Cutaneous ectopic schistosomiasis

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Summary

Two cases of cutaneous schistosomiasis due to ectopic ova have recently been seen. Both patients presented with abdominal papular lesions, which were found on biopsy to contain schistosoma ova. To reach these abdominal sites mature worms probably migrate from the portal circulation to the paraumbilical veins, where they anastomose with veins of the caval system.

Introduction

Throughout wide areas of the world (fig 1) the three anthropophilic species of *Schistosoma* are responsible for much serious disease of the renal and gastrointestinal tracts, liver, lungs, central nervous system, and heart. The cutaneous manifestations are of three types: schistosomal dermatitis due to penetration of the skin by cercariae; urticaria and oedema, which are part of the "allergic" phenomena in invasion; and late cutaneous schistosomiasis due to the presence of ova in the dermis. Dermal ova are usually deposited in the genital area, although rarely this ectopic deposition occurs at more distant sites in the skin.

Case 1

A 20-year-old woman from Sierra Leone who had lived in Britain for seven months presented in the clinic complaining of an itchy rash on her trunk. This had first appeared three years earlier, since when new lesions had developed around the umbilicus and on the flanks. She had experienced intermittent lower abdominal pain ever since a single episode of haematuria five years earlier, for which she had received some tablets. There were no recent symptoms referable to the urinary or gastrointestinal tracts.

Examination showed a lichenified papular eruption with grouping, particularly around the umbilicus and sparsely on the flanks (fig 2). There were no other abnormal physical findings.

Skin biopsy of one of the lesions showed apparently viable ova of *Schistosoma haematobium*, with characteristic terminal spines and miracidia, in the dermis associated with collections of eosinophils (figs 3 and 4). In one section an ovum was apparently undergoing transepidermal elimination. Positive staining of the ova was seen with Ziehl-Neelsen and Gram stains. Calcified ova of *S. haematobium* were seen in rectal "snips" (scissors biopsy). Cystoscopy showed the characteristic "grains of sand" appearance in patches on the bladder wall; biopsy of these lesions was also positive. The schistosoma complement fixation test was positive to a titre of 1/80. Repeated examination of centrifuged urine for schistosoma eggs was unrewarding. Full blood count, erythrocyte sedimentation rate, liver function test results, urea and electrolyte levels, and serum iron concentration were normal. Serum IgE was raised at 875 µg/l. Other immunoglobulin levels were normal. Chest x-ray findings and an intravenous pyelogram were unremarkable. No calcification was seen on an abdominal x-ray picture.

The patient was treated with niridazole (Ambilhar) 500 mg three times a day, and prednisone 5 mg three times a day for one week with

FIG 1—Global distribution of anthropophilic *Schistosoma* species.

sequential reduction of the steroid dosage. In the course of treatment she experienced severe symptoms of malaise and headache. Eight weeks after treatment the lesions had become flattened and non-pruritic.

Case 2

A 33-year-old Saudi Arabian woman presented with a five-month history of mildly pruritic papular lesions on the anterior abdominal wall. She had experienced dysuria and vulval irritation during this period.

Two papular lesions on the upper abdominal wall were present on examination. Biopsy of these lesions showed the ova of *S. mansoni*. Viable *S. mansoni* ova were also seen on stool microscopy.

The schistosoma complement fixation test was positive to a titre of 1/10. Other investigations gave negative results.

During 10 days' treatment with niridazole 500 mg three times a day she developed headache and some lower abdominal pain, and on the eighth day of treatment she developed a mild maculopapular rash on the forearms and thighs.

FIG 2—Case 1. Periumbilical distribution of papules of cutaneous ectopic schistosomiasis.

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FIG 3—Case 1. Collections of schistosoma ova in the dermis with associated infiltrate of chronic inflammatory cells including eosinophils. (Haematoxylin and eosin $\times 59$.)

FIG 4—Case 1. Ova with terminal spines characteristic of *S haematobium* lying in dermis. (Haematoxylin and eosin $\times 117$.)

Discussion

Cutaneous lesions of schistosomiasis attributable to ectopic eggs are extremely rare, even in areas where schistosomiasis is endemic.¹⁻³ Clinical diagnosis may be difficult in the sporadic cases that present in Britain, but examining serial sections of a deep skin biopsy specimen of a nodule should allow a conclusive diagnosis to be made in every case. The schistosoma eggs lie in the dermis, often in groups associated with a scattered

inflammatory infiltrate containing a predominance of eosinophils. Identifying the type of schistosoma in skin sections depends on the position of the spine on the oval eggs, being terminal in *S haematobium* and lateral in *S mansoni*. Older lesions in which ova are disintegrating may be associated with necrosis and a granulomatous infiltrate.³

Manson⁴ states that ova of *S mansoni* are acid- and alcohol-fast, while those of *S haematobium* are not. In case 1 the ova of *S haematobium* were positive to Ziehl-Neelsen staining, which suggests that this method of differentiation is unreliable.

The life cycle of *Schistosoma* species is well established.⁴ In man, the cercariae, after penetrating the skin, reach systemic blood vessels via lymphatics. They mature into adult worms in portal sinusoids, and from there they reach the venous plexuses around the bladder and rectum, having migrated as pairs against the portal blood stream.

It has been suggested that where a less stable host-parasite relationship exists, such as areas where schistosomiasis is not endemic, the worms may stray from their usual pathway, causing ectopic cutaneous lesions.⁵⁻⁶ This variation may also be encouraged by inadequate treatment of an initial heavy infestation.³⁻⁷ The history in case 1 suggests that two years before the development of the skin lesions this patient may have received a course of treatment for schistosomiasis; if she did so it was obviously insufficient to eradicate the infestation.

Ectopic cutaneous lesions develop from ova that have been deposited by adult worms. Several routes have been suggested for the migration of these worms. Many workers suggest that the parasites may travel from pelvic veins via the vertebral plexus to spinal vessels, which would explain the disseminated and zosteriform distribution.^{2,8-8} Others have suggested that they pass through portosystemic anastomoses in the pelvic veins to the inferior vena cava and subsequently to various ectopic sites.⁷⁻⁹

The predominantly abdominal distribution has often been observed,¹⁰ and, in particular, the lesions have been observed in groups around the umbilicus, as in our patients.^{1-3,9-11} We suggest that the likely route in such cases is from the portal circulation via the left portal vein to the paraumbilical veins accompanying the ligamentum teres hepatis to the umbilicus, where they anastomose with the superficial and deep epigastric veins of the caval system.

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