Hospital, for taking blood specimens; and Drs Goodman and Lall for access to clinical information.

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Gastric secretion in peripheral vascular disease

An association has been reported between arteriosclerotic cardiovascular disease and peptic ulcer.[1] Both gastric and duodenal ulcers occur more frequently than expected in necropsy studies and retrospective reviews of patients with abdominal aortic aneurysms. It has even been suggested that elective vagotomy should be carried out at the same time as aortic reconstruction. Duodenal ulcer has also been associated with "occlusive" peripheral vascular disease.[2] We therefore investigated basal and maximal acid output in patients with occlusive peripheral vascular disease who had no known peptic ulcer.

Patients, methods, and results

We studied a series of 71 consecutive patients who presented between March 1977 and January 1978 to the vascular unit with occlusive arteriosclerotic peripheral vascular disease. They were under the age of 80, their claudication or rest pain was sufficiently severe to warrant translumbar aortography, and they were fit for general anaesthesia. Eleven patients (16%) had already been treated for peptic ulcer (one gastric, 10 duodenal), and they were not studied further. Fourteen of the remaining 60 were unwilling to swallow the tube and in four the test was technically unsatisfactory. Finally, 42 (32 men and 10 women) underwent satisfactory acid secretion studies. All the patients were Caucasian. Their mean age was 59·3 years—men 58·6 (range 41-69), women 61·4 (range 44-77). Their mean weights were: men 69·1 kg, women 60·8 kg. Distribution of the ABO blood groups was in the accepted British range. Only one man and one woman were non-smokers. Three men and two women were diabetic. Eight men and one woman had had myocardial infarcts. Ten men (31%) and nine women (90%) complained of dyspepsia (the distribution of dyspepsia was the same in those patients who could not tolerate the test as for those who completed it).

The acid secretory state of the 42 patients was established by a standard basal-pentagastrin gastric function test. After an overnight fast a nasogastric tube was passed. Basal acid output (BAO) was measured over 30 minutes and was followed by a 90-minute intravenous infusion of pentagastrin (6 μg/kg/h). Peak acid output (PAO) was calculated in mmol/h by trebling the highest 20-minute acid output. The results have been compared with previous measurements of basal and maximal acid output by one of us (JHB) in 31 people without dyspepsia[3] and 60 patients with duodenal ulcer[4] aged 30 years or older. Significant differences were calculated by Student's *t* test for peak acid output and by the non-parametric Mann-Whitney U test for basal acid output because these measurements were not normally distributed.

In both men and women with peripheral vascular disease basal acid output was significantly higher than it was in normal people and not statistically different from output in patients with duodenal ulcer (table). Peak acid output in women with peripheral vascular disease was also significantly higher than in normal women and not significantly different from output in women with duodenal ulcer. The peak acid outputs in the men in our study were significantly higher than in normal men but significantly lower than in men with duodenal ulcer.

Comment

Patients with peripheral vascular disease without known ulcer disease may be acid hypersecretors almost in the duodenal ulcer range, but we cannot explain this association. Smoking may cause peripheral vascular disease but probably does not produce either duodenal ulcers or gastric hypersecretion.[5] It is impracticable to measure gastric secretion in all patients undergoing vascular operations, but we are currently assessing the prophylactic use of cimetidine.

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Difficulty in stopping lithium prophylaxis?

Textbooks and articles about lithium prophylaxis for recurrent manic-depressive psychosis tend to imply that, once begun, prophylaxis should continue indefinitely.[1 2] Little has been written about the indications for stopping lithium or when and how to do this. Our experience in a lithium clinic suggests that attempts to stop the drug can result in unexpected clinical difficulties. This paper reports the development of an acute confusional state associated with stopping lithium.

Case report

A woman, now aged 58, with a bipolar manic-depressive psychosis required six hospital admissions between 1964 and 1971. Lithium prophylaxis was begun in 1971. She was given lithium carbonate, between 800 and 1600 mg/day, by mouth. Serum lithium concentrations always remained within our "therapeutic range" of 0·5-1·5 mmol(mEq)/l during treatment. In November 1977 she asked to stop taking lithium. She had been well for six years, had not needed hospital admission during this period, and no

Comparison of basal (BAO) and peak acid outputs (PAO) in patients with peripheral vascular disease (PVD) with published data in normal subjects[3] and patients with duodenal ulcer[4]

	BAO (mmol/h)				PAO (mmol/h)			
	No	Median	Range	P	No	Mean	Range	P
Men:								
Normal	16	0·6	0-3·8	<0·00003	16	18·7	0·3-45·0	<0·02
PVD	32	1·8	0-10·6	NS	32	32·3	1·8-69·3	<0·005
DU	41	2·8	0·1-17·9		43	41·8	15·0-66·6	
Women:								
Normal	15	0·2	0-2·7	<0·025	15	8·0	0·4-15·8	<0·001
PVD	10	1·5	0-7·6	NS	10	29·8	10·8-52·5	NS
DU	17	1·9	0-5·7		17	32·4	18·8-82·6	

drugs other than benzodiazepines for night sedation had been prescribed for her since early 1973. We agreed to her request. In February 1978 she was asked to reduce her dose of lithium of 1200 mg/day by 400 mg at intervals of three weeks and then to stop treatment. In April, two weeks after stopping lithium, her husband reported that she had become unwell. She was found to be mildly confused and anxious but not depressed or excited. We thought that her condition might be a lithium withdrawal effect, so lithium was restarted at a dose of 400 mg/day. After 10 days she had improved, so lithium again was stopped. After two days she relapsed and was admitted to hospital. She was unkempt and her fluctuating mental state was characterised by mild impairment of consciousness, brief lucid intervals, restlessness, perplexity, incoherent speech, and labile mood. Assessment of cognition was impossible because she was distractable and inattentive. The clinical picture resembled an acute toxic confusional state, but physical examination and investigations showed no abnormality. Lithium was restarted at a dose of 1200 mg/day. A fortnight later her mental state was normal but she remained amnesic for the period of the acute illness. Psychological tests done just before discharge showed her to have average intelligence with no areas of cognitive impairment.

Comment

Adverse reactions to psychotropic drug withdrawal are well known,[3] but an acute psychosis, with the features of this case, has not been reported after withdrawal of lithium. We found no other cause for our patient's illness. The link between it and stopping lithium is inferred. She returned to her former state of health when lithium was restarted, and remains well taking the drug.

General principles and the risks of permanent damage from lithium side effects suggest that its use in each case should be reviewed regularly. When deciding to stop lithium prophylaxis consideration should be given to when and how to do it. From our experience we think that lithium might be best withdrawn slowly over some months. Since some patients resist when advised that it is in their interest to stop taking lithium it might be better if they were told initially that treatment would be given for only a specified period in the first instance.

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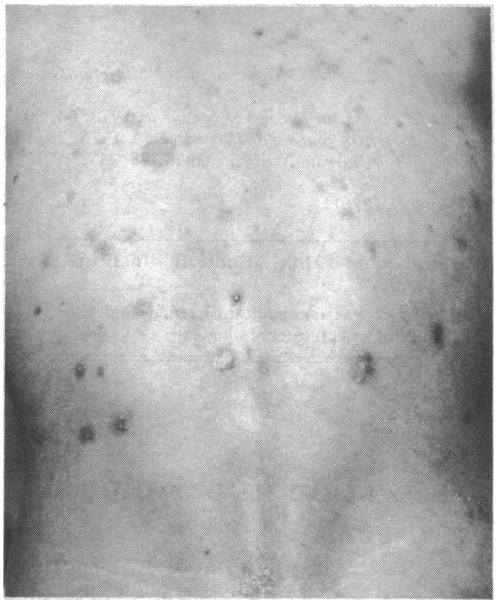
Psoriasis developing during dialysis

Remission of psoriasis in patients receiving dialysis for renal failure has been reported.[1-3] Peritoneal dialysis has therefore been advocated for treating refractory psoriasis in patients with normal renal function.[4 5] We report the case of a patient whose psoriasis not only first developed during dialysis for chronic renal failure but also persisted unchanged despite three years of regular haemodialysis.

Case report

A 41-year-old white man was noted to have albuminuria in 1955 when aged 18. Intravenous pyelography showed that the right kidney was absent. He was lost to follow-up until January 1971, when he presented with end-stage renal failure. Regular haemodialysis was instituted in July 1971 via an arteriovenous shunt in the right leg. Home dialysis began in December 1971. An arteriovenous fistula in his right forearm was put into regular use in November 1975. In December 1975 he had developed typical guttate patches of psoriasis along the line of the fistula together with patches scattered widely over the trunk and scalp. He had no family history of psoriasis. The psoriasis persisted despite active treatment including at varying times dithranol, coal tar and salicylic acid ointment, and topical corticosteroids. In November 1978 he had extensive psoriasis of the scalp and hairline, widespread small plaques on the trunk (figure), and Köebner lesions along the line of the fistula in the right forearm and at venepuncture sites in the antecubital fossae. Several finger nails were pitted. The histology of a lesion on the chest showed changes characteristic of psoriasis. Haemodialysis has continued throughout apart from a two-week interlude of peritoneal dialysis after the arteriovenous fistula clotted in May 1978. Dialysis has been adequate, biochemical and clinical criteria satisfactory, and the patient has carried on in full-time employment.



Widespread psoriatic plaques on back.

Comment

McEvoy and Kelly[1] in 1976 first reported complete clearing of psoriasis in a patient with renal failure two weeks after starting dialysis. The remission lasted for a year with dialysis and for a further 11 months after a successful renal transplant and immunosuppressive treatment. Muston and Conceicao[2] recorded clearing of psoriasis in one patient within eight weeks of starting dialysis and remission was long lasting. In another patient psoriatic lesions vanished within three weeks and remission was maintained for two and a half years with dialysis, but psoriasis recurred after cadaveric transplantation and cessation of dialysis. Chen *et al*[3] also reported remission in two patients on haemodialysis, one of whom has remained clear for over five years. Twardowski *et al*[4] used peritoneal dialysis for severe disabling psoriasis in the absence of renal failure. Two patients unresponsive to conventional treatment rapidly cleared with this treatment. A third patient with erythrodermic pustular psoriasis failed to respond. A further 16 patients with severe psoriasis without renal failure were treated with peritoneal dialysis over an eight-month period. All were somewhat improved, half substantially so.[5]

Owing to these reports it has been suggested that a noxious metabolite, or "psoriasis factor," accumulating over many years, in some way stimulates the increased epidermal cell turnover characteristic of psoriasis, and that it is better removed by dialysis than by normal renal clearance. Our case is the first to be reported of psoriasis developing in a patient already being treated with haemodialysis. It conflicts with the "psoriasis factor" theory. We therefore counsel caution against optimistic expectations of dramatic benefit from dialysis in patients with psoriasis.

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