Global Myocardial Ischemia as a Complication of an Acute Type A Aortic Dissection

Rapid Diagnosis of a Case by Transesophageal Echocardiography

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A 36-year-old female was admitted for severe chest pain followed by profound shock. Electrocardiography showed severe ST segment depression (0.5-0.7 mV) in all leads except aVR and aVL. Echocardiography revealed an intimal flap in the ascending aorta and coexisting grade 3 aortic regurgitation. She was immediately intubated and transferred to the intensive care unit. Transesophageal echocardiography (TEE) demonstrated an intimal tear at 2 cm above the sinotubular junction, and the ostium of the left main trunk was oppressed by the intimal flap during diastole. Emergency graft replacement of the ascending aorta and aortic hemiarch concomitant with aortic valve resuspension was performed successfully. The ECG changes reversed to normal immediately after the operation. The patient was extubated 2 days postoperatively and discharged from the hospital 14 days postoperatively. TEE is useful for the rapid evaluation of coronary malperfusion as a complication of acute aortic dissection, especially in patients with hemodynamic instability. (Jpn Cir J 2000; 64: 533–536)

Key Words: Acute aortic dissection; Coronary malperfusion; Shock; Transesophageal echocardiography

Malperfusion of the coronary arteries in acute aortic dissection (AAD) is particularly problematic because it may be disguised by myocardial ischemia or infarction. The incidence of coronary malperfusion due to AAD is reported to be 1–9%.7–16 Although AAD is life-threatening, patients with coronary malperfusion are often misdiagnosed and treated as a coronary emergency. Transesophageal echocardiography (TEE) is one of the most sensitive and specific modalities for diagnosing AAD and we use it routinely for diagnosis of AAD in the emergency setting7–9 because it can show not only the intimal flap or entry tear, but also the coronary malperfusion.10

Case Report

A 36-year-old female was referred to the emergency department because of the sudden onset of back pain followed by profound shock. She had no medical history. Her father had died of AAD and her younger brother had had graft replacement of the descending thoracic aorta because of chronic aortic dissection. All these family members had non-Marfan physique. The patient was already on 10 μg·kg⁻¹·min⁻¹ of dopamine infusion for shock and her systolic blood pressure on arrival was approximately 60 mmHg. She was drowsy and had severe hypoxia (PaO₂ 48 mmHg) despite administration of oxygen. ECG showed marked ST segment depression in almost all leads except aVR and aVL (Fig 1). Transthoracic echocardiography showed an intimal flap in the ascending aorta associated with grade 3 aortic regurgitation (AR). The wall motion of the left ventricle was generally hypokinetic (left ventricular ejection fraction = 40%). There was no pericardial effusion and Doppler examination demonstrated an elevation of the right ventricular pressure (62 mmHg). The patient was immediately intubated and examined under computed tomographic (CT) scan, which also demonstrated the intimal flap extending from the ascending aorta to the bilateral iliac arteries. Thereafter she was admitted to intensive care unit (ICU) and underwent TEE examination, which showed the continuity between the true lumen and bilateral coronary ostia. However, there was an obvious narrowing of the true lumen because of compressed by the false lumen during diastole (Fig 2) and we considered this to be the reason for the global myocardial ischemia in the patient. Emergency surgical intervention was indicated without prior coronary angiography (CAG).

The operation started 1 h after admission and 4 h after the onset. Cardiopulmonary bypass (CPB) consisted of right atrial venous drainage and right femoral arterial return. After aortic clamping, the cardioplegic solution was directly perfused into the bilateral coronary ostia. The entry tear was found in the posterior wall of the ascending aorta 2 cm above the sinotubular junction and the bilateral coronary arteries originated from the true lumen. The aortic valve was detached from the dissection and resuspended at 3 commissures by mattress sutures with 5-0 Prolene reinforced with a Teflon pledget. The aorta was transected at

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Fig 1. Preoperative electrocardiography showing the depressed ST-segment in almost all leads.

Fig 2. Preoperative transesophageal echocardiography (transverse view). (Left) Systole; (Right) diastole. The ascending aorta was dissected by the intimal flap (*), the left main coronary artery (arrow) was connected to the true lumen (T) that was obviously compressed by the false lumen (F) during diastole.
the level of the sinotubular junction and a collagen impregnated graft (Hemashield®; Boston Scientific, Inc, Natick, MA, USA) was anastomosed using a 4-0 Prolene running suture with a reinforcing Teflon-felt strip externally after fixation of the dissecting layer with GRF glue. Distal anastomosis of a proportion of the hemiarch was performed in the same fashion under hypothermic circulatory arrest (rectal temperature 22°C). The aorta was then unclamped and CPB restarted, changing the arterial return from the femoral artery to the prosthetic graft. Weaning from CPB and hemostasis was uneventful. The times for the operation, CPB, aortic clamping and circulatory arrest were 273, 190, 66 and 24 min, respectively. Postoperative TEE revealed grade I AR, improvement of the left ventricular wall motion and almost negligible flow in the false lumen of the aortic arch and descending aorta. ECG performed just after the operation showed recovery of the ST segment to almost normal (Fig 3). The postoperative peak creatine kinase-MB was 48 IU/ml.

The patient woke at 4 h and was extubated at 36 h postoperatively. She was discharged from hospital 14 days postoperatively and is doing well at 8 months since the operation.

**Discussion**

Malperfusion of the coronary arteries in AAD is particularly problematic because the acute dissection may be disguised by myocardial ischemia or infarction. The incidence of coronary malperfusion in AAD has been reported to be 1–9.5%.1–6 From our experience in the decade 1989–1998, coronary malperfusion was detected in 8.9% of patients (11 of 123 patients) with AAD.

Although AAD is life-threatening, patients with coronary malperfusion are often misdiagnosed and treated as coronary emergencies. Thrombolytic therapy in the patient with AAD may cause catastrophic hemorrhage.10–13 One review of 21 cases of AAD in which thrombolysis was used inadvertently reported an early mortality rate of 71%, with hemorrhagic tamponade as the cause of death in most instances.4

A variety of mechanisms are involved in the genesis of coronary ischemia due to AAD, the most important reasons being (1) disruption of the coronary ostium, (2) direct extension of the intimal flap into the coronary system, and (3) compression of coronary ostium by the false lumen.5,8,10,15–17 Coronary angiography will usually only demonstrate the first situation and it is time consuming to perform CAG in an emergency case. In our department, once a patient is suspected to have AAD, we routinely transfer the patient to the ICU and perform TEE immediately under strictly controlled hemodynamic conditions.7–9 If the patient’s hemodynamic condition is unstable, the
transfer is to operating room, and TEE is performed under anesthesia.

We use TEE because it can show not only the existence of aortic dissection, but also cardiac complications such as AR, cardiac tamponade, ventricular dysfunction and coronary involvement, and because it is a rapid and highly reliable technique that can be easily done by the bedside. TEE is also useful for the monitoring of malperfusion during surgery, especially after the establishment of CPB.8,18,19 A phenomenon that we reported as occurring during CPB in approximately 13.6% of cases with AAD.20 If malperfusion is detected during CPB, the site of arterial inflow (usually the femoral artery) should be changed immediately to the axillary artery or another site to obtain sufficient flow in the true lumen.21

There are 3 methods of surgical management of acute coronary malperfusion associated with aortic dissection. (1) Coronary artery bypass grafting is indicated in patients with an intimal flap that continues into the coronary arteries or disrupts the coronary ostium.2,6,16,22 (2) Aortic root replacement may be required in patients with coexisting anululaoaortic ectasia or an aortic valve lesion.15,23 (3) In patients with the complication of compression of the coronary ostium by the false lumen, prosthetic graft replacement of the ascending aorta will resolve the problem, as described in the present case. Rapid evaluation of the pathophysiology using TEE and immediate surgical intervention contributed to the overall prognosis.

References