Short reports

Raised concentration of plasma creatine kinase BB isoenzyme in myelodysplasia

M Crook, A Williams, A Sankaralingam, P Tutt

Abstract
A 72 year old woman presented with a suspected myocardial infarction. An echocardiograph showed no acute changes but her plasma creatine kinase (CK) activity was increased at 343 U/l (<175 normal range). The apparent creatine kinase-MB activity by a CK-M subunit immunoinhibition assay was 350 U/l. In view of the discrepancy between the total creatine kinase and CK-MB activity plasma creatine kinase electrophoresis studies were performed which showed not only a band of creatine kinase-MM but also a band of creatine kinase-BB, 53% of the total creatine kinase activity. No band of CK-MB was seen. It later transpired that the patient had been diagnosed before with myelodysplasia. This diagnosis had been made following a full blood count which showed a haemoglobin concentration of 140 g/l, a white cell count of 22.6 x 10^9/l (neutrophils 13.1 x 10^9/l lymphocytes 2.03 x 10^9/l monocytes 1.36 x 10^9/l, eosinophils 0.68 x 10^9/l basophils 0.90 x 10^9/l metamyelocytes 3.39 x 10^9/l and myelocytes 1.13 x 10^9/l and platelets 389 x 10^12/l. Furthermore, a bone marrow aspirate was hypercellular with an increase in myeloid cells but no excess of blast cells. A trephine biopsy specimen had confirmed hypercellularity, but there was no increase in reticulin. Cytogenetic studies showed no abnormality.

A few months later she was reviewed in the haematology outpatients department and a plasma total creatine kinase determination was 49 U/l but plasma creatine kinase electrophoretic studies again showed a high proportion of the CK-BB isoenzyme, this time being 56% of the total creatine kinase activity.

Methods
Plasma creatine kinase total activity and CK-MB (by immunoinhibition) were measured on a Kodak Ektachem analyser (Kodak, Welwyn, Herts, England). Blood counts were performed on a Sysmex E3000. Plasma creatine kinase electrophoresis was performed on agarose gels (Corning, Halstead, Essex, England) and the bands of creatine kinase activity visualised using total creatine kinase (NAC) and CK-MB (NAC) reagents from Merck (Poole, Dorset, England); the latter contained antibodies to the CK-M subunit. Quantitation of the fluorescent bands of enzyme activity was performed using a Corning (Halstead, Essex, England) densitometer.
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Discussion
The CK-MB determination on the Ektachem uses an immunoinhibition method. Here an antibody to the CK-M subunit inhibits its activity in both CK-MM and CK-MB isoenzymes. Residual creatine kinase activity is then multiplied by 2 to give the CK-MB activity, the assumption being that there is negligible CK-BB activity present and that the M and B subunits have the same enzyme activity. The spuriously high “apparent” plasma CK-MB activity alerted us to the possibility of the presence of CK-BB in the sample.

We suggest that the explanation for this woman’s increased plasma CK-BB is her myelodysplasia which is considered a premalignant haematological condition. In a review by Griffiths CK-BB was reported to be increased in several malignant conditions. Another study by Rubery and co-workers found that CK-BB was raised in about 34% of patients with malignant disease while Abbott and Lott found raised serum CK-BB in patients with acute myeloid leukaemia and chronic lymphocytic leukaemia. Changes in cell differentiation seem to result in the increased production of CK-BB which could possibly be useful as a tumour marker.

In conclusion, we report increased total plasma creatine kinase in a woman with myelodysplasia, which was mainly the CK-BB isoenzyme. Spuriously high apparent CK-MB activities, using an immunoinhibition method, should alert biochemistry staff to the possible presence of non-CK-M activity. Premalignant and malignant haematological conditions should be considered in patients with an unexplained increase in plasma CK-BB.

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