Perigenital cutaneous schistosomiasis

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SUMMARY Perigenital cutaneous schistosomiasis was diagnosed in a patient who had no previous genitourinary or gastrointestinal symptoms suggesting schistosomiasis; his only symptom was a pruritic papular rash in the perineum. Late cutaneous schistosomiasis due to deposition of ova in the dermis is rare but can affect the genital and periumbilical areas. This report highlights the difficulty in diagnosing cutaneous schistosomiasis and the need for biopsy of itchy cutaneous lesions in patients from localities where the infection is endemic.

Introduction

Schistosomiasis is a widespread disease with variation in the predominant species of causative organism in different areas. In Nigeria *Schistosoma haematobium* and *Schistosoma mansoni* are responsible for most cases, causing serious vesical and gastrointestinal lesions respectively (Edington and Gilles, 1971).

The cutaneous manifestations of schistosomiasis result from: (a) penetration of the skin by the infective stage cercariae resulting in schistosomal dermatitis; (b) urticaria, oedema, fever, and pruritus which are hypersensitivity manifestations of invasion; and (c) deposition of ova in the dermis and subcutaneous tissues by adult worms which have passed into the superficial veins, usually becoming manifest several months after systemic invasion (MacDonald and Morrison, 1976). Although cutaneous ectopic schistosomiasis around the umbilicus has been described, deposition of ova in the genital area is very rare. We decided to report this case of perigenital cutaneous schistosomiasis, at the same time focussing attention on the genital manifestations of this tropical disease.

Case report

A 10-year-old Nigerian boy was referred to the Special Treatment Clinic for the investigation of a three-month history of perineal rash and pruritus,

which had persisted despite the application of various creams with and without antimicrobial agents. The onset of the eruption could not be attributed to any factor. He had no constitutional symptoms and no history of cough or episodes of haematuria and had had no gastrointestinal symptoms in recent months. He had never had sexual exposure and there was no history of urethral discharge or genital ulceration.

On examination numerous, discrete, firm, painless, papular eruptions were visible in the perineum (Fig. 1). No other physical sign was found and the patient was given a topical antipruritic agent while awaiting the result of our investigations.

INVESTIGATIONS

Skin biopsy showed both viable and calcified schistosome ova within the epidermis and in the dermis (Fig. 2). Tissue reaction around these varied from predominantly eosinophilic polymorphonuclear infiltrates to a granulomatous picture with few giant cells. The schistosome ova showed a terminal spine characteristic of *S. haematobium* (Fig. 3). Urine examination on four occasions showed no red or white blood cells. No schistosome ova were present and no pathogens were cultured.

The white cell count was within the normal range with a relative eosinophilia while the schistosome complement fixation test gave a positive result. Heaf test gave a grade I positive result and no mycobacterium was isolated from the biopsy material. Tissue culture for viruses also gave a negative result. Stool examination showed no schistosome ova. The Venereal Disease Research Laboratory test gave a negative result. Chest and abdominal radiographs and intravenous urogram were normal.

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Fig. 1 Discrete, firm, painless, papular eruptions in the perineum

Fig. 2 Clusters of schistosome ova in dermis associated with chronic inflammatory cells including eosinophils (Haematoxylin and eosin $\times 100$)

The patient was treated with two courses of niridazole (Ambilhar) 100 mg thrice daily for a week; a good response was achieved within six weeks of treatment with remission of the pruritus and disappearance of the eruption.

Discussion

Schistosomiasis is endemic in many areas of the world. In Ibadan, Nigeria, *S. haematobium* is widely endemic (Edington *et al.*, 1970). Man is infected by

Fig. 3 Ova with terminal spine characteristic of *S. haematobium* amidst inflammatory infiltrate in the dermis (Haematoxylin and eosin $\times 190$)

active penetration of the intact skin by cercariae which have emerged from the fresh-water snail intermediate host. The cercariae migrate to various tissues of the body and finally enter the systemic blood stream by way of the lymphatics; they mature into adult worms in the intrahepatic vessels. When sexually mature, and depending on the species, the adult worm then migrates to the mesenteric veins (*S. mansoni*, *Schistosoma japonicum*, *Schistosoma intercolatum*) or to veins of the pelvic plexuses (*S. haematobium*) where natural egg production begins. The period between infection—that is, cercarial penetration of the skin and recovery of eggs from the stool or urine of the host—is usually four to six weeks (Woodruff, 1974). Black (1945) found a correlation between the time of appearance of eggs in the urine and the onset of cutaneous lesions. The mechanism responsible for their selective organ habitation is not fully understood.

Renal or gastrointestinal symptoms, for which treatment is usually sought several months earlier, almost invariably antedate the onset of cutaneous schistosomiasis. Even in areas where the disease is endemic the cutaneous lesions due to deposited eggs are rarely seen (Findley and Whiting, 1971; MacDonald and Morrison, 1976). The lesions are small, deep-seated, and discrete with numerous deposits in the subcutaneous tissue during early

stages (Black, 1945). The early lesion therefore resembles subcutaneous tuberculosis and should be differentiated from it by biopsy of the lesion, which shows a granulomatous infiltration with a preponderance of eosinophils in which viable or calcified schistosome ova or both are found in groups—so called 'schistosomal tubercles'. Tissue eosinophilia is usually associated with peripheral blood eosinophilia, as shown in this case.

Cutaneous schistosomiasis is believed to arise from deposition of ova by adult female worms that have migrated to the superficial veins through anastomoses between the superficial veins and the pelvic venous plexuses. The production of a perigenital cutaneous lesion, as in this case, might have been due to adult worms that strayed from their normal path into the perineal vessels. Inadequate treatment of previous infection (Haber and Cowley, 1957) and altered host-parasite relationship may be contributory factors in the development of ectopic cutaneous lesions (Findley and Whiting, 1971).

We have no explanation for the absence of previous renal or gastrointestinal symptoms in our patient, but gastroenteritis is so common in our locality that it may have been overlooked. Identification of the species of schistosome in skin sections can be made by serial sections of the biopsy material for the position of the spine of the eggs,

which is lateral in *S. mansoni* and terminal in *S. haematobium*. In our case the position was terminal indicating that the species was *S. haematobium*.

Successful treatment of schistosomiasis with niridazole (Ambilhar) has been previously reported (Mahmoud and Warren, 1974; MacDonald and Morrison, 1976).

This case illustrates the difficulties in diagnosing genital lesions that may be encountered in tropical countries, since tropical diseases may affect the genital areas and thus need to be considered in the differential diagnosis of unusual lesions of the genital area.

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