Acute Myocardial Infarction in a Patient With Essential Thrombocytemia

Successful Treatment With Percutaneous Transluminal Coronary Recanalization

Einosuke Mizuta, MD; Shin-ichi Takeda, MD*; Norihito Sasaki, MD*; Junichiro Miake, MD; Toshiroo Hamada, MD*; Masaki Shimoyama, MD*; Fumihito Tajima, MD**; Osamu Igawa, MD*; Chiaki Shigemasa, MD*; Ichiro Hisatome, MD

A 65-year-old woman with essential thrombocythemia (ET) was admitted to hospital where she was diagnosed as acute myocardial infarction (AMI). Because of abundant thrombus of right coronary arteries, percutaneous transluminal coronary recanalization by administration of urokinase was selected as the reperfusion therapy, resulting in successful revascularization with Thrombolysis in Myocardial Infarction grade III coronary flow. The maximum creatine kinase reached 507 IU/L, and left ventriculography performed at 1 month after initiation of both anticoagulant and antiplatelet therapies revealed reduced motion in the inferior wall with an ejection fraction of 57%. Despite good recovery of left ventricular function, bleeding complications, such as epistaxis or ecchymoma, which did not require blood transfusion, occurred during the clinical course. Because ET causes not only thrombus formation but also bleeding tendency, it is very important to carefully follow-up any clotting abnormality in AMI patients with ET. (Circ J 2005; 69: 1000–1002)

Key Words: Acute myocardial infarction; Essential thrombocythemia; Percutaneous transluminal coronary recanalization

Essential thrombocythemia (ET) is a clonal myeloproliferative disorder of unknown origin, characterized by a persistent increase in the platelet count. In the past, ET was described as hemorrhagic thrombocytosis1–3 but recent studies have reported that thromboembolic complications, especially thrombus in the cerebral, coronary, and peripheral arteries, were more frequent than hemorrhages in patient with ET.4,5 Pulmonary embolism and deep-vein thrombosis were less frequent than arterial thrombus.6 The incidence of acute coronary disease in patients with ET was reported as 9.4% and that of acute myocardial infarction (AMI) as more frequent in patients older than 40 years. Smoking and hypertension are risk factors of arterial ischemic complications, including AMI, in patients with ET.

Several agents are now available for initial treatment of ET to prevent thrombus.8 There is evidence that hydroxyurea, which controls platelets, is effective in preventing thrombus in ET patients at high risk for systemic artery disease.9 Low-dose aspirin therapy (100 mg/day) reduces coronary thrombosis without increasing bleeding complications in patients with elevated platelet count and common atherosclerotic risk factors.9 There are many reports about the efficacy of drug therapy for preventing coronary artery disease in patients with ET, but no guidelines to treat AMI in such patients, as ET causes the high-risk complications of thrombosis, as well as hemorrhage, during acute treatment of AMI.

We present a case of ET accompanied by AMI which was characterized by both coronary artery thrombus and bleeding during the clinical course.

Case Report

A 65-year-old woman visited the Emergency Department of Tottori University Hospital because of severe chest pain lasting for 3 h. She was a smoker and had a past history of hypertension, chronic renal failure, and ET, but did not have a family history of heart disease or hemorrhagic disorder. She had been already treated with hydroxyurea and ticlopidine because of the previous diagnosis of ET when her platelet count was 1,500,000/mm3. The vital signs on admission were as follows: blood pressure, 140/76 mmHg; pulse rate, 64 beats/min, regular; temperature, 35.4°C. Laboratory results were as follows: white blood cell count, 13,400/mm³; hemoglobin, 12.3 g/dl;
platelet count, 469,000/mm³; serum sodium, 143 mmol/L; serum potassium, 4.1 mmol/L; chlorine, 102 mmol/L; blood urea nitrogen 29 mg/dl; serum creatinine, 1.61 mg/dl; serum glutamate oxaloacetate transaminase, 151 IU/L; serum glutamate pyruvate transaminase, 18 IU/L; serum lactate dehydrogenase, 458 IU/L; serum creatine kinase (CK), 173 IU/L; CK-MB, 171 IU/L; blood sugar, 144 mg/dl; C reactive protein, 0.08 mg/dl; serum troponin T levels were high. No pulmonary congestion was observed on chest X-ray. Electrocardiography revealed heart rate, 52 beats/min, sinus rhythm; ST segment elevation in leads II and III, and augmented voltage foot (aVF); poor R progression in leads III and aVF. Echocardiography showed that the inferior wall motion was severely reduced. These findings were compatible with AMI accompanied by ET, so intravenous heparin (3,000 U), intravenous and oral nitroglycerin (2.5 mg), and oral aspirin (200 mg) were initiated and emergency coronary catheterization was performed. When she arrived at the catheter laboratory, her chest pain had entirely disappeared, and the ST segment elevation had recovered and an inverted T wave appeared in leads II, III, and aVf. Coronary angiography (CAG) revealed that the mid region of the right coronary artery (RCA) was obstructed by abundant thrombus with Thrombolysis in Myocardial Infarction (TIMI) grade II flow (Fig 1). Because the thrombus was mainly composed of platelet and fibrin¹⁰ percutaneous transluminal coronary recanalization (PTCR) with intra-right coronary injection of urokinase (240,000 units) was selected as the early reperfusion therapy, resulting in improving RCA flow from TIMI grade II to TIMI grade III.

Subsequently, she was treated with anticoagulant and antiplatelet drugs, such as heparin, aspirin, ticlopidine, and warfarin, to prevent the formation of new thrombus and to dissolve the existing multiple thrombi. Hydroxyurea was continued to control the platelet count. As a result, bleeding time was prolonged to 8 min and the platelet aggregation test showed that 10⁸/ml/L ADP-induced secondary aggregation was inhibited. The maximum concentration of CK was 507 IU/L at 24 h after the onset of chest pain, and no remarkable complications did not occur during her clinical course other than ecchymoma at the puncture site of the right femoral artery, which did not appear until the day after angiography. Epistaxis occurred frequently, but was stopped by compression of the nose for a few minutes. The size of the ecchymoma did not increase and vascular echo study showed extravasation of blood had already stopped. These complications did not cause a severe anemia that required blood transfusion. Von Willebrand factor is known to play crucial role in the onset of arterial thrombosis¹¹–¹⁵ and her plasma von Willebrand factor was high at 184%.

One month later, the ecchymoma had disappeared, and so a treadmill test and repeat CAG were performed. The treadmill test showed no ischemic change under sufficient load. Although there was still moderate stenosis in the mid RCA (Fig 2), CAG revealed that blood flow had dramatically improved and the thrombus had disappeared as expected. Left ventriculography showed that inferior wall motion had improved. Her platelet count was kept stable at approximately 600,000/mm³.

Discussion

ET is a myeloproliferative disorder characterized by abnormal proliferation of megakaryocytes and it causes thrombus formation in systemic arteries including the coronary arteries. In AMI associated with ET, the affected coronary artery is often occluded with a large amount of thrombus, thus a careful therapeutic strategy is required for successful revascularization. We report here that PTCR followed by sufficient antiplatelet and anticoagulant therapy resulted in a good clinical course of AMI in a patient with ET.

We selected PTCR for the early reperfusion therapy rather than balloon angioplasty because the occlusion was caused by abundant thrombus. In addition, we thought that the peripheral blood circulation might recover spontaneously when cardiac catheterization began, because by that time her chest pain had disappeared with recovery of ST segment elevation.
We consider that it is rational to carefully monitor the inhibition of platelet aggregation in patient with ET because the complications of ET are not only thrombus but also hemorrhage. Regarding the anticoagulation therapy for patient with ET, we treated with aspirin, ticlopidine and warfarin and the patient’s bleeding time was increased to 8 min and ADP-induced secondary aggregation was inhibited.

Moreover, we treated with hydroxyurea to decrease the platelet count. Platelets are a major source of von Willebrand factor, which is known to play a major role in the onset of coronary arterial thrombosis. In the present patient, von Willebrand factor was high (184%), emphasizing the need to control the platelet count.

It is also important to control the risk factors for preventing ischemic heart disease in patients with ET, especially arterial blood pressure and cigarette smoking, because a previous study has shown that they have strong relation with the thromboembolic complications of ET. The present patient was a habitual smoker and had a past history of hypertension, so we persuaded her to quit smoking immediately and monitored her blood pressure very carefully.

In summary, a case of ET had a good clinical course after AMI treated by PTCR followed by anticoagulant and antiplatelet therapy. Careful follow-up of any clotting abnormality is necessary for AMI patients with ET.

References