Treatment of Intracoronary Thrombus Using Tirofiban in a Patient With Normal Coronary Arteries

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SUMMARY

We report a 64-year-old Turkish man who presented with unstable angina pectoris. Coronary angiography revealed massive intracoronary thrombus, which completely occluded the distal part of the left circumflex coronary artery. The thrombotic segment and the rest of the coronary tree were free of atherosclerosis. The patient was treated with intravenous tirofiban, a glycoprotein IIb/IIIa inhibitor. A control angiography was performed one week later and showed total dissolution of the thrombus with tirofiban therapy. (Jpn Heart J 2004; 45: 343-346)

Key words: Intracoronary thrombus, Tirofiban, Normal coronary arteries

INTRACORONARY thrombus (ICT) is rarely seen in normal coronary arteries. The pathophysiological mechanism remains unclear. We report a case who had ICT in normal coronary arteries and was successfully treated with tirofiban.

Case presentation: A 64-year-old man visited to our hospital with complaints of palpitations and chest pain-like angina. The patient said he fell down and remained unconscious for 3-5 minutes one week before coming to the hospital. Neurological and cardiac examinations were normal. Blood pressure and heart rate were 110/80 mmHg and 90 beats/minute respectively. Electrocardiography showed normal sinus rhythm and no ischemic changes. An electroencephalogram and 24-hour Holter monitoring were normal. Cardiac enzyme levels and other laboratory results were within normal ranges. The patient was diagnosed as having acute coronary syndrome (ACS) and a transient ischemic attack. He was given acetylsalicylic acid, a $\beta$-blocker, a statin (PO), nitroglycerin (IV), and low-molecular weight heparin (SC).

Coronary angiography performed two days later revealed a massive ICT, which completely occluded the distal part of the left circumflex coronary artery (Figure 1). A significant coronary artery spasm was also observed at the mid seg-
ment of the left circumflex coronary artery. The thrombotic segment and other coronary tree were free of atherosclerosis. Due to the inappropriate coronary structure and length of the thrombus, percutaneous coronary intervention was not performed. The patient was treated with tirofiban (0.4 $\mu$g/kg/min bolus) over 30 minutes followed by 0.1 $\mu$g/kg/min for 24 hours. A control coronary angiogram obtained one week later showed total dissolution of the coronary thrombus and normal clearance of the culprit vessel (Figure 2).

The patient experienced atrial fibrillation 3 months later; no thrombus was detected with transesophageal echocardiography. DC cardioversion was then performed, but normal sinus rhythm could not be obtained.

**DISCUSSION**

ICT may occur in ACS spontaneously, sometimes as a complication of interventional procedures and frequently superimposes on an existing atherosclerotic lesion.\(^1\)\(^-\)\(^6\) However, an ICT or myocardial infarction with normal coronary arteries is a syndrome resulting from numerous conditions, but the exact cause in a majority of patients remains unknown. Possible mechanisms include embolism, vasospasm, nonatherosclerotic coronary diseases, hypercoagulable states, trauma, an imbalance between oxygen demand and supply, intense sympathetic stimulation, and endothelial dysfunction.\(^7\)
There have been a few reports of ICT and coronary spasm without any atherosclerotic lesion. The patients had no history of use of any drug, connective tissue disease, cardiomyopathy, or valvular disease. In our case, the spasm was demonstrated to be due to a thrombus occluding the left circumflex artery and its side branch without any atherosclerotic lesion. A control angiogram revealed the coronary spasm and thrombus had disappeared, and total coronary clearance and no stenosis or plaque formation. A possible mechanism of the thrombus formation may be spasm-induced coronary stagnation and the stimulatory effect of blood streaming endothelial injury for the platelet aggregation and adhesion. ICT can occur either as a result of coronary spasm, or it may cause the development of coronary spasm. Another possible mechanism is thromboembolism from the left atrium and left atrial appendage. Coronary embolism is an infrequent clinical situation that comprises one of the nonatherosclerotic causes of ACS. We speculated that the ICT and transient ischemic attack that occurred in our patient were due to thromboembolism which formed as a result of paroxysmal atrial fibrillation. However, we did not detect any thrombus in the transeosophageal echocardiography performed during atrial fibrillation.

Both platelets and thrombin play an essential role in the pathophysiological mechanism of ACS. Although aspirin and heparin have been used as therapeutic mainstays for ACS, the activation of platelets is not always inhibited by aspirin or heparin.9) The final common pathway to the coronary thrombosis underlying ACS involves the aggregation of platelets mediated by the binding of soluble fibrinogen to the platelet receptor glycoprotein (GP) IIb-IIIa.9) The first and most important field of use of GP IIb/IIIa inhibitors is during percutaneous interventional treatment. The PRISM-PLUS study suggests that the combination of tirofiban plus heparin reduces the ICT burden of the culprit lesions, improves the perfusion grade, and decreases the severity of the obstruction in patients with ACS.8)

There is no certain algorithm available for the therapeutic options of ICT. The conventional approach to coronary thrombosis is either an interventional procedure or bypass surgery. Different medical approaches have been suggested.10-12) It is believed anticoagulant and antiplatelet medications contribute to clot dissolution. Spontaneous reperfusion has been reported in 11% of patients within 4 hours of the clinical onset, reaching 35% at 12 to 24 hours after onset of myocardial infarction due to an endogenous fibrinolytic process.13) Endogenous fibrinolytic activity continues afterwards in all patients with ACS. We did not use a thrombolytic agent since there were no findings of myocardial infarction. Percutaneous coronary intervention was not performed due to the inappropriate coronary structure and length of the thrombus. The patient was treated with tirofiban because its efficacy can be controlled and it is safer. GP IIb/IIIa inhibitors,
although not having primary fibrinolytic activity, can be helpful for dissolving coronary thrombus by assisting spontaneous fibrinolysis. The contribution of GP IIb/IIIa inhibitor therapy to clot dissolution remains unclear. However, we believe that tirofiban had a beneficial effect on coronary clot dissolution in our case.

**Conclusion:** Patients with ICT who are unsuitable for interventional treatment may be treated with a GP IIb/IIIa inhibitor. This treatment can help to dissolve the thrombus.

**REFERENCES**