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. 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 × 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 U/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 burnetii, 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 autoantibodies 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 documented although the mechanisms are not clear.

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References

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 patients 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, or therapy itself.

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, may enhance compensatory responses sufficiently enough to cope with subsequent ACEI doses. Improved auto-regulation of cerebral blood flow, and induction of angiotensin-converting enzyme may also be factors.

Packer et al. 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, who suggests that a starting dose of captopril 6.25 mg
should be used in 'at risk' patients. This line of action should ensure that any symptomatic first-dose responses are short-lived, but it may lead to a false sense of security. This case demonstrates that the pharmacological differences of captopril and enalapril are more profound than may be realised, and I would therefore suggest that 'test-dosing' should be done with a small dose of the same ACEI that is intended for long-term therapy.

The relative bradycardia seen during this patient's hypotensive reaction is noteworthy, and may suggest that tests of autonomic function could be used to screen for at-risk patients.

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References

Facioscapulohumeral syndrome with cardiomyopathy

Sir,
Cardiomyopathy and electrocardiogram (ECG) abnormalities have been associated with several neuromuscular disorders. In facioscapulohumeral FSH syndrome, cardiac involvement may be present in some patients in the form of atrial paralysis or even generalized cardiomyopathy.

Patients with genetic (FSH) weakness may have histological evidence of myopathy, neuropathy and inflammation. The term FSH syndrome is presently used to designate this entity.

We have investigated a 12 year old boy with clinically and electromyographically proven FSH syndrome who had normal early motor development until age 6 years when mild weakness in his arms was noted.

On examination, he appeared healthy and showed marked muscle wasting and weakness of the upper arms, the neck flexors and shoulder girdle. He also had a mild facial weakness when whistling. There was no pelvic or peroneal muscle involvement. Tendon reflexes were slightly reduced.

Serum cardiac enzyme levels were within normal limits. Electromyogram (EMG) examination indicated a myopathic process with a normal motor nerve conduction velocity. A muscle biopsy was normal apart from a few central nuclei. Chest X-ray was normal. The surface ECG showed only inverted T waves. The echocardiography findings were suggestive of a cardiomyopathy. The posterior ventricular wall motion was found to be slightly diminished. Cardiac catheterization revealed a probable cardiomyopathy.

To verify the diagnosis an endomyocardial biopsy was done from the right ventricle during catheterization. Findings disclosed unusual morphological changes; fatty infiltration amongst the bundles and hyperkinetic nuclei along with increase in cardiac connective tissue.

Our case shows that the presence of cardiac involvement in muscle disorders cannot always be detected by performing solely simple-to-perform cardiac evaluation studies with chest X-ray, ECG and echocardiography. Cardiac muscle biopsy is an invasive test. We do not propose it should be done more routinely, but the point should be kept in mind that normal findings in the screening tests mentioned above do not necessarily exclude possible cardiac involvement.

Unexpected first dose hypotensive reaction to enalapril.

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