CASE REPORT

MultivesSEL coronary thrombosis, acute myocardial infarction, and no reflow in a patient with essential thrombocythaemia

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Abstract

Essential thrombocythaemia (ET) has been reported rarely to cause coronary thrombosis, but the management is still undefined. A 63 year old woman with multivesSEL coronary thrombosis, acute myocardial infarction (MI), and no reflow in reperfused coronary artery in association with ET is presented. The patient's platelet count was only moderately raised at the onset of MI, but peripheral blood smear and bone marrow evaluation revealed clumping giant platelets and numerous large hyperplloid megakaryocytes. The patient was initially treated using aspirin and ticlopi dine, but caused coronary dissection. Then 3.5 × 18 mm gfx stent (Artereal Vascular Engineering, CA, USA) was successfully implanted. The occluded lesion improved significantly, but the perfusion delay in the LAD remained (no reflow, fig 1C). After these procedures, repeat angiography showed disappearance of the filling defect in the right coronary artery. Before the procedure, the patient received aspirin (81 mg) and ticlopidine (100 mg). Heparin (1 × 10^4 IU/day) and low molecular weight dextran (100 ml/h) were administered during and after the procedure. Peak serum creatine kinase was 4760 IU with an MB isozyme level of 483 IU.

The platelet count rose to a maximum of 113.8 × 10^4/mm^3 two weeks after admission, without an initial drop. Oral busulfan was administered and then the platelet count fell to 47 × 10^4/mm^3 two weeks after admission. Peripheral blood smear evaluation revealed clumping giant platelets with heterogeneous morphology. Bone marrow examination showed numerous large hyperplloid megakaryocytes. Normal myeloipoiesis and erythropoiesis were present. Cytogenetics were normal and no Philadelphia chromosome was detected.

At repeated angiography one month after the procedure, there were no thrombus-like filling defects or significant athlosclerotic lesions in the coronary arteries, and the coronary flow in...
LAD was not slow (fig 1D and E). Left ventriculography showed anteroapical hypokinesis with left ventricular ejection fraction of 68%. She was discharged, without chest pain, with a platelet count of 60.6 × 10⁴/mm³ and on aspirin (81 mg/day), ticlopidine (200 mg/day), and busulfan (2 mg/day). She developed no major bleeding complications.

Discussion

ET has clinical tendencies to both haemorrhage and thrombosis. However, coronary thrombosis and acute MI have been observed rarely in ET compared with other myeloproliferative disorders (for example, polycythaemia vera).5 Several theories have been postulated for the cause of coronary thrombosis from thrombocytosis. These are: abnormal activation of the fibrinolytic system, enhanced platelet procoagulant activity, and increased plasma viscosity.3 4 Previous studies showed that platelet size, rather than the absolute platelet count, is correlated with thrombotic complications.7 In our patient, peripheral blood smear evaluation revealed clumping giant platelets although the initial platelet count was only moderately raised. Fagher et al described patients with acute MI who had an initial drop in platelet count followed by an increased and even thrombocytosis.8 They reported that in 25% of acute MI patients the platelet count increased by almost 70% three weeks after the onset. The transient rise of the platelet count in our patient after admission might be reactive, but there was no initial drop and the rise was over 100%. Therefore, it is possible that the platelets were more haemostatically active at the onset of coronary thrombosis.

The aggressive inhibition of platelet aggregation and production may play an important role in treating ET associated ischaemic syndromes with coronary thrombosis. Michaels et al used a glycoprotein IIb/IIIa receptor inhibitor, abciximab, for acute MI due to ET.4 However, the use of antiplatelet agents in these patients has been suspected of inducing haemorrhage rather than preventing thrombosis. Rossi et al showed that a low dose of aspirin can reduce the coronary thrombosis without increasing bleeding complications.5 Our patient did not develop major haemorrhagic complications during treatment with aspirin and ticlopidine, probably because of the low dosage adopted. There are also recent data from randomised clinical trials showing hydroxyurea to be effective in preventing ET related thrombotic complications by keeping the platelet count < 60 × 10⁴/mm³.9 In our patient, busulfan was used to reduce platelet production.
Our patient also had no reflow phenomenon after recanalisation of the LAD. Several mechanisms have been advocated for the no reflow phenomenon in animal models, and a major candidate is direct ischaemic microvascular injury. Our patient had acute MI with angiographically normal coronary arteries. The obstruction of LAD does not appear to be due to the rupture of atherosclerotic plaque, but is likely to be transient thrombosis. Therefore, it is possible that multiple microvascular thrombi contributed, at least in part, to the no reflow phenomenon in our patient.

In conclusion, we recommend long term prophylaxis with antiplatelet agents in patients with ET, even if the platelet count is only moderately raised. Cytoreductive treatment in combination with antiplatelet agents may be effective for the second prevention when thrombotic complications occur.


