

septicaemia is unusual.¹ We report a patient in whom staphylococcal pneumonia resulted in multiple abscesses of the skeletal muscles.

A 25 year old male was admitted with the chief complaints of high-grade fever, cough and expectoration of mucopurulent sputum of 10 days duration. Five days prior to admission, the patient complained of a painful swelling on the left side of the chest extending toward the left shoulder.

Examination revealed a toxic, dehydrated, diaphoretic, moderately built and nourished patient with a pulse rate of 132 beats/minute, respiratory rate of 40/minute regular and a blood pressure of 90/60 mmHg. The temperature was 102.4°F. Chest examination revealed bilateral coarse crepitations, more on the left side. There was a hot, tender, brawny, rubbery hard, pitting swelling on the left side of the chest anteriorly extending to the tip of the left shoulder. The movements of the left arm were markedly restricted and painful.

Investigations showed a total leucocyte count of $16 \times 10^9/l$ with 74% neutrophils showing toxic granules. ESR was 45 mm in the first hour. Blood sugar, urea, serum electrolytes and creatinine were normal. The chest X-ray displayed bilateral fluffy opacities occupying the whole of the lung fields without the presence of effusion or pneumatocele. Blood culture was repeatedly sterile. Sputum culture showed a growth of *Staphylococcus aureus*.

The patient was given cefazolin and gentamicin along with supportive treatment. During his stay in the hospital, the patient developed abscesses on the medial aspect of the right thigh extending anteriorly and on volar surface of both forearms. These abscesses were drained and revealed intramuscular collection of pus which grew *Staphylococcus aureus*. The patient showed a steady recovery.

'Tropical pyomyositis' is frequently seen in the tropics. In 90% of the cases, *Staphylococcus aureus* is the incriminating organism.² The aetiology of this condition is still unknown. Various predisposing factors have been described.¹ Defect in host immunity may have a role in the pathogenesis. Pneumonitis with abscess or pleural thickening is seen in 5% of cases of tropical pyomyositis.¹ However, staphylococcal pneumonia and septicaemia causing metastatic abscess is unusual.³ As far back as 1930, Sayers⁴ described pulmonary complications in six out of 26 cases of tropical pyomyositis of whom one had lobar pneumonia. Taylor *et al.*⁵ found at autopsy that 50% of the 19 cases had macroscopic evidence of bronchopneumonia. All these cases had presented with symptoms and signs related to the musculoskeletal system. Our case presented with respiratory complaints following which he developed multiple abscesses. From the literature that we could go through, we did not come across any report of bronchopneumonia causing pyogenic muscle abscess.

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References

1. Manson, P.E.C. & Bell, D.R. Musculoskeletal disease. In: Manson Bahar, P.E.C. & Bell, D.R. (eds) *Manson's Tropical Disease*, 19th edn. English Language Book Society, London, 1987, pp. 1130-1132.
2. Cheidozi, L.C. Pyomyositis—review of 205 cases in 112 patients. *Am J Surg* 1979, **137**: 255-259.
3. Levin, M.J., Gardner, P. & Waldrogl, F.A. Tropical pyomyositis—an unusual infection due to *Staphylococcus aureus*. *N Engl J Med* 1971, **284**: 196-198.
4. Sayers, E.G. Tropical myositis and muscle abscess. *Trans R Soc Trop Med Hyg* 1930, **23**: 385-400.
5. Taylor, J.F., Templeton, A.C. & Henderson, B. Pyomyositis. A clinicopathological study based on 19 autopsy cases Mulago Hospital 1964-1968. *East Afr Med J* 1970, **47**: 493-502.

Hyponatraemia and dopaminergic agents

Sir,

A case of symptomatic hyponatraemia following commencement of L-dopa/carbidopa therapy, given inadvertently for a drug-induced akinetic-rigid syndrome, is reported.

A 73 year old woman was referred with a year's history of progressive slowing of her mobility and mentation. Examination revealed a paucity of facial expression, shuffling gait, truncal and limb rigidity, bradykinesia, synkinesia, but no tremor. Haematological and biochemical indices were normal (sodium 136 mmol/l), as was a computed tomographic (CT) head scan. A diagnosis of Parkinson's disease was made, and the patient was commenced on L-dopa/carbidopa (100 mg/10 mg three times a day; Sinemet). Four days later she was admitted having become unsteady on her feet and fallen twice without loss of consciousness. In addition to her previous neurological signs, gait and limb ataxia were now observed. Blood tests revealed a sodium of 115 mmol/l and a plasma osmolality of 239 mosmol/kg. L-dopa/carbidopa was stopped, and a moderate fluid restriction enforced (1.5 l/day), on which regimen her sodium corrected over the next few days and the ataxia disappeared. Chest radiograph was normal.

Further questioning of her family revealed that she had been taking the thioxanthene neuroleptic flupenthixol (0.5 mg/day) for several years for depression. This too was stopped, with improvement in her akinesia and rigidity. A subcutaneous apomorphine challenge test was negative.

Lammers & Roos recently reported symptomatic hyponatraemia complicating therapy with the dopaminergic agents L-dopa/carbidopa and amantadine hydrochloride in a patient with Parkinson's disease, the probable pathophysiological mechanism being inappropriate secretion of anti-diuretic hormone (ADH).¹ In animals there is anatomical and electrophysiological evidence for a dopaminergic input to magnocellular ADH-secreting neurons in the hypothalamic supraoptic and paraventricular nuclei; dopamine has been shown to facilitate ADH release, whereas dopaminergic antagonists induce a diuresis, in normally hydrated goats.² In man, apomorphine, a mixed D1 D2 dopaminergic agonist, produces an increase in plasma ADH levels in some subjects, concurrent with a sensation of nausea, effects blocked by pretreatment with the dopaminergic antagonists halo-

peridol and fluphenazine.³ Bromocriptine, a selective D2 dopaminergic agonist, also stimulates ADH secretion in normal subjects,⁴ and has been reported to cause hyponatraemia, probably by this mechanism.⁵

Hence it is possible that chronic blockade of dopamine receptors on magnocellular neurons by flupenthixol, a combined D1 D2 dopaminergic antagonist, may have rendered them supersensitive to the effects of dopamine formed from the prescribed L-dopa. Recent studies of the reflex control of ADH release from magnocellular neurons suggest that the system is not inflexibly 'hard-wired', but functionally adaptive, reflecting complex and plastic interactions between magnocellular neurons and the environment.² This variability may explain individual idiosyncratic susceptibility to hyponatraemic complications of therapy with L-dopa and other dopaminergic agents. Its occurrence following the inadvertent use of L-dopa/carbidopa in a patient with drug-induced parkinsonism suggests that this is an effect of the drug *per se* and not a reflection of underlying neuropathology.

Acknowledgement

Thanks to Professor A.C. Williams for permission to report this patient.

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References

1. Lammers, G.J. & Roos, R.A.C. Hyponatraemia due to amantadine hydrochloride and L-dopa/carbidopa. *Lancet* 1993, **342**: 439.
2. Cunningham, E.T. Jr & Sawchenko, P.E. Reflex control of magnocellular vasopressin and oxytocin secretion. *Trends Neurosci* 1991, **14**: 406–411.
3. Rowe, J.W., Shelton, R.L., Helderman, J.H., Vestal, R.E. & Robertson, G.L. Influence of the emetic reflex on vasopressin release in man. *Kidney Int* 1979, **16**: 729–735.
4. Robinson, B.G., Clifton-Bligh, P., Posen, S. & Morris, B.J. The effect of bromocriptine on circulating vasopressin. *Clin Sci* 1982, **63**: 367–372.
5. Marshall, A.W., Jakobovits, A.W. & Morgan, M.Y. Bromocriptine-associated hyponatraemia in cirrhosis. *Br Med J* 1982, **285**: 1534–1535.

Cutaneous leukocytoclastic vasculitis preceding renal cancer

Sir,
Cutaneous leukocytoclastic vasculitis (CLV) has been sporadically reported in association with malignant haematological diseases.^{1,2} Three cases of CLV preceding renal carcinoma have been reported.^{1,3} I describe a patient in whom the occurrence of CLV led to the discovery of clear cell renal carcinoma.

A 56 year old man presented with a one week history of

palpable purpura of lower limbs evolving into papular, vesicular and pustular lesions with umbilications, and fever and arthritis of the right ankle joint. Skin biopsy revealed small vessel vasculitis with intense intravascular and perivascular neutrophil infiltration, leukocytoclasia, fibrinoid necrosis of the vessel walls, and extravasation of erythrocytes.

Ultrasound of the abdomen showed a right renal tumour. A tumour measuring 4 × 5 cm without capsular, vascular or lymph node involvement was removed at nephrectomy. Histopathology revealed clear cell renal carcinoma and crescentic glomerulonephritis suggestive of microscopic polyarteritis.

One month after nephrectomy, the urine showed proteinuria (1+) and numerous dysmorphic erythrocytes. He was put on oral cyclophosphamide (100 mg/day) and prednisolone (60 mg/day). Over the next 3 months, the urinary abnormalities cleared and renal function returned to normal. Repeat computed tomographic scans showed no tumour relapse or metastases. Twenty months after nephrectomy, purpuric lesions and arthritis similar to those of the initial presentation appeared with active urinary sediment and deranged renal function (BUN 7 mmol/l; creatinine 146 mmol/l). The recurrence as with the original episode was anti-neutrophil cytoplasmic antibody negative. In view of the recurrence of CLV and microscopic polyarteritis of the solitary left kidney, therapy with cyclophosphamide and prednisolone was reinstated, and one month later neither vasculitis nor tumour metastases was seen. The patient was seen 24 months later without relapse of CLV or neoplasia.

Paraneoplastic manifestations of renal cancer (inter-*neuro*'s tumour) are frequent and numerous.⁴ Renal tumour antigen may bind up in circulating immune complexes and induce CLV¹ by activating the complement cascade causing lysosomal enzyme release from the accumulated polymorphonuclear cells leading to vessel wall injury, thrombosis, and haemorrhage.⁵ CLV has not hitherto been recognized as a cutaneous paraneoplastic syndrome. It should perhaps be included in the long list of paraneoplastic manifestations of renal cancer, although the relapse of CLV without apparent recurrence of tumour suggest coincidence.

Acknowledgements

I thank Dr J.N. Pande and Dr Sachi Diwan for useful advice.

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References

1. Lacour, J.P., Castanet, J., Perrin, C., Vitetta, A. & Ortonne, J.P. Cutaneous leukocytoclastic vasculitis and renal cancer; two cases. *Am J Med* 1993, **94**: 104–108.
2. Longley, S., Caldwell, J.R. & Panush, R.S. Paraneoplastic vasculitis; unique syndrome of cutaneous angiitis and arthritis associated with myeloproliferative disorders. *Am J Med* 1986, **80**: 1027–1030.