theless, seven were unhappy about the appearance of the scars and three found that they had some limitation of arm movement.

A recent paper by Jemec⁴ reported his technique of subcutaneous curettage of the axillae. The marked area of skin is undermined with a pair of scissors through one or, if necessary, two short incisions. A sharp gynaecological curette is used to remove all subcutaneous fat in the area demarcated by the starch-iodine test. Curetting is continued until small petechiae appear on the surface of the skin and the scraping takes about 10 minutes. The wound is drained, the small incision sutured, and the stitches removed after about 10 days. Reporting on 20 patients submitted to this procedure, evaluated six to nine months later, Jemec found that 12 were entirely satisfied, four partly satisfied, three dissatisfied, and one was lost to follow-up. None of the patients complained of troublesome scars. A similar number of patients submitted to the excision operation achieved roughly similar results, but four of these were troubled by the axillary scarring.

Because of the encouraging nature of this report, we decided to carry out a trial of the procedure in patients, to whom the problem was first carefully explained. Twelve patients (two males and 10 females) were operated on by this technique between January and April 1976. Review in February 1977 shows that two patients were completely satisfied with the result. One has a good result on one side but no effect on the other, and one says she is 50% improved. One patient completely failed to respond to the operation and the remaining seven relapsed completely from one to five months after curettage. Five of these have already been re-operated upon by the axillary skin excision technique and one is on the waiting list for operation.

We have therefore abandoned this subcutaneous curettage and we have returned to the well-tried axillary excision operation.

¹ Ellis, H, *Practitioner*, 1976, **217**, 416.

² Skoog, T, and Thyresson, N, *Acta Chirurgica Scandinavica*, 1962, **124**, 531.

³ Hurley, H J, and Shelley, W B, *Journal of the American Medical Association*, 1963, **196**, 109.

⁴ Jemec, B, *Scandinavian Journal of Plastic and Reconstructive Surgery*, 1975, **9**, 44.

(Accepted 29 March 1977)

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Amyloidosis complicating psoriatic arthritis

We report here a patient with deforming psoriatic arthritis and steatorrhoea. Biopsy of a skin nodule, jejunum, and rectum confirmed the diagnosis of amyloidosis.

Case report

A 50-year-old man was referred with a five-month history of diarrhoea. He was passing 1-4 loose motions a day which flushed away from the lavatory pan with difficulty. His appetite was normal and he denied weight loss or abdominal pain. For 14 years he had been known to have psoriatic arthritis affecting the hands, elbows, ankles, knees, sacroiliac joints, and cervical spine. Skin lesions had never been observed but he had the typical nail changes of psoriasis. He had taken prednisolone for 12 years, but the arthritis had become progressively worse and led to his retirement four years previously. He appeared Cushingoid, with a blood pressure of 130/80 Hg. His nails were thickened, yellow, brittle, and pitted. Two small nodules

were present on the skin of the palm of the left hand. Asymmetrical, generalised deforming arthritis was present. His liver and spleen were not palpable. The results of physical examination were otherwise normal. His haemoglobin was 11.2 g/dl; ESR 87 mm/h; total serum proteins 76 g/l, serum albumin 33 g/l, serum globulin 43 g/l. Electrophoresis showed an increase in alpha-2 globulin and a diffuse increase in gammaglobulin. Immunoglobulins: IgG 29.3 g/l; IgA 3.55 g/l; IgM 1.9 g/l. Twenty-four hour collection of urine contained 1.06 g of protein, but Bence-Jones protein was not detected. Serum rheumatoid factor, antinuclear factor, and anti-DNA antibodies were absent. HLA type 3, 14, W18, W19. Serum urate concentration was normal. Serum urea 11.6 mmol/l, serum creatinine 0.18 mmol/l. Creatinine clearance 40 ml/min. Repeated stool cultures were negative. Faecal fat excretion was 156 mmol over 3 days (normal up to 60 mmol). Bromsulphthalein retention was 30% at 45 minutes. D-xylose absorption test result: 0.24 g of 5-g dose excreted in five hours. Serum amylase and lipase concentrations and secretin-pancreozymin test of pancreatic function were normal, as were the results of liver and pancreatic scans.

Skeletal survey showed generalised loss of bone density and sacroiliitis. Appreciable erosive changes were present in the phalanges, metatarsals, and around the wrists and knees. The results of both barium meal and barium enema examinations were essentially normal. Small intestinal biopsy showed a normal villous pattern in the jejunum. All other morphological indices also appeared normal, but staining with crystal violet disclosed a positively reacting substance in the walls of many of the small blood vessels in the submucosa. This substance also stained positively with congo red and was then birefringent when viewed by polarised light. Similar findings were noted in the submucosal vessels of an otherwise morphologically normal rectal biopsy and in the small dermal vessels of a biopsy specimen from one of the nodules on the left palm.

Discussion

This patient had deforming psoriatic arthritis with typical psoriatic nail changes, which may be the only manifestation of psoriasis.¹ The skin is said to be rarely implicated in secondary amyloidosis but in this patient examination of a biopsy specimen of a skin nodule disclosed amyloid material in the dermis. Amyloidosis complicating psoriatic arthritis is rare. Lambert *et al*² reported two cases of amyloidosis in 43 children with psoriatic arthritis, but only one case in nearly 300 adult patients. In a review of psoriatic arthritis³ there were three cases of uraemia due to secondary amyloidosis among 32 deaths but none in a recently published follow-up study of 227 cases of psoriatic arthritis.⁴ We are aware of only one other reported case in British publications⁵ of amyloidosis in psoriatic arthritis presenting with diarrhoea, but this patient did not have steatorrhoea.

We thank Professor H T Howat for permission to report this case.

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⁴ Roberts, M E T, *et al*, *Annals of Rheumatic Diseases*, 1976, **35**, 206.

⁵ Ferguson, A, and Wilson Downie, W, *Annals of Rheumatic Diseases*, 1968, **27**, 245.

(Accepted 31 March 1977)

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