SHORT REPORTS

Late-onset atopic eczema and multiple food allergies after infectious mononucleosis

Atopy is thought to be hereditary, though the mode of inheritance has not been completely established. It has been suggested[1] that heredity is not the only important factor in the pathogenesis of the condition and that precipitating factors that modify immune regulation such as viral infections may also play a part. The following case may be an example of this.

Case report

A 22-year-old woman presented to her general practitioner in October 1976 with malaise and fever. Infectious mononucleosis was diagnosed and was confirmed by examination of a blood film and a positive Monospot test result. She had no history of skin problems or manifestations of atopy (including food allergy), and there was no family history of atopy.

Four weeks after the illness began the patient developed an erythematous itchy rash on her face, which spread to the cubital and popliteal fossae, presenting the typical appearance of atopic eczema. Six months later, after eating an egg, she developed angio-oedema with swelling of the mouth and face, which lasted several hours. She was advised to avoid eggs and referred to hospital. Skin prick tests with Bencard allergens elicited no positive reactions to inhalants (grass, pollens, cat and dog fur, house dust, and house-dust mite) or foods (milk, egg, cheese, fish, shellfish, wheat, mixed vegetable, mixed meats, and mixed fruits). Total serum IgE concentration (double antibody method[2]) was raised at 2150 U/ml, but radioallergosorbent tests (RAST; Pharmacia, Uppsala, Sweden) to three inhalants and 10 foods (egg, milk, fish, wheat, peanut, hazelnut, Brazil nut, almond, crab, and shrimp) elicited a positive result only to cat fur (table). There were no other findings of note apart from flexural eczema.

The eczema continued but she had no further angio-oedema until October 1979, when she had an episode after drinking cows' milk. After this her eczema worsened and she was advised to avoid cows' milk. In December 1979 she had a further episode of angio-oedema accompanied by asthma after eating cheese, and in February 1980 a similar episode occurred after taking goats' milk, which had been recommended as a substitute for cows' milk. By that time she was showing positive skin-prick reactions to grass pollens, cat and dog fur, and house dust, as well as to milk, egg, and cheese. She was also giving strongly positive results in radioallergosorbent tests (Phadebas RAST score system, 0-4) with cat fur, egg, cows' milk, and cheese (table).

Total serum IgE concentrations and radioallergosorbent test scores, June 1977 to September 1980. (Radioallergosorbent test scores of 2-4 considered positive)

Date	Total IgE (U/ml)	Radioallergosorbent test scores			
		Cat fur	Egg	Milk	Cheese
June 1977	2150	3	1	0	0
October 1978	5100	3	3	2	2
June 1979	3900	3	3	3	2
January 1980	6200	3	3	4	3
June 1980	5800	3	2	4	3
September 1980	7230	4	2	4	3

During the next six months she had frequent episodes of angio-oedema without obvious cause and the total serum IgE concentration increased to 7230 U/ml. She therefore began oral sodium cromoglycate (Nalcrom) in October, 1980, with apparent improvement initially. In December, however, despite advice to the contrary, she ate macaroni cheese, which precipitated a severe episode of angio-oedema accompanied by bronchospasm, necessitating treatment with intravenous hydrocortisone and chlorpheniramine. Despite increasing the dose of sodium cromoglycate she had further exacerbations of the eczema on her face and in the flexures with episodes of angio-oedema. She continued to pose a considerable management problem.

Comment

Most patients with atopic eczema develop the condition during the first two years of life, but on occasion it may present later: our patient developed the condition during adulthood. The causal relation between food allergy and atopic eczema remains controversial. A recent double-blind controlled cross-over study[3] showed that out of 20 children with atopic eczema aged 2-8 years, most responded more favourably to a diet which excluded cows' milk and eggs, though there was not complete clearing of the skin condition.

Our patient was interesting not because she developed atopic eczema in early adulthood but because it appeared soon after an episode of infectious mononucleosis, which being a viral infection of lymphocytes might well upset mechanisms of immune regulation. This would accord with Katz's hypothesis of "allergic breakthrough,"[1] and certainly in children viral infections have been associated with the first manifestations of atopy.[4] Our patient's atopic eczema presented several months before the manifestations of food allergy, and this is backed up by the results of prick tests and radioallergosorbent tests: this might argue against the possibility that atopic eczema and food allergy are causally related but rather that both may result from lack of immunosuppression, which has been shown to be a feature of atopy.[5]

1 Katz DH. The allergic phenotype. Manifestation of "allergic breakthrough" and imbalance in normal "damping" of IgE antibody production. *Immunol Rev* 1978;**1**:77-108.

2 Merrett TG, Pantin CFA. Increasing the precision and speed of the separation step in radio-immunoassays. *Clin Chim Acta* 1975;**65**:131-4.

3 Atherton DJ, Sewell M, Soothill JF, Wells RS, Chilvers CED. A double blind controlled cross-over trial of an antigen-avoidance diet in atopic eczema. *Lancet* 1978;i:401-3.

4 Frick OL, German DF, Mills J. Development of allergy in children. Association with virus infections. *J Allergy Clin Immunol* 1979;**63**: 228-41.

5 Strannegard IL. Lymphocyte stimulation with phorbol myristate acetate in atopic and non-atopic individuals. *Int Arch Allergy Appl Immunol* 1979;**58**:175-81.

(*Accepted 15 July 1981*)

Department of Dermatology, Royal Infirmary, Edinburgh EH3 9YW
R StC BARNETSON, MD, MRCP, consultant dermatologist
R A HARDIE, MRCP, lecturer in dermatology

RAST Allergy and Research Unit, Benenden, Kent
T G MERRETT, PHD, director

Renal embolisation for urinary fistula caused by irreparable ureteric injury

Urinary leakage from ureteric damage during colonic surgery is a rare complication. In two elderly poor-risk patients not fit for further surgery it was successfully stopped by transfemoral catheterisation of the appropriate renal artery and embolisation of the kidney.

Case reports

CASE 1

An 84-year-old woman presented with left iliac fossa pain, constipation, and weight loss over two months. Examination showed a hard mass in the left iliac fossa and barium enema confirmed a carcinoma of the sigmoid colon.

At operation an extensive colonic tumour affecting the left ureter was found. This was divided and ligated with two ligatures during the resection of the growth. A Hartmann operation was performed with closure of the rectal stump and formation of end colostomy. A drain was inserted into the left iliac fossa. Urinary leakage occurred on the third postoperative day and continued with volumes ranging from 500 to 900 ml urine a day. Intravenous pyelography confirmed a leak from the left ureter. In view of her age and frail condition surgery was not justified, but three weeks after the original operation, under local anaesthetic, the right femoral artery was catheterised and a catheter manipulated into the left renal artery. This was embolised with Gelfoam and a wire spring to achieve permanent occlusion. Urinary leakage ceased immediately and there were no sequelae. The patient was discharged to a nursing home three weeks later.

CASE 2

A 66-year-old man was admitted with diarrhoea, abdominal pain, and weight loss of six months' duration. Barium enema showed a malignant

stricture in the sigmoid colon. At laparotomy a large carcinoma was found in the sigmoid colon and a second similar lesion was found in the transverse colon. While resecting the large sigmoid growth a portion of the right ureter was removed. A primary colonic anastomosis was performed and covered by a double-barrelled colostomy fashioned in the transverse colon after resection of the second lesion. Urinary leakage occurred soon after operation and an intravenous pyelogram confirmed a leak from the right ureter. Urinary leakage continued postoperatively, and because of the poor general health of the patient renal embolisation was considered. The left femoral artery was catheterised under local anaesthesia and a right renal arteriogram was obtained. Two, or most probably three, renal arteries were seen supplying the right kidney and embolisation of two of these was achieved with Gelfoam and wire springs. Urinary leakage was considerably diminished subsequently. A further intravenous pyelogram showed minimal delayed excretion from the right kidney, suggesting perfusion from a small accessory artery. Leakage ceased completely after two months.

Comment

Ureteric damage may occur during resection of any extensive colonic or pelvic growth and if it is recognised at the time primary repair or transureteroureterostomy, reimplantation into the bladder and nephrectomy are the procedures of choice.[1 2] Ureteric ligation is simpler, but there is a risk of subsequent infection and leakage. If this occurs, or if the injury is not recognised at the time, the surgeon is faced with the prospect of further surgery in the postoperative period if the leak does not stop spontaneously. Renal embolisation is an alternative in the elderly, poor-risk patient not fit for further surgery, provided contralateral renal function is adequate. This can be assessed by intravenous urography or divided renal function tests with a renal scan. Leakage of urine by vesicoureteric reflux must also be excluded.

In one patient the leakage ceased dramatically, and was reduced in the second patient, though it did not stop completely for several weeks owing to a small aberrant renal artery. Both patients left hospital after a simple procedure and neither experienced any appreciable loin pain. Transrenal ureteric embolisation with direct closure of the defect has also been described.[3]

Therapeutic embolisation is now widely practised by radiologists for several indications including renal tumours, hepatic metastases, bleeding oesophageal varices, and complicated arteriovenous fistulae.[4] In certain poor-risk patients it would appear to be useful for post-operative ureteric leakage.

[1] Mendez R, McGinty DM. The management of delayed recognised ureteral injuries. *J Urol* 1978;**119**:192-3.
[2] Bright TC, Peter PC. Ureteral injuries secondary to operative procedures. *Urology* 1977;**9**:22-6.
[3] Gunther R, Marberger M, Klose K. Transrenal ureteral embolization. *Radiology* 1979;**132**:317-9.
[4] Allison DJ. Therapeutic embolizations. *Br J Hosp Med* 1978;**20**:707-15.

(*Accepted 8 July 1981*)

St Helier Hospital, Carshalton, Surrey SM5 1AA
A R L MAY, FRCS, senior surgical registrar
E A NORTH, FRCR, consultant radiologist
A G NASH, FRCS, consultant surgeon

Localised amyloid deposit producing paraplegia

Localised deposits of amyloid have been described in the bladder, larynx, lung, and breast[1] without evidence of generalised amyloidism. Although amyloidosis may be localised to the central nervous system, and localised disease of the gasserian ganglion[2] has been described, we have been unable to find previous reports of localised amyloid affecting the spinal extradural space.

Case report

A 76-year-old woman was admitted with a six-month history of increased weakness and paraesthesis of the legs with nocturia and incontinence of urine. On examination she had a spastic paraparesis with associated loss of position sense in the feet and vibration sensation in the legs. Coned thoracolumbar views with tomography showed a left paraspinal tumour containing calcification adjacent to a considerably narrowed disc space. Myelography showed a complete block at the level of T11/12. At operation a friable extradural tumour was removed. The result of histological staining with Congo Red was positive, and there were a few clumps of well-differentiated plasma cells and a few multinucleated giant cells.

No further evidence of amyloidosis has been found: results of bone-marrow examination showed no abnormality, and plasma immunoglobulin concentrations were within normal range. Erythrocyte sedimentation rate was normal, and results of analysis of urine for Bence Jones proteinuria was negative. Videocystometry showed uninhibited detrusor contractions with instability at 300 ml filling.

After intensive rehabilitation the patient returned home to live independently and continence returned.

Comment

Amyloid deposits may occur as part of aging[3] or may be secondary to conditions such as rheumatoid arthritis, chronic infection, or myelomatosis. In this case, although plasma cells were found in the tumour specimen, there was no other evidence of myeloma either biochemically or in bone-marrow sections.

Amyloidosis is found in about one-quarter of patients with paraplegia at necropsy,[4] but this is the only case we can find where the paraplegia was caused by the amyloid deposit.

[1] Kyle RA, Bayrd ED. Amyloidosis: review of 236 cases. *Medicine (Baltimore)* 1975;**54**:271-99.
[2] Daly DD, Love JG, Dockerty MB. Amyloid tumour of the gasserian ganglion: report of a case. *J Neurosurg* 1957;**14**:347-53.
[3] Anonymous. The senile amyloidoses. *Br Med J* 1981;**281**:846.
[4] Malament M, Friedman M, Pschibul F. Amyloidoses in paraplegia. *Arch Phys Med Rehabil* 1965;**46**:406-11.

(*Accepted 14 July 1981*)

Department of Geriatric Medicine, Withington Hospital, Manchester M20 8LR
B K PRASAD, MRCP, senior registrar
KEITH ANDREWS, MRCP, consultant geriatrician

Department of Neurosurgery, Manchester Royal Infirmary, Manchester M13 9WL
J DUTTON, FRCS, consultant neurosurgeon

Department of Pathology, University of Manchester, Manchester M13
H REID, MB, BS, lecturer in neuropathology

Skin necrosis after heparin injection

Skin necrosis is an uncommon complication of anticoagulant therapy with coumarin derivatives and is extremely rare after heparin administration.[1-3] Only three such patients have been reported to the Committee on Safety of Medicines (personal communication) and we here present the fourth.

Case report

A 79-year-old man was admitted for surgical treatment of his ischaemic left foot. He was given 5000 units of porcine sodium heparin containing chlorocresol preservative (Pularin, Duncan Flockhart) subcutaneously into the abdominal wall before femoral arteriography; he had never been given heparin before. Five days later he was given 10 000 units of heparin intravenously during exploration of the left femoral artery and received three further subcutaneous doses of heparin, each of 5000 units, on the second and third days after operation. By the fourth day areas of skin necrosis were noted at the sites of the three postoperative heparin injections.

These areas were up to 2·5 cm across. Two were excised, and they showed the following histological changes. Blisters were present in the epidermis with some haemorrhage into the blister spaces, and haemorrhagic infarction extended into subcutaneous fat. An acute necrotising angiitis was also present, affecting primarily the small dermal blood vessels, but also extending into the subcutaneous fat (see figure). This was associated with fibrinoid changes and infiltration of the walls and adjacent tissue by polymorphonuclear leucocytes. Some of the leucocytes showed fragmentation of their nuclei (leucocytoclastic vasculitis).