Aortic dissection is defined as longitudinal cleavage of the aortic media layer by a dissecting column of blood. If untreated, it is associated with mortality rates of 1-2% per hour for the first 24-48 hours, 33% within 24 hours, 50% within 48 hours, 75% within the first 2 weeks and 90% at 1 year. Factors known to predispose to aortic dissection include hypertension, inherited arteriopathies often characterized by cystic medial necrosis, trauma, pre-existent aortic aneurysm, a bicuspid aortic valve, aortic coarctation and various vasculites. A less known cause of aortic dissection is cocaine use. Cocaine, particularly crack cocaine, may have a significant role in precipitating aortic dissection in the young (age 41±8.8 years), predominantly black and hypertensive individuals.
CASE REPORT

A 26-year-old male patient was admitted to the emergency service with acute chest pain. He had increased blood pressure and the history of antihypertensive drug use. The chest, lung, and cardiac physical examinations were reported as normal; and the remainder of the physical examination was unremarkable. He explained that he was an addict for more than 6 years. In addition, he recently used cocaine prior to aortic dissection, which was defined by cocaine consumption within 12 hours prior to the onset of symptoms. Moreover, he had no other signs of dissection-related disease such as Marfan syndrome. His laboratory values were within normal limits. Electrocardiogram showed sinus rhythm and left ventricular hypertrophy with repolarization changes. A chest X-ray demonstrated a widened mediastinum with a prominent ascending aorta. Transthoracic echocardiography revealed aortic dilatation with the presence of a small intimal flap in the proximal ascending aorta (DeBakey’s Type II aortic dissection). Echocardiography demonstrated a severe aortic insufficiency and mild concentric left ventricular hypertrophy without other abnormalities. Magnetic resonance imaging showed a 6 cm sized aneurysm in the ascending aorta (Figures 1, 2).

The patient’s blood pressure was controlled with intravenous esmolol and nitroprusside. After obtaining his informed consent, the patient was taken into the operating room immediately. He had a Bentall procedure including replacement of ascending aorta and the aortic valve and also coronary re-implantation. Postoperative period was uneventful and he was discharged on a beta blocker on 6th day after the operation. Six months after the operation, he was still clinically stable.

DISCUSSION

Cardiovascular complications of cocaine abuse include myocardial ischemia and infarction, dysrhythmias, cardiomyopathies and aortic dissection. Although the literature regarding cocaine-related aortic dissection consists primarily of case descriptions, The International Registry for Aortic Dissec-
tension in cocaine-related dissections.\textsuperscript{3,8} Consistent with our findings, Hsue and colleagues observed a primarily younger patient cohort with a short onset of symptoms following cocaine consumption, and the mean interval between cocaine use and the onset of symptoms was over 12 hours.\textsuperscript{9} Similar to the patients with aortic dissection unrelated to cocaine use, pain is the most common symptom. Daniel et al. reported thoracic pain as a presenting symptom in 82% of the patients with cocaine-related aortic dissection.\textsuperscript{10} Although not as well described as in patients with aortic dissection unrelated to cocaine, aortic regurgitation may also serve as a complication. It occurs in 25-36% of patients with cocaine-related aortic dissection due to retrograde dissection into the aortic root causing dilation or into an aortic valve cusp causing leaflet dysfunction or flail.\textsuperscript{9,10}

When cocaine abuse is considered, aortic dissection is thought to relate first to an underlying process that has weakened the media of the aorta and, secondly, to the severe sheering forces that result from sudden and profound hypertension and tachycardia which accompany cocaine (particularly crack) use.\textsuperscript{4} The excessive adrenergic vasoconstriction caused by cocaine use could lead to such a sheer stress on the aorta’s intima resulting in small tears. Other possible mechanism is that chronic cocaine use itself leads to premature atherosclerosis, with weakening of the intima, although in our case atherosclerosis was absent. In our case, the aortic dissection seems to be related to the use of crack cocaine, and the underlying possible abnormality of the arterial wall can be affected by hypertension. Besides, aging of arterial wall seems unlikely in such a young person, and but it is conceivable that chronic cocaine consumption and resulting hypertension might have accelerated the progression of degenerative changes.

Consequently, when treating young cocaine users who present with sudden onset of chest pain, clinicians must have a high level of suspicion, and consider aortic dissection in the differential diagnosis.

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Conflict of Interest

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