The Editor,

A 27-year-old male professional bodybuilder presented with excruciating retrosternal chest pain, extending to his neck and shoulder. He gave a history of taking traditional medication (containing opium) for the pain that was self-prescribed but the pain did not subside with that treatment, and his general physician referred him for cardiac evaluation in the emergency department. He gave a history of long-term anabolic steroid usage and multiple intramuscular injections of human growth hormone in the recent past. There was no history of early coronary artery disease or sudden death in the family. On physical examination, he was well built and nourished with prominent muscles over pectoral girdle, hypertrophied pectoralis, biceps, triceps, rectus abdominis, and quadriceps muscles. In the electrocardiogram, Q waves and S-T elevation in lead V1–V6 were found. The creatine phosphokinase myocardial band and troponin levels were raised. The patient was referred to the coronary care unit and was treated with intravenous trinitroglycerin, aspirin, clopidogrel, statin, and heparin for acute coronary syndrome (ACS). The transthoracic echocardiogram showed reduced left ventricular systolic function (ejection fraction 40%) with apical hypokinesia. The cardiac troponins increased serially from 3 to 8 (units). Coronary angiography was performed which showed proximal to middle spiral dissection of the left anterior descending artery with severe distal stenosis. The left main right coronary and left circumflex arteries were normal [Figure 1]. The case was referred for surgery by interventional cardiologist as percutaneous coronary intervention was not preferred because of a long segment of dissection in artery, ongoing ischemia, and the presence of significant lesion distal to dissection. Individuals with a history of ACS but without evidence of ongoing ischemia and no significant stenosis on cardiac angiography may be managed with medical treatment. Medical treatment was not a choice in our patient due to ongoing ischemia.

Discussion

Although spontaneous coronary artery dissection (SCAD) is a very rare cause of ACS, few reported cases have occurred in young women.[1] First case report of SCAD was reported in a young pregnant female by Pretty in 1931; however, postmenopausal woman is not spared from this disease.[2] Apart from peripartum periods in women, the most important risk factor in men (as in our case) is intense physical activity and associated anabolic steroid abuse. The known conditions associated with SCAD include anatomic abnormalities such as kinking, atherosclerosis, connective tissue disease, systemic autoimmune disease, oral contraceptives, illicit drugs (cocaine and heroin use), and hypereosinophilic syndrome.[3] The combination of intense physical activity such as lifting weights heavier than the usual norm and the weakness of the arterial medial wall by anabolic steroid and acute hypertension could prepare a basis for the medial arterial wall to be separated while on the other hand, atherosclerosis, coronary vasospasm, and coronary artery dilatation and beading-like sequelae that are complications of anabolic steroid abuse are also believed as the main reasons for dissection.[4,5] Anabolic steroids are atherogenic, and atherosclerotic plaques may be ruptured by severe hypertension during weight lifting. The clinical presentation of SCAD is related to severity of coronary vessels involvement. It could be asymptomatic in patients with critical proximal stenosis and good retrograde filling by collateral vessels or it can present as subendocardial myocardial infarction, unstable angina, ventricular fibrillation, sudden death, or myocardial infarction as it did in our patient. The diagnostic imaging of choice for SCAD is coronary angiography that may reveal two parallel lumen, false and true in their course in the affected coronary artery. Seventy percent of female patients with SCAD have associated fibromuscular dysplasia (FMD) with complex angiographic findings such as stenosis, dilatation, and beading pattern; hence, these findings are more commonly detected in female patients with FMD. However, in the patients without FMD, the only finding on angiography may be the presence of atherosclerosis in other vessels.[6]

Conclusion

SCAD is an exceedingly rare cause of ACS in patients with anabolic steroid abuse. Although it is commonly reported in postpartum women with unknown risk factors, it has been seen in the middle-aged and older males with atherosclerotic coronary artery disease as well. Its occurrence in young males without any risk factors except anabolic steroid abuse has not been reported yet.
Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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