

Perianal infection with group A streptococcus

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SUMMARY Anal fissure in childhood usually heals quickly after treatment with stool softeners and a local anaesthetic ointment; infection does not usually occur. Two cases are reported in which Lancefield group A β haemolytic streptococci were isolated from cultures from the perianal skin, which was erythematous and excoriated.

Case reports

CASE 1

An otherwise healthy 4 year old boy presented with a three week history of perianal irritation and discharge. At first a white discharge had been noticed on his pyjamas. On closer examination his mother had found small pustules surrounding the anus and a small, immobile white streak which she assumed to be a threadworm. The whole family had been treated with piperazine for a week, but the patient's symptoms had not improved. Miconazole and hydrocortisone cream had produced transient but incomplete relief of the irritation, and the discharge had continued. On examination the perianal region was erythematous and excoriated but no evidence of trauma was found. There was a sticky white discharge from the anus. He was treated with antiseptic baths and an emollient ointment, pending the results of a Sellotape test for threadworm ova (negative) and culture of a perianal swab, which yielded a profuse growth of group A β haemolytic streptococci. An oral course of phenoxymethylpenicillin resulted in prompt and complete recovery.

CASE 2

An otherwise healthy 5 year old girl presented with a three week history of perianal rash, which had initially appeared as tiny, yellow headed pustules. The main symptoms were itching, pain on defecation, and dysuria. There were no other urinary symptoms. Clotrimazole cream had been used for a week with no improvement. On examination there was erythema, excoriation, and fissuring surrounding both the anus and vulva but more obvious around the anus. A Sellotape test for threadworm ova was negative, but group A β haemolytic

streptococci were grown from culture of a perianal swab. She was asymptomatic after one week of oral treatment with phenoxymethylpenicillin.

Neither child was systemically unwell, but both had suffered considerable discomfort. Bacterial cultures from other sites were not available. There were no signs of sexual interference.

Discussion

The duration of symptoms before antibiotics were started together with the prompt response to penicillin indicate that the streptococcus played a true pathogenic part in these two cases.

Group A streptococci have been isolated from the perianal region in both symptomatic¹ and asymptomatic² adults. Infections of both the vagina³ and the perianal area⁴ have been reported in children, although streptococcal infection is rarely mentioned in standard textbooks. In the 10 cases of perianal cellulitis due to group A streptococci that were described by Amren *et al* the predominant features were intense erythema and swelling, the skin surface being intact in all but one case.⁴ Intense inflammation was also the principal feature in nine preadolescent girls with group A streptococcal vaginitis.³

Perianal infection with group A streptococcus in childhood causes considerable discomfort, which may be unduly prolonged without appropriate antibiotic treatment. It should be suspected when there is obvious inflammatory change, with or without fissuring or discharge. The cases reported here suggest that the presence of papules at an early stage may also be characteristic. A perianal swab should be obtained in all such cases.

It is important to remain vigilant for sexual abuse. When group A streptococci are not isolated from perianal eruptions organisms related to sexually transmitted diseases should be sought.

I thank Dr Peter Husband for permission to report these cases.

References

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Muscle carnitine deficiency presenting as familial fatal cardiomyopathy

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SUMMARY Three siblings presented with fatal cardiomyopathy confirmed by electron microscopy, and normal serum but low muscle carnitine concentrations. A fourth had similar signs but remained asymptomatic. He was treated with carnitine orally which increased the concentration in muscle, though it remained below normal. Electron microscopic features were unchanged.

Carnitine deficiency was first described by Engel and Angelini in 1973.¹ Carnitine is a carrier in the long chain fatty acid transport system that is essential for the transport of fatty acids across the inner mitochondrial membrane. Deficiency of carnitine affects mitochondrial oxidation of fatty acids



Fig 1 *Electron photomicrograph of muscle biopsy specimen from case 3 showing a myofibril with central nucleus* ($\times 16\ 500$).

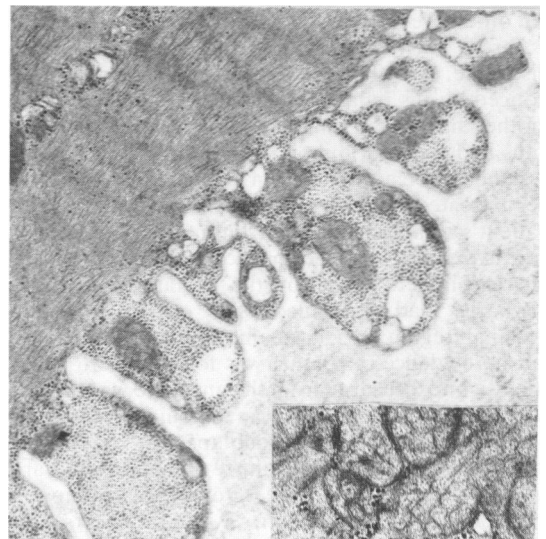


Fig 2 *Electron photomicrograph of muscle biopsy specimen from case 4 showing irregular folds of sarcolemma. Note presence of small lipid droplets under the sarcolemma and between the myofibrils* ($\times 22\ 100$). Inset: *cluster of mitochondria with tubular cristae* ($\times 32\ 500$).

and results in the accumulation of lipids in the cytoplasm.²

We describe a non-consanguineous Sephardic family with two healthy children and three children who presented with fatal cardiomyopathy probably due to muscle carnitine deficiency (fig 1). A sixth and youngest surviving sibling was asymptomatic at