Letters to the Editor

Recurrent myositis with infection

Sir,

We would like to report a sequel to the clinical case described by Bennett *et al.*¹ They described a previously unreported complication of mycoplasma infection, namely myositis. However, the same patient recently represented after an interval of 3 years with the same symptoms.

He gave a 4-day history of nausea, anorexia, lethargy and night sweats. This was followed by myalgia affecting his thighs and lower back, and on the day of admission, jaundice and pruritus. On examination he was deeply jaundiced, had fever of 37.5°C and a sinus tachycardia. Abdominal examination revealed tenderness in the right hypochondrium, no viscera were enlarged. His quadriceps were noted to be slightly wasted, tender and weak. All reflexes were normal and plantar responses flexor.

Investigations revealed a total white cell count of $5.9 \times 10^9/l$. Liver function showed a bilirubin of 200 µmol/l (normal ranges 1–26), alkaline phosphatase 371 U/l (7–320), aspartate transaminase 3387 IU/l (5–30). His creatine kinase was grossly elevated at over 40,000 U/l (1–200) and no myoglobin was detected in his urine. An infection screen excluded recent *Mycoplasma pneumoniae*, chlamydia, *Coxiella burnettii*, Epstein-Barr virus, cytomegalovirus, influenza A and B infection. Hepatitis B serology was negative but IgM and IgG antibodies to hepatitis A were both detected indicating recent infection. His antinuclear factor and liver auto-antibodies were negative.

Seven days later, his creatine kinase had fallen to 596 U/l and after 3 weeks was normal. At 3 weeks his bilirubin had fallen to 50 µmol/l, with a normal alkaline phosphatase and an aspartate transaminase of 92 U/l.

We feel that this patient has a particular tendency to develop acute myositis in association with acute intercurrent infections and that this problem should not be considered as a specific complication of *Mycoplasma pneumoniae*. The occurrence of severe myositis in association with acute viral illness is well documented² although the mechanisms are not clear.

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References

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2. Lane, R.J.M. Muscle involvement in systemic and iatrogenic disease. In: Kennard, C. *Recent Advances in Clinical Neurology*, vol. 5. Churchill Livingstone, Edinburgh, 1988, pp. 201–238.

Unexpected first dose hypotensive reaction to enalapril

Sir,

The reason why some patients suffer a marked hypotensive reaction to small initial, but not larger subsequent, doses of an angiotensin-converting enzyme inhibitor (ACEI) is not clear.

The first ACEI dose causes a rapid reduction in circulating angiotensin II levels. Susceptible patients¹ may now develop symptomatic hypotension because of a failure of compensatory autonomic nervous system reflexes. This dysfunction is perhaps related to the chronically raised tissue angiotensin II levels of the underlying disease,² or therapy itself.³

This first dose, however, will also begin to reduce tissue angiotensin II levels. This effect, which is probably responsible for the long-term benefits of ACEI therapy,⁴ may enhance compensatory responses sufficiently enough to cope with subsequent ACEI doses. Improved autoregulation of cerebral blood flow,⁵ and induction of angiotensin-converting enzyme⁶ may also be factors.

Packer *et al.*⁷ reported hypotensive reactions after enalapril (10 mg) in 2 of 42 patients who had tolerated a single test dose of captopril (25 mg) given 24 hours previously. One would, however, expect antecedent long-term therapy with one ACEI to render the patient resistant to a first-dose response with an equivalent dose of another ACEI. The following case report provides evidence to the contrary.

A 52 year old male Caucasian was found to have severe hypertension at a routine medical examination, with blood pressures consistently of the order of 190/120 mmHg. A thorough assessment failed to identify a cause of secondary hypertension, or evidence of end-organ damage. Over the following 15 months, treatment with various anti-hypertensives, such as atenolol, triamterene and hydrochlorothiazide (Dyazide, Bridge), hydralazine, prazosin, and slow-release nifedipine (Adalat Retard, Bayer), conferred only marginal benefit. Lack of compliance was considered most unlikely because of the adverse effect of the uncontrolled hypertension on the patient's Service career.

He was then started on captopril (Capoten, Squibb), an initial test dose (12.5 mg) of which was well-tolerated. The captopril dosage was gradually increased to 25 mg thrice daily, but, despite the subsequent addition of frusemide 40 mg daily, his blood pressure control remained unsatisfactory.

After 3 months, the captopril was replaced by 10 mg enalapril (Innovace, MSD). A hypotensive reaction occurred, which responded to head-down tilt of the bed (Figure 1). The next day, a 5 mg dose of enalapril was well-tolerated.

Despite increasing the enalapril dosage to 40 mg daily over the next few months, significant hypertension persisted, and a combination of atenolol and minoxidil was eventually required.

The initiation of ACEI therapy is discussed by Reid,⁸ who suggests that a starting dose of captopril 6.25 mg