SUMMARY AND CONCLUSIONS

Twelve cases subjected to upper femoral osteotomy for paralytic luxation of the hip are analysed. It is suggested that the cause of recurrence in a proportion of the younger cases is inadequacy of the osteotomy to ensure congruous reduction, particularly during weight-bearing, rather than persistence of muscle imbalance.

It is concluded that upper femoral osteotomy is at present the most satisfactory procedure for the correction of paralytic luxation, but that it must procure adequate varus to ensure complete congruity under all circumstances, as evidenced by a postoperative, weight-bearing radiograph.

In some younger patients with grossly shelving acetabula, it may be necessary to supplement the femoral osteotomy by a Salter pelvic osteotomy, but iliopsoas transfer is unlikely per se to reduce the recurrence rate and probably has little place in the management of the condition.

In patients in whom the luxation develops slowly and who present for treatment at a relatively later age, recurrence is less likely; and bone modelling seems to take the form of adaptation to the less than perfect relationships, rather than deterioration and relapse.

I wish to thank Dr J. W. Tate, Superintendent of Wynberg Orthopaedic and Convalescent Hospitals, and Prof. C. E. Lower Allen, of the Department of Orthopaedics, University of Cape Town, for permission to publish this paper, and Mr de Meneaud for the photographs.

REFERENCES


AMOEBIASIS OF THE ANTERIOR ABDOMINAL WALL*

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Amoebiasis is well known for its varied and often bizarre mode of presentation in sites other than the gastrointestinal tract. One of us (I.N.), in fact, recorded a case of amoebic granuloma of the buttock, as long ago as 1947.

This communication concerns a case of amoebiasis of the anterior abdominal wall in which the diagnosis could not be firmly established initially. The true nature of the condition only became apparent when the patient subsequently presented with a discharging sinus of the anterior abdominal wall.

CASE REPORT

J.S., a 22-year-old Bantu male resident in Alexandra Township, Johannesburg, presented at Edenvale Hospital on 25 September 1968, complaining of a painful epigastric swelling of some 2 weeks’ duration.

There had been no alteration in his bowel habit, no dysentery, nausea or vomiting, and there were no factors which aggravated or relieved the pain. He had not left the local environs over the past year. This was the first occasion on which he had sought medical advice for an abdominal complaint.

On examination the patient was found to be well nourished. He was afebrile with a normal blood pressure and pulse rate. Systematic examination was essentially normal, the only abnormal feature being that of a firm, tender mass situated in the midline 3 in. above the umbilicus. The mass appeared to lie in the anterior abdominal wall. There was no detectable difference in temperature between the mass and the surrounding structures and no skin oedema was present. No impulse was detected on coughing.

Rectal examination, stool and urine analysis were normal.

At this stage it was impossible to establish a firm diagnosis and the patient was submitted to laparotomy, the tentative diagnosis being that of an incarcerated ventral hernia or a desmoid tumour.

*Date received: 10 February 1969.

Fig. 1. Site and healing of sinus 4 days after commencement of treatment.
At operation an extraperitoneal mass was found lying between the rectus muscles and peritoneum. The mass was hard in consistency, firmly attached to the surrounding structures and macroscopically was thought to be a sarcoma.

The peritoneum was opened but nothing abnormal could be found in the liver, stomach, transverse colon and small bowel. The mesenteric lymph nodes were not enlarged. The mass was excised and submitted for histological examination.

Biopsy report (Dr E. Henschel, pathologist, SAIMR, Johannesburg). The specimen consisted of skeletal muscle, fascia and non-specifically inflamed fibro-fatty tissue. The latter possibly represented peritoneal tissue attached to the fascia. The inflammatory response was chronic in nature and no evidence of malignant neoplasia was present. The features suggested a non-specific process involving the anterior abdominal wall.  

The patient's postoperative course was uneventful and he was discharged on the tenth postoperative day, the wound being fully healed.

Subsequent Course

He was readmitted on 27 October 1968, approximately one month after the operation, as the wound had broken down. Reddish-yellow purulent matter was being discharged from the wound, and the surrounding skin was excoriated. The wound was extremely tender.

Specimens of the discharge, together with a biopsy specimen of the wall of the ulcer, were submitted for laboratory investigation. The pus swab disclosed the presence of vegetative forms of Entamoeba histolytica while the biopsy revealed necrotic debris, inflammatory cells and amoebae.

He was placed on the following regimen: oxytetracycline, 500 mg. 6-hourly orally for 5 days; metronidazole, 200 mg. thrice daily; and chloroquin sulphate, 800 mg. orally for 48 hours, followed by 400 mg. daily in divided doses for 19 days. Following the commencement of treatment the discharge rapidly decreased in amount, the pain and tenderness disappeared, and the wound was fully healed 10 days later.

DISCUSSION

It is now well documented that amoebiasis may present in many forms other than the classical amoebic liver abscess or amoebic dysentery. There are numerous reports of perianal and perirectal skin involvement, and also of amoebiasis involving the skin secondary to the drainage of amoebic liver abscesses, adjacent to colostomies and following appendicectomies.

Primary involvement of healthy normal skin by amoebae rarely, if ever, occurs and we would agree with previous authors that the majority of cases of amoebic skin infection are secondary to damage and devitalization of the skin.

We are aware of cases of amoebiasis of the anterior abdominal wall unassociated with any underlying overt visceral amoebic infection. One of these cases is highly instructive in that the amoebae could not be cultured from the pus alone and the diagnosis could only be conclusively proved on biopsy of the ulcer wall.

We are in full agreement with Osborn' who described a very similar case to the one presented above. He is of the opinion that these cases represent the spread of amoebic emboli via the portal vein from the gut by way of the para-umbilical veins which lie in the free border of the falciform ligament and anastomose with the superior and inferior epigastric veins of the anterior abdominal wall.

In support of this viewpoint we should like to draw attention to the close similarity between our case and the first case described by Osborn.

The difference of duration of symptoms, viz. 2 weeks compared with 2 months, probably accounts for the fact that our case presented only with a painful mass and not an abscess on the point of rupture, as did Osborn's patient.

Both patients denied any dysentery. It is interesting to note that amoebae apparently passed from bowel to anterior abdominal wall without leaving any signs or symptoms in their wake. There were no macroscopic changes present at the time of operation in the liver or large or small bowel. The diagnosis of this condition depends on the finding of amoebae in pus swabs or in their biopsy specimens.

As has been repeatedly stressed by previous authors, the pus swabs must be taken directly from patient to laboratory because the time lag and subsequent drop in temperature will result in a loss of motility and will make identification more difficult. In our case the pus swabs were immersed in warm saline and rushed to the laboratory. These precautions still do not guarantee a 100% success rate and it is advisable that a generous biopsy specimen be taken from the ulcer wall and submitted for histological examination. This, however, is also not infallible, as was demonstrated in the initial biopsy specimen, but it does ensure a higher success rate. Repeated pus swabs and biopsy specimens may be necessary to prove the diagnosis.

SUMMARY

A case of amoebiasis of the anterior abdominal wall is presented. The origin of such lesions is briefly discussed. A plea is made for repeated pus swabs and histological examinations in the diagnosis of this condition.

We should like to thank Dr J. Meyer, Senior Physician and Acting Superintendent of Edenvale Hospital, who was responsible for the medical management of this patient, for permission to publish this case.

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