The result of the Paul-Bunnell test was negative, as were tests on sputum and urine and the cerebrospinal fluid. Blood culture grew no organisms, the chest x-ray film was normal, while the throat swab grew β -haemolytic streptococci group F. Two days later his condition deteriorated, with temperature 41°C, tachycardia 130 beats/min, and triple rhythm. Electrocardiogram showed S-T elevation in anterior chest leads. Haemoglobin 9.9 g/dl; leucocytes $3.0 \times 10^9/l$ (3000/mm³), neutrophils 42%, lymphocytes 39%, monocytes 19%; platelets $47 \times 10^9/l$; ESR 110 in 1 h; aspartate aminotransferase 208 IU (normal 0-40 IU), creatine phosphokinase 67 IU (normal 0-50 IU), lactate dehydrogenase 2320 IU (normal 200-560 IU), and gamma-glutamyltranspeptidase 46 IU (normal 0-45 IU). Tests for Epstein-Barr (EB) virus and cytomegalovirus antibodies were negative. A bone marrow film showed moderate cellularity but no significant abnormality. The histology revealed histiocytic granulomas with poorly formed giant cells and some foci of necrosis (see figure). Many of the granulomas surrounded circular spaces which contained fat.

Histiocytic granulomas. (Haematoxylin and eosin $\times 60$.)

Fever continued without remission, and he was given minocycline, 200 mg daily, for suspected brucellosis without effect. After six days this was changed to co-trimoxazole. Nevertheless, brucella agglutinations, complement fixation, and antiglobulin tests were negative. His weight had fallen 17 kg, and this continued, so that antituberculous treatment was started because the bone marrow histology was thought to suggest tuberculosis. At the same time information was received that the patient's daughter had glandular fever. So Paul-Bunnell, differential absorption, and EB-virus antibodies tests were all repeated in the patient and were found to have become strongly positive. Sheep erythrocytes agglutination titre 1/512, after absorption with guinea-pig kidney titre 1/512, after ox cells 1/14. EB-virus antibody titre IgG 1/128, IgM positive. At that time the haemoglobin was 9.6 g/dl; leucocytes $2.9 \times 10^9/l$ (2900/mm³), neutrophils 51%, lymphocytes 35%, monocytes 11%, basophils 2%, eosinophils 1%, platelets normal.

Anti-tuberculous treatment was stopped, and 24 days after admission prednisolone, 60 mg daily, was begun, the dose later being steadily reduced. One week later haemoglobin was 11 g/dl, leucocytes and platelets normal. The abnormal enzyme values and electrocardiogram returned to normal, and prednisolone was stopped after nine weeks. One year later he remained in good health. The results of tests on the bone marrow, blood count, and serum enzymes and electrocardiogram were all normal; the EB-virus antibody titre was IgG 1/128, IgM negative.

Comment

The granulomas of brucellosis, sarcoidosis, and tuberculosis are similar to those of infectious mononucleosis, though the lesions of the first three are larger, more nodular, and more circumscribed than those of infectious mononucleosis—which are smaller and usually lack giant cells.¹ Pancytopenia only rarely complicates infectious mononucleosis,² and a report which illustrated its association with a lack of clinical manifestations has postulated that there is also a

focal granulomatous process in the marrow rather than a simple hyperplasia.³

Bone marrow examination is rarely needed in infectious mononucleosis. The main recorded reasons² for it are anaemia, or low platelet or granulocyte counts, usually associated with a definite generalised hyperplasia of erythroid, myeloid, megakaryocytic, and reticulum cell elements. Granulomas have been found in about one-third of these cases.

Serious suppression of marrow function is also rare, though aplastic anaemia has been reported.⁴ Granulomas usually arise in response to an antigenic stimulus.⁵ As there are many antibodies in infectious mononucleosis, granulomas may be more common than suspected,² although often asymptomatic. This patient, who lacked any of the normal clinical manifestations of infectious mononucleosis, showed a significant rise in heterophil and EB-virus antibodies during his illness. He not only had a myocarditis and pancytopenia, but illustrated another variation in the type of granulomas that are found. Unlike described lesions, these showed histiocytic cells with occasional giant cells surrounding a central circular area of fat.

I thank Dr J W Todd for permission to report on a patient under his care, and Dr C C Thomas, Dr C E Watson, and Dr R W Ainsworth for their help and advice. Virological investigations were undertaken by Professor J E Banatvala.

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Axillary hyperhidrosis: failure of subcutaneous curettage

Axillary hyperhidrosis (excessive perspiration) is a condition which is being encountered more and more frequently in general practice, dermatological clinics, and surgical departments. This is probably due to the stricter hygienic and social conditions of modern life and also to the increasing awareness of patients that effective treatment is now available.

The vast majority of patients have primary or idiopathic hyperhidrosis.¹ Secondary hyperhidrosis due to hyperthyroidism, obesity, anxiety states, the menopause, or disseminated malignant disease, and paroxysmal sweating due to pheochromocytoma are usually easy enough to differentiate because of their obvious accompanying manifestations. The primary type affects males and females equally, but girls and women are far more likely to complain of the axillary sweating.

Surgical treatment

Axillary sweating originates mostly from the eccrine glands located in a comparatively small area which roughly corresponds to the hair-bearing zone of the axilla. This can be mapped out easily by the application of a starch-iodine powder, which turns blue in contact with moisture. Surgical treatment of this condition comprises ablation of the sweat glands from this region. Skoog and Thyresson² first reported a technique of excision of these sweat glands in 1962. Four skin flaps are raised and the glands are snipped away from the underlying surface of the flaps by fine scissors. Hurley and Shelley³ reported good results after excision of the involved ellipse of axillary skin with primary wound closure. The results are satisfactory but undoubtedly a rather wide and ugly scar may result in the axilla, particularly in the skin excision technique. A follow-up of our first 50 patients treated by axillary skin excision (7 male and 43 female), reviewed after a year, showed that 47 were pleased with the results. Only three noted no improvement. Never-

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.

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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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