MEDICAL MEMORANDA

Steatorrhoea in Henoch's Syndrome

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The bowel lesions of the Henoch-Schönlein syndrome have been described as extensive submucous haemorrhages, often in the terminal ileum, intussusception being a common feature (Osler, 1914; Gairdner, 1948). Bloody diarrhoea and abdominal pain are the usual presenting features, and the characteristic rash may precede or coincide with the abdominal symptoms. Protein loss from the bowel in patients with hypoalbuminaemia and without significant proteinuria has been described, (Jones *et al.*, 1966; Cream *et al.*, 1970), but steatorrhoea does not appear to have been noted. In the present case a patient with Henoch's syndrome had clinical evidence of steatorrhoea and a malabsorption state for several weeks, as well as nephritis and typical skin lesions, with eventual complete recovery.

Case Report

A 43-year-old housewife was admitted to hospital on 25 November 1968 with a two-day history of nausea and colicky central abdominal pain accompanied by vomiting of green fluid containing streaks of blood. There was no notable past history except for some acid regurgitation, heartburn, and infrequent attacks of migraine. On examination she was well and afebrile. Her blood pressure was 110/80 mm Hg. There were no abnormal findings in the abdomen, but rectal examination showed fresh blood. The urine contained a trace of blood and protein. The stools appeared fatty macroscopically and contained fresh blood. The haemoglobin level was 15 g/100 ml, W.B.C. 13,000/mm³, and E.S.R. 6 mm in one hour. Serum electrolytes were normal, blood urea was raised (65 mg/100 ml), serum calcium reduced (6.8 mg/100 ml), and serum total protein reduced (4 g/100 ml). Chest radiograph and barium-meal examination were normal.

One week after admission she developed a purpuric rash over the lower back, upper thighs, and arms. Some of the lesions were papular, and a skin biopsy on 3 December showed "marked vasculitis" in the deep corium with a large perivascular inflammatory reaction. Two weeks later the haemoglobin was 10.5 g/100 ml and W.B.C. 11,000/mm³, with normal differential count and platelet count. Xylose excretion was reduced at 2 g in five hours after a 25-g dose. Three-day faecal fat estimations gave 13 g/24 hr and 16 g/24 hr. A jejunal biopsy specimen obtained with a Watson capsule showed normal histological appearances. The urine now contained protein + + +, a few red cells, and granular casts. The blood urea and electrolyte levels were now normal, but endogenous creatinine clearance was reduced (53 ml/min.). A glucose tolerance test gave a flat curve. Proteinuria

varied from 0.1 to 0.5 g/2 4hr. Barium-enema and barium-meal examinations were normal. A follow-through examination on 18 December showed persistent spasm in the caecum and terminal ileum, but a repeat examination on 22 January 1969 was normal. The serum folate level was low at 1.7 mg/ml. An intravenous pyelogram and an electrocardiogram were both normal.

Five weeks after admission the stools were macroscopically normal and faecal fat excretion was 2.8 g/24 hr. Xylose excretion was 5.5 g after a 25-g dose. The serum total protein was 6 g/100 ml and the serum calcium, phosphate, and electrolytes were normal. Renal biopsy was performed on 3 January because of persistent proteinuria and showed vascular thickening but no evidence of periarteritis. There was pronounced crescent formation in the glomeruli and protein casts in the tubules, the appearances being consistent with a patchy proliferative nephritis.

The patient remained well during her stay in hospital except for three episodes of colicky abdominal pain, vomiting, and bloody diarrhoea lasting only a few hours. The rash on her back and limbs faded, but a few new lesions appeared over three to four weeks. She was treated with iron by mouth and analgesics as required. When discharged on 15 February she was symptom-free and had a fading purpuric rash mainly on the legs. She was seen periodically thereafter and remained very well, with no recurrence of steatorrhoea or abdominal pain, but continued to have mild proteinuria of 0.1-0.5 g/24 hr.

Comment

This patient had clinical steatorrhoea for about four weeks but relatively mild abdominal symptoms, and the diagnosis of Henoch's syndrome was made only after the appearance of the characteristic rash. There was evidence of nephritis but only slight proteinuria, and the low serum protein at the time of admission was presumably the result of loss of protein in the bowel as described by Jones *et al.* (1966). Gastrointestinal involvement is a common feature of Henoch's syndrome, and 34 out of 77 patients reported on by Cream *et al.* had evidence of bowel lesions. The probable cause of the present patient's steatorrhoea was either vasculitis of the bowel, as described by Gairdner (1948), or interruption of bile salt absorption in the terminal ileum. Some support for the latter mechanism is provided by the initial finding on barium follow-through examination of spasm in the caecum and terminal ileum, presumably caused by local submucosal haemorrhage. Transient steatorrhoea may possibly be a common feature of Henoch's syndrome, but in a series of five other adult patients studied there was no increase in the faecal fat excretion.

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References

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