Postgrad Med J (1994) **70**, 145 - 147

Letters to the Editor

Biochemical diagnosis of myocardial infarction? Cardiac enzymes or *caveat emptor*

Sir,

The use of biochemical markers, and in particular serum creatine kinase (CK) activity, in the diagnosis of myocardial infarction has received renewed interest because of the advent of thrombolytic therapy,[1] which has resulted in pressure to make an early diagnosis in order to maximize the efficacy of treatment. Unfortunately, CK is also present in skeletal muscle and is released following muscle trauma, ischaemia or any inflammatory episode.[2] This limits its diagnostic specificity as a test for myocardial infarction. We wish to report a case of initial misdiagnosis in a patient at high risk of myocardial infarction because of familial hypercholesterolaemia, who was subsequently found to have a drug-induced skeletal myopathy caused by a lipid-lowering drug.

A 46 year old man with known coronary artery disease was admitted to the coronary care unit following several episodes of crushing central chest pain. An electrocardiogram (ECG) showed no evidence of ischaemia but his serum CK activity was 1,380 U/l (reference range 24-195). In view of the history and high serum CK activity a diagnosis of myocardial infarction was made. Over the next 48 hours, he experienced no further chest pain and no ECG changes developed, However, the serum CK activity continued to rise, reaching 5,825 U/l at 48 hours. A possible skeletal muscle origin for the CK was therefore considered. There had been no muscle trauma or vigorous exercise. He denied excessive alcohol intake. He was biochemically euthyroid. Serum CK-MB concentration was normal (less than 10 ng/ml), and cardiac troponin-T was undetectable in all samples. On subsequent questioning it emerged that he had been commenced on treatment with simvastatin for familial hypercholesterolaemia 10 days before admission. The most likely cause for the rise in serum CK activity was therefore a simvastatin-induced myopathy. Treatment was stopped, the serum CK activity peaked within 72 hours and then fell to normal levels within 21 days. The original diagnosis of myocardial infarction was revised to unstable angina.

There is a greatly increased incidence of ischaemic heart disease in familial hypercholesterolaemia.[3] Many patients with this condition are now treated with an HMG CoA reductase inhibitor such as simvastatin. Skeletal myopathy is a rare complication of treatment, although subclinical myopathy with a rise in serum CK activity is commoner.[4] It follows that there is a small but significant possibility of misdiagnosis of myocardial infarction if a patient on treatment with an HMG CoA reductase inhibitor presents with non-infarcting ischaemic chest pain. The absence of ECG evidence of myocardial infarction in the presence of a very high serum CK activity is the initial characteristic feature of this presentation, and subsequent measurements will fail to demonstrate the expected fall in CK activity after 24 hours. Measurement of serum CK-MB[2] or cardiac troponin T[5] levels are valuable second line investigations. Above all, we believe that the clinician should be aware of this cause of misdiagnosis of myocardial infarction, especially when considering treatment with potentially hazardous thrombolytic therapy.

A.P. Day
D. Stansbie
Department of Chemical Pathology, Bristol Royal Infirmary, Bristol, BS2 8HW, UK.

References

1. Rapaport, E. Thrombolytic agents in acute myocardial infarction. *N Engl J Med* 1989, **320:** 861-863.
2. Moss, D.W., Henderson, A.R. & Kachmar, J.F. Enzymes. In: Tietz, N.W. (ed.) *Fundamentals of Clinical Chemistry*, 3rd edn. W.B. Saunders Co., Philadelphia, 1987, pp. 346-421.
3. Goldstein, J.L. & Brown, M.S. Familial hypercholesterolaemia. In: Stanbury, J.B., Wyngaarden, J.B., Frederickson, D.S., Goldstein, J.L. & Brown, M.S. (eds) *The Metabolic Basis of Inherited Disease*, 5th edn. McGraw Hill, New York, 1983, pp. 672-712.
4. Walker, J. Simvastatin: the clinical profile. *Am J Med* 1989, **87:** 44S-46S.
5. Mair, J., Artner-Dwarzak, E., Lechleitner, P. *et al.* Cardiac troponin T in diagnosis of acute myocardial infarction. *Clin Chem* 1991, **37:** 845-852.

Thrombophlebitis and Reiter's syndrome

Sir,

Seronegative asymmetric arthropathy, urethritis and conjunctivitis make up the characteristic triad that most commonly defines Reiter's syndrome.[1,2] Different complications have been reported, among them thrombophlebitis of leg deep veins.[3] However, the intimal-coagulation abnormality is unknown. We describe herein a patient with Reiter's syndrome who developed this complication in the acute stage of his disease and in whom a thorough coagulation study failed to find any abnormality.

A 29 year old man was admitted because of urethritis and polyarthritis of metatarsophalangeal joints. He was well until 5 days after an unprotected sexual contact with a prostitute when he experienced dysuria and a purulent urethral discharge. Three days later, he developed arthritis in the metatarsophalangeal joints of both feet and in the left knee. On physical examination there were slight conjuctival injection in both eyes, along with signs of arthritis in those joints. No stomatitis was present but a few painless, superficial erosions with surrounding erythema were evident on the glans penis.

Blood cell counts were normal and the ESR was 77 mm/hour. There were slight increases in transaminases and a polyclonal increase in gammaglobulins. Other biochemical parameters were normal including C_3 and C_4 measurement. The VDRL and the FTA-ABS tests were