damage in our patient, as speculated by Vachon *et al.*,⁵ because liver function was normal or borderline, and HBV antigenaemia persisted even after the anaemia improved and the DAGT had become negative. The role of HBV in the pathogenesis of AIHA has thus been unclear.

From many recent studies of type B hepatitis⁶ it is suggested that a defect of cell-mediated immunity may yield the carrier state of HBV. This hypothesis is consistent with the fact that newborn babies may be infected from their HBV-carrier mother, resulting in the persistent carrier state.⁷ Likewise, the role of cellular immunity in the pathogenesis of AIHA has become clearer. Krüger *et al.*⁸ suggested that an imbalance between reduced T-cells and increased but uncontrolled humoral immune response towards red cell antigens may lead to immunological haemolysis.

Taking these suggestions into consideration, it is possible that in our patient AIHA was induced by an upper respiratory infection or some other agent, in the presence of an immunological defect which permitted the HBV carrier state. Further study will be necessary to confirm the relationship between persistent HBV antigenaemia and AIHA. Zuelzer *et al.*² have attached importance to cytomegalovirus as a virus with which occult infection was observed in children with AIHA. It appears that attention should also be paid to HBV. None of the immuno-

logical tests in our patient showed any abnormality, nor was there any evidence of other autoimmune disease.

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Perineal amoebiasis

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SUMMARY A 14-month-old girl with invasive perineal amoebiasis is reported. There was destruction of the anus, the rectovaginal septum, the pelvic floor, and the perineum. The rapid progress and destructive potential of perineal amoebiasis is noted and the benefits of diagnostic awareness emphasised. Treatment with metronidazole will prevent further damage.

Amoebiasis may spread to affect the perineum or the female genitalia.¹⁻⁶ The lesions are often superficial and of long duration⁴ but in children may be rapidly invasive.^{3,5} The infection can produce extensive ulceration around a colostomy or ileostomy.⁷ An undermined, spreading ulcer may start in drainage

sites or surgical wounds after surgery to a patient suffering from amoebiasis.^{2,5}

The following case is reported to illustrate the destructive potential of the parasite in the perineum of a child.

Case report

The patient, a 14-month-old girl of mixed descent, was referred from a country hospital where she had been admitted with a 2-week history of diarrhoea. While in hospital she had developed a perianal abscess which had burst.

Examination showed a seriously ill, toxic, pyrexial, anaemic child. Further abnormal findings were confined to the perineum where there was extensive

undermined ulceration surrounding and destroying the anus. The posterior vulva and vaginal introitus were extensively ulcerated with only a small bridge of skin remaining in the perineum (Fig. 1). Digital examination showed a craggy cavity where the anus, rectum, and vagina had been. The rectovaginal septum had been destroyed. Induration extended widely into the tissues on each side.

The haemoglobin was 5.3 g/dl, and the white blood count $20.8 \times 10^9/l$, with neutrophils 61%, monocytes 1%, lymphocytes 35%, eosinophils 2%. Serum electrolytes were normal. Cultures showed *Escherichia coli* and *Pseudomonas aeruginosa*. Examination of warm stools was negative for *Entamoeba histolytica* but the latex agglutination test was positive and biopsies of the ulcer showed numerous *E. histolytica* in granulation tissue.

Treatment was started immediately with metronidazole (Flagyl) 50 mg/kg per day and intravenous fluids, blood, and antibiotics. A left iliac colostomy was performed 10 days later.

The response to treatment was immediate. Constitutional signs resolved within 3–4 days and the perineum healed rapidly. Barium studies 2 months later (Fig. 2) showed destruction of the rectovaginal septum so that rectum and vagina formed a common cavity, separated at the lower end by a small bridge of tissue. There was a stricture of the upper rectum where it entered this cavity but the colon above this was normal.

Repair was performed 4 months after admission. The perineal bridge was divided, the rectal stricture

Fig. 1 Extensive perianal and vulval ulceration on admission.

Fig. 2 Barium studies showing the rectal stricture and rectovaginal cavity.

dilated, and the vagina and rectum reconstructed leaving a Silastic prosthesis in the rectum to maintain patency through the stricture. Only a few muscle fibres were found in the bridge of tissue. The remaining anal muscles had been destroyed. The perineum was reconstituted by mobilising tissues on each side.

The repair healed well. The prosthesis was removed 2 months later and the colostomy closed soon afterwards. The upper stricture has not recurred but 4 years later there is still extensive fibrosis around the anus with a stricture which has not responded to dilatations or minor plastic procedures. The 5-year-old child is incontinent. Social conditions have so far not permitted a permanent colostomy which will be necessary.

Discussion

E. histolytica may be a harmless commensal in the bowel or it may become invasive causing ulceration with amoebic dysentery. Deeper invasion with perforation and peritonitis occurs in 3% of adults and 9% of children with acute amoebic dysentery. A localised chronic granulomatous response will produce an amoeboma.⁸

Occasionally the subcutaneous tissues around the anus, an ostomy, or a wound are invaded.^{2-3 7} The

process is sometimes a chronic one but rapidly spreading necrotising infection may occur with destruction of skin, fat, and muscle. The response to metronidazole is striking and early diagnosis with prompt treatment will prevent further destruction. The diagnosis should be considered if spreading ulceration occurs in the perineum or abdominal wall. Stool examination, serology, scrapings from the ulcer, and biopsy are of value in confirming the diagnosis of amoebiasis but, if this seems the likely cause, treatment in cases with rapidly spreading ulceration should begin immediately without waiting for the results of these tests.

Cutaneous amoebiasis may occasionally occur in a patient without previous intestinal infection by external inoculation of a wound.^{3 5} Penile and vaginal amoebiasis occurring in adults is thought to be venereal in origin.^{1 3 5}

The vulva is particularly vulnerable to infection in the small child still wearing a nappy as this holds infected stools in close contact with the perineum.^{2-3 5-6} Pronounced destruction can occur. In the case reported here the vulval ulceration and perianal ulceration were separated by a bridge of skin and there was extensive induration in the ischiorectal fossa and pelvis with destruction of the anal muscles, pelvic floor, and rectovaginal septum.

The history that the initial presentation had been as a perianal abscess suggests that the invasive process started more deeply within the anus or rectum.

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A normal paediatric amylase range

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SUMMARY A normal paediatric range of plasma α -amylase activity was determined using the Phadebas blue starch method. The range for children over one year was 98-405 IU/l. Plasma amylase activity increased throughout infancy. Mature levels of activity were observed in some children by age 2 months and in most of them by 9 months.

The Phadebas amylase test (Pharmacia) is a simple and accurate method for determining plasma or serum α -amylase activity.¹⁻² Although the original report on this method gave a normal range for individuals under 20 years, a satisfactory normal range for children has not been established. This report describes such a range.

Plasma was obtained from heparinised blood samples taken from 116 children; 54 from children

immediately after induction of general anaesthesia, 47 from children having a venepuncture for other investigations, and 15 from cord blood of term normal neonates. Children with renal, hepatic, or gastrointestinal disease or those with a history of fits or recent infection were excluded. 53 children were under one year, the eldest was 15 years.

Plasma total α -amylase activity was measured by the Phadebas blue starch method according to the manufacturer's instructions. The calibration was adjusted to enable the determination of activities below 30 IU/l.

Statistical comparisons were made using the Wilcoxon rank sum test.

Results

Plasma α -amylase activity was lowest in the neonates