he incidence of ischemic heart disease (IHD) in women before menopause, especially in young women aged less than 30 years, is low because of the apparent protective effect of estrogen. In previous reports of acute myocardial infarction in young Japanese females, there is predominantly a non-atherosclerotic etiology, such as Takayasu aortitis, Kawasaki disease, or systemic lupus erythematosus (SLE). The present case of IHD occurred in a young Japanese female who did not have a history of underlying diseases that induce IHD and who had a peculiar configuration of the coronary artery, suggesting some kind of arteritis with a coronary anomaly as its etiology. She was successfully treated with percutaneous transluminal angioplasty (PTCA).

Case Report

A 25-year-old female complaining chest oppression and palpitation was admitted to hospital under the presumptive diagnosis of ischemic heart disease (IHD), although no obvious underlying disease associated with IHD was detected. Coronary angiography showed stenosis at the proximal site of left anterior descending artery (LAD), with dilatation and tortuosity at the bifurcation of the first and the second septal branches. Intravascular ultrasound imaging of the LAD showed intimal thickness without calcification at the site of stenosis. The stenosis was successfully and smoothly dilated by percutaneous transluminal angioplasty. Even with precise evaluation, the cause of the coronary artery disease in this young female patient was not clarified. Further careful follow-up is needed. (Jpn Circ J 2001; 65: 465–467)

Key Words: Coronary dysplasia; Kawasaki disease; Percutaneous transluminal angioplasty; Unstable angina pectoris
The body was U/ml, anti-mal coagulopathy was not observed. Anti-cardiolipin antibodies were indicative of connective tissue disease, and lipoprotein(a) was 16.9 mg/dl. There were no serological findings. Total cholesterol was 34 mg/dl, triglyceride was 68 mg/dl, and the cardiothoracic ratio was 38%.

After inhospital treatment with atenolol (25 mg/day), the palpitations and chest oppression completely disappeared, even while walking. No significant abnormalities were observed on echocardiography. Although the presumptive diagnosis was IHD, dipyridamole stress Tl scintigraphy was carried out to confirm this because the incidence of IHD is rare in a young female without underlying disease. There was a clear up-take defect in the anteroseptal-apex area on the initial stress images and a partial redistribution on the delayed images of this area, which strongly indicated myocardial ischemia associated with the left anterior descending coronary artery (LAD). Therefore, coronary angiography (CAG), left ventriculography (LVG) and aortography were performed. Calcification was not observed on fluoroscopy. The right coronary arteriogram was normal, but on the left coronary arteriogram, there was 99% stenosis at the proximal site of LAD with delayed contrast filling of the branch distal to the stenosis (Fig 2). In addition, the coronary artery was dilated and tortuous at the bifurcation of the first and the second septal branches of the LAD (Fig 2). LVG detected slightly reduced wall motion at the LV apex and the LV ejection fraction was 67%. The ascending aorta, aortic arch and major arterial branches from the aortic arch were normal without dilatation and stenosis. After anti-coagulation therapy for 3 weeks, CAG was performed again. Although the stenotic lesion at the proximal site of the LAD had improved a little, the residual stenosis was 90%. Before PTCA was performed, intravascular ultrasound imaging (IVUS) at the site of the coronary stenosis (Fig 3A) showed slightly eccentric intimal thickness, which was absent in the dilated region (Fig 3B). Calcification was not detected anywhere from the ostium to the mid-portion of the LAD. PTCA was performed using a 3.0-mm diameter balloon, which was inflated 3 times at 6 atm for 60 s. The stenosis was successfully and smoothly dilated. As the residual stenosis was 25% (Fig 4), coronary stent was not implanted. One week after PTCA, she was discharged without symptoms. Repeat CAG performed at 6- and 12-months later did not show re-stenosis at the inflated site.

Discussion

The incidence of IHD in young female is reported to be low; the incidence of acute myocardial infarction (AMI) in this group is 2–4% of all AMI patients. Previous studies have reported that a non-atherosclerotic etiology more frequently causes AMI in young Japanese females and this would appear to be the case for the present patient. However, Takayasu aortitis, SLE and other underlying diseases were excluded by the physical findings, laboratory data and angiographic findings. The long-term clinical sequelae of Kawasaki disease are coronary artery lesions that develop aneurysms, thrombotic occlusion and progress to IHD. Most patients with Kawasaki disease with cardiac sequelae are diagnosed in their infancy or childhood, but some have reported patients with Kawasaki disease in their teens and they have noticeable calcification in their coronary artery. In adult cases, the characteristic chest X-ray finding is ring calcification. The angiographic findings are aneurysms, occlusion, dilatation and stenosis of the coronary artery and prominent collateral vessels. However, in the present case, neither chest X-ray nor fluoroscopy detected calcification, nor did IVUS of the ostium to the mid-LAD. Although the thickened intima seen on the IVUS images is compatible with Kawasaki disease, this stenosis was easily and successfully dilated by PTCA, which indicates that this stenosis was relatively soft. These findings are not consistent with a previous
report by Ino et al who suggested that PTCA should be attempted within 6–8 years of the onset of the disease, because the intimal thickening and coronary calcification develop gradually over a long period after the onset of the disease. Taking all of these features into consideration, the present case of coronary artery disease is inconsistent with Kawasaki disease as the causative agent.

In addition to the absence of a calcified lesion, the partial dilatation and tortuosity of the LAD at the bifurcation of the first and the second septal branches was unusual. These anatomical abnormalities might arise from some kind of anomaly associated with arteritis and although etiology of this tortuous LAD is unknown, it may have influenced the coronary stenosis and partial dilatation.

In conclusion, a young Japanese female with IHD was successfully treated with PTCA. This case has an apparent non-atherosclerotic etiology without calcification, confirmed by IVUS, although Kawasaki disease was not completely excluded.

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References