of opiates. Presumably naloxone antagonises opiate suppression of the sympathetic system resulting in a sudden increase in its activity.

Our patient had clinically unsuspected but pathologically proved cardiomyopathy. Cardiac arrhythmias are well recognised in patients with alcoholic cardiomyopathy as well as in heavy drinkers with no clinical evidence of heart disease.⁴ We fear that in an emergency adverse cardiovascular reactions are seldom recognised as being due to reversal of the effects of opiates unless they occur repeatedly. In reversing opiate coma naloxone should be used cautiously and with electrocardiographic monitoring in patients with pre-existing cardiac disease or those predisposed to cardiac arrhythmias. Clonidine might possibly be useful because it abolishes increases in pulse and blood pressure after reversal of opiate effects with naloxone.⁵

¹ Pallasch TJ, Gill CJ. Naloxone associated morbidity and mortality. *Oral Surg* 1981;52:602-3.

² Lawrence JR, Lee FR. Ventricular fibrillation after narcotic withdrawal. *Lancet* 1975;ii:717.

³ Desmonts JM, Bohm G, Conderc E. Hemodynamic responses to low doses of naloxone after narcotic-nitrous oxide anesthesia. *Anesthesiology* 1978;49:12-6.

⁴ Ettinger PO, Wu CF, De la Cruz C, et al. Arrhythmias and the "holiday heart": alcohol-associated cardiac rhythm disorders. *Am Heart J* 1978;95:555-62.

⁵ Fantozzi R, Luciani G, Masini E, et al. Clonidine and naloxone induced opiate withdrawal: a comparison between clonidine and morphine in man. *Subst Alcohol Actions Misuse* 1980;1:369-73.

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Treatment with captopril for peripheral ischaemia induced by ergotamine

Although clinical manifestations of ergotism have been known since antiquity, severe cases of ergotism are still occasionally encountered. Most of these are caused by overdosage of or undue sensitivity to ergotamine tartrate, which is used to treat migraine or as an abortifacient. Although peripheral ischaemia induced by ergotamine may resolve solely on withdrawal of the drug, this usually requires several days; in most cases active treatment is indicated to shorten the duration of ischaemia and reduce the risk of irreversible ischaemic damage. We report the rapid reversal of ischaemia induced by ergotamine on oral treatment with the angiotensin converting enzyme inhibitor captopril.

Case report

A 43 year old man was admitted to hospital with intense pains in his hands and feet, which had started one week earlier. He was a heavy smoker, consuming four packs of cigarettes daily. For 20 years he had been taking one to six tablets daily of a preparation containing ergotamine tartrate 2 mg. Within the past two years he had developed intermittent claudication and impotence. One week before admission he had developed a febrile disease with chills and a productive cough, which was treated with amoxycillin. His pains had started shortly thereafter.

On admission he was in intense pain. Temperature was 37.6°C and pulse rate 72 beats/minute and regular. Blood pressure could not be measured by sphygmomanometry. His hands and feet were cold, cyanotic, and pulseless. Carotid and femoral pulses, however, were present, and a Doppler examination produced flow murmurs over the popliteal, posterior tibial, brachial, and radial arteries. A chest radiogram showed infiltration of the right lower lobe. Routine biochemical investigation yielded unremarkable results except for a raised serum aspartate transaminase activity of 240 IU/ml and lactate dehydrogenase activity of 450 IU/ml; both reverted to normal within a few days.

Ergotamine tartrate was stopped, and captopril, 50 mg thrice daily by mouth, was given for four days. Within three hours after the start of treatment the brachial pulses became palpable, and within six hours all peripheral

pulses could be palpated. This was accompanied by improvement in colour and temperature and the resolution of ischaemic pains. Within the following days perfusion remained excellent but burning, neuralgic pains developed on the soles of both feet. These were accompanied by reduced sensitivity to pain and vibration and impaired nerve conduction typical of peripheral ischaemic neuropathy. These abnormalities improved gradually throughout the following weeks.

Comment

Severe arterial spasm of the hands and feet is a well documented complication of treatment with ergotamine tartrate; its incidence is estimated at less than 0.01% in patients receiving therapeutic doses of the drug.¹ Factors that may aggravate ergotism or precipitate ergot toxicity include renal or hepatic insufficiency, thyrotoxicosis, and febrile diseases. In our patient, with chronic exposure to excessive amounts of ergotamine tartrate, pneumonia probably caused the sudden aggravation of symptoms.

Sympathetic blockade, surgical sympathectomy, low molecular weight dextran, anticoagulants, and intravenously or intra-arterially injected vasodilators such as tolazoline have been used to treat ergot toxicity with variable success. More recently sodium nitroprusside, a potent direct acting vasodilator, has been advocated for the management of peripheral ischaemia induced by ergotamine.² Our decision to use the angiotensin converting enzyme inhibitor captopril was prompted by the documented increase in peripheral venous renin activity in some patients with ergotamine poisoning,³ the ability of ergotamine to enhance the sensitivity of vascular smooth muscle to angiotensin,⁴ and the reported efficacy of captopril in patients with Takayasu's syndrome.⁵ The rapid response to oral captopril, as shown by the reappearance of peripheral pulsations within three hours, is similar to the response to parenteral nitroprusside. Captopril may be the drug of choice in the management of severe peripheral ischaemia induced by ergotamine because it is easily administered and is associated with limited risk at the dose used in this case.

¹ Merhoff GC, Porter JM. Ergot intoxication: historical review and description of unusual clinical manifestations. *Ann Surg* 1974;180:773-9.

² Carliner NH, Denué DP, Finch CS, Goldberg LI. Sodium nitroprusside treatment of ergotamine-induced peripheral ischemia. *JAMA* 1974;227:308-9.

³ Fedotin MS, Hartman C. Ergotamine poisoning producing renal artery spasm. *N Engl J Med* 1970;283:518-9.

⁴ Gillman AG, Goodman LS, Gilman A, eds. *The pharmacological basis of therapeutics*. 6th ed. New York: Macmillan, 1980:939-47.

⁵ Grossmann A, Knecht A, Rosenstein Z, Nussiniwitz N, Boykes C, Rosenthal T. Captopril therapy in Takayasu's syndrome [Abstract]. In: Hershko C. *Proceedings of the biannual congress of the Israel Society of Internal Medicine*. Tel-Aviv: Kenes, 1983:14.

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Multiple spontaneous ruptures of tendons in renal transplant recipient

Spontaneous rupture of tendons has been described in patients with chronic renal failure¹⁻⁴ but not to our knowledge in recipients of successful renal transplants. We report multiple spontaneous ruptures of tendons that occurred five years after transplantation in a patient with good renal function.

Case report

A 40 year old man presented in 1971 with renal failure and heavy proteinuria (4.5 g/day). Chronic glomerulonephritis was diagnosed clinically because he had suffered from scarlet fever in childhood, showed no evidence of any systemic disease, and on intravenous urography had small kidneys without scarring or calyceal clubbing. Maintenance haemodialysis was started in

1974. Routine skeletal x ray examinations and bone biopsies yielded normal results.

He received a cadaveric renal allograft in 1976 and was in excellent general health thereafter, with satisfactory and stable renal function (mean serum creatinine concentration $156 \mu\text{mol/l}$ (1.8 mg/100 ml), serum urea concentration 9.5 mmol/l (57 mg/100 ml), creatinine clearance 51 ml/min). Other biochemical variables including ionised and total serum calcium concentrations, serum phosphate concentration, and serum alkaline phosphatase activity were consistently in the normal range. Immunosuppression after transplantation was achieved with prednisolone (initially 100 mg/day , reduced progressively to a maintenance dose of 10 mg/day) and azathioprine ($2.5 \text{ mg/kg body weight/day}$). A single, mild episode of acute rejection diagnosed on the eighth day after operation was treated with additional oral prednisolone for three days.

In September 1981 he experienced sudden pain in his right lower calf while walking. Clinical examination indicated a ruptured Achilles tendon. At surgery the diagnosis was confirmed and the tendon successfully repaired. In May 1982 he suddenly developed pain and a lump in his left upper arm while changing gear in his car. Examination showed that the long head of the biceps had ruptured near its insertion at the shoulder. No treatment was considered to be necessary.

In September 1982 he noticed that he was no longer able to extend his right little finger and that a painless swelling on the dorsum of his right hand, present for at least 18 months, had enlarged considerably. Six weeks later the right ring finger also dropped, and shortly after this he became unable to extend his right middle finger (figure). Radiographs of the wrist and

Hand showing inability to extend middle, ring, and little fingers and swelling on dorsum before operation.

hand did not show any osteoporosis, erosions, or other changes indicative of hyperparathyroidism. Serum parathyroid hormone concentration was normal ($<0.1 \mu\text{g/l}$). At operation widespread synovitis of the extensor tendons was found and rupture of the middle, ring, and little finger extensors confirmed. The extensor indicis proprius was transferred and sutured to the three ruptured tendons with a good functional result. Histological examination of the resected synovium showed granulation tissue and acellular fibrin on the surface, with a non-specific chronic inflammatory reaction in the subsynovium.

Comment

The cause of the tendon ruptures in this patient is unknown. There was no evidence of amyloid deposition, rheumatoid arthritis, or other inflammatory arthropathy. Attrition of the tendons due solely to prolonged treatment with corticosteroids is unlikely because rupture of tendons has not been observed in patients receiving long term steroids for ulcerative colitis and asthma.⁵ Steroids would not have been responsible for the synovial reaction that preceded the rupture of tendons in the right hand. Secondary hyperparathyroidism, which has been implicated in tendon ruptures in patients receiving haemodialysis, seems improbable because of the lack of any biochemical or radiological evidence of the condition. The suggestion that the chronic metabolic acidosis associated with impaired renal function causes tendon ruptures in patients receiving haemodialysis does not help to explain the present case as renal function had been almost normal for over five years.¹

In the absence of any other explanation it seems likely that the rupture of tendons in this patient was in some way a consequence of renal transplantation, perhaps related to long term immunosuppression with azathioprine and prednisolone. It is therefore surprising that spontaneous rupture of tendons has not been reported in other recipients of renal transplants. Possibly such ruptures have occurred but have not been reported because of the lack of any obvious cause.

- ¹ Murphy KJ, McPhee I. Tears of major tendons in chronic acidosis with elastosis. *J Bone Joint Surg* 1965;**47A**:1253-8.
- ² Morein G, Goldschmidt Z, Pauker M, Seelenfreund M, Rosenfeld JB, Fried A. Spontaneous tendon ruptures in patients treated by chronic hemodialysis. *Clin Orthop* 1977;**124**:209-13.
- ³ Lotem M, Bernheim J, Conforty B. Spontaneous rupture of tendons. A complication of hemodialyzed patients treated for renal failure. *Nephron* 1978;**21**:201-8.
- ⁴ Rosenfeld N, Rascoff JH. Tendon ruptures of the hand associated with renal dialysis. *Plast Reconstr Surg* 1980;**65**:77-9.
- ⁵ Cooney LM, Aversa JM, Newman JH. Insidious bilateral infrapatellar tendon rupture in a patient with systemic lupus erythematosus. *Ann Rheum Dis* 1980;**39**:592-5.

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Fractures on chest radiographs as indicators of alcoholism in patients with liver disease

Alcoholism is increasingly being recognised as an important cause of illness in patients admitted to general hospitals, but the diagnosis is still often missed. Some patients at first deny excessive alcohol consumption, while in other instances doctors fail to take an adequate drinking history. Although mean red cell volume and serum γ -glutamyltransferase activity correlate with alcohol intake, their practical value as screening tests has been called into question.¹ Recently, reports of two studies claimed that the presence of fractures on chest x ray films is a useful indicator of alcoholism both in patients presenting via casualty services and in those with liver disease.^{2,3} Our impression that fractures were relatively rare in patients with alcoholic liver disease led us to carry out the study described here.

Patients, methods, and results

We studied 278 consecutive patients referred to the liver unit over 18 months who had undergone liver biopsy for the first time and in whom a chest x ray film was available for examination. In 103 patients alcoholic liver disease was diagnosed on the basis of a history of alcohol consumption exceeding 80 g/day for men or 40 g/day for women, as established by a standardised interview schedule,⁴ and liver biopsy appearances suggestive of alcoholic liver disease as judged by a specialist histopathologist. The remaining 175 patients had a variety of non-alcoholic liver diseases. Cirrhosis was present in 65 (63.1%) and 31 (17.7%) of the groups with alcoholic and non-alcoholic liver disease respectively. The mean ages of the two groups were similar (52.3 (SD 11.7) and 50.3 (16.4) years respectively).

Only nine patients with alcoholic liver disease had a fracture visible on the chest x ray film, which gives a sensitivity of such a finding for the diagnosis of alcoholic liver disease of only 8.7%. The specificity (the proportion of patients with non-alcoholic liver disease who did not have a fracture) was 96.6% (169/175), and the positive predictive value (the proportion of patients with a fracture who proved to have alcoholic liver disease) was 60%. Only five patients (4.9%) with alcoholic liver disease and two (1.1%) with non-alcoholic liver disease had bilateral or multiple rib fractures. Within each group there were no differences between the proportions of patients with cirrhosis who had fractures compared with those with precirrhotic liver disease and fractures.

Comment

Two previous surveys, in an alcoholism unit² and a gastroenterology unit,³ showed that 25-30% of alcoholic patients had rib or other fractures on chest x ray films. On the basis of the distribution and