institute of Pathology, Washington D.C., U.S.A., for reviewing the case and for taking photomicrographs and to Mr. M. C. Thompson and Mr. Z. A. Rashid for the histological preparations.

REFERENCE