The site of protein loss in Schönlein-Henoch purpura

DEREK G. WALLER*
B.Sc., M.B. B.S., M.R.C.P.

KATHARINE L. DALZIEL
M.B. B.S.

Professorial Medical Unit, Southampton General Hospital, Tremona Road, Southampton

Summary
Schönlein-Henoch purpura may be complicated by hypoproteinaemia, which in most patients is due to the development of nephrotic syndrome. However, in some, proteinuria is insignificant and enteric protein loss has been suggested as the cause. A case with supportive evidence for this is reported.

Case report
A previously healthy 62-year-old man developed a purpuric rash over the buttocks, extending down the anterior surface of both legs and then the backs of the hands and wrists. Two days later he suffered joint pains, initially in both ankles and elbows, wrists and knees with an effusion in the right knee. On the fourth day of the illness he had central colicky abdominal pain with vomiting, abdominal tenderness, distension and increased bowel sounds. He also had oedema of the dorsum of both hands and feet. The rash became necrotic in some areas with bulla formation in others. There was no recent history of upper respiratory tract infection or drug ingestion.

Blood and platelet counts were normal; there was no evidence of a clotting defect. ESR was 19 mm/hr (Westergren). ANF and rheumatoid factor were negative and complement levels and ASO titres were normal. Blood, urine and knee aspiration cultures were sterile. Urea was slightly raised at 7-2 mmol/l, but creatinine and electrolytes were normal, as were the liver function tests and serum amylase. Ward urine analysis showed proteinuria and haematuria and the 24-hr urine protein loss was 0-2 gm; serum albumin was 38-5 g/l.

The joint symptoms fluctuated in severity and fresh crops of purpura appeared during the next

* Present address: Professorial Medical Unit, St. Mary’s Hospital, Portsmouth

FIG. 1. The relationship between serum albumin and urinary protein loss.
few days. Severe abdominal pain and vomiting continued to be major problems. A barium follow-through meal showed gross mucosal oedema affecting the duodenum, the jejunum and distal ileum with a suspicion of a transient jejunal intussusception. Intravenous hydrocortisone was started and the abdominal symptoms improved within 48 hr, finally settling 8 days later.

During the first 4 weeks of the illness the serum albumin fell progressively to a minimum of 25.2 g/l (Fig. 1) and the patient developed pitting oedema to the waist. At no time during this period did repeated urine collection show proteinuria in excess of 4.4 g/24 hr. Four weeks after the onset a 7-day stool collection following i.v. 51Cr-chromic chloride showed enteric protein loss equivalent to a daily plasma clearance of 177 ml (normal, <20 ml/day).

Discussion
The case clearly indicates that hypoproteinaemia can occur in the absence of nephrotic syndrome and in this patient it occurred in the absence of loss of protein in the urine amounting to >5 g/24 hr and accepted as the minimal level for nephrotic syndrome to manifest itself. In common with 2 previously reported cases (Jones, Creamer and Gimlette, 1966), this patient had severe abdominal symptoms. Assuming that protein loss from the gut is unselective and that the appearance of 51Cr in the stool represents bulk protein loss from the plasma (Barth et al., 1964), it was equivalent to a daily loss of 9-1 g/day in this patient at a time when abdominal symptoms were settling.

There are other reports of unexplained hypoproteinaemia in Schönlein-Henoch purpura (Cream, Crumpel and Peachey, 1970; Taylor, Amin and Mruthyanjaya, 1971; Bryn et al., 1975). Although albumin synthesis was not studied, we believe this case emphasizes the importance of enteric protein loss as a cause of hypoalbuminaemia, particularly in patients with severe abdominal symptoms.

Acknowledgment
We wish to thank Professor C. F. George for his advice and permission to report this case.

References