Chapter 31
Coronary Artery Aneurysm

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Abstract A 33-year-old man presented in our emergency department with diagnosis of inferior ST-segment elevation myocardial infarction. The past medical history was positive for smoking. He was a bodybuilder and regularly consumed anabolic steroids and human growth hormone (hGH) for the past two years. The angiogram of right coronary artery (RCA) showed totally thrombotic cut off at mid part. After RCA wiring, the angiogram revealed ectasia in proximal part, a large coronary aneurysm in mid part with huge thrombus burden in mid to distal part segment.

History and Clinical Presentation

A 33-year-old man presented in our emergency department with acute chest pain from 6 months ago. The past medical history was positive for smoking. He was a bodybuilder and regularly consumed anabolic steroids and human growth hormone (hGH) for the past 2 years.

Para-Clinic Assessment

The first electrocardiogram showed ST-segment elevation in inferior leads and ST-segment depression with T-wave inversion in lateral leads.

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Managements

The patient immediately underwent coronary angiography with a diagnosis of inferior ST-segment elevation myocardial infarction. The angiogram of the left coronary system was unremarkable, but the right coronary artery (RCA) injection showed a totally thrombotic cut off at mid part (Figs. 31.1 and 31.2). After RCA wiring, the angiogram revealed ectasia in the proximal part, a large coronary aneurysm in mid part with a huge thrombus burden in mid to distal part segment (Fig. 31.2). After several manual thrombectomies, acceptable TIMI flow was achieved. Although in the final injection there is some thrombus in the coronary aneurysm at the mid part of RCA and also some in the ostio-proximal part of the posterior descending artery (PDA) (Fig. 31.3). Then, the patient was treated with an infusion of Glycoprotein IIb/IIIa inhibitor and heparin. Five days later the patient was discharged with dual antiplatelet and high dose statin.

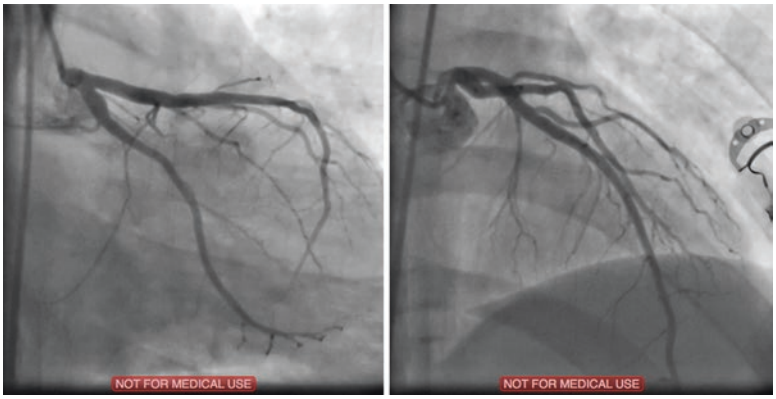

Fig. 31.1 The angiogram of left coronary system showed patient coronary arteries in RAO caudal (left figure) and AP cranial (right figure) view

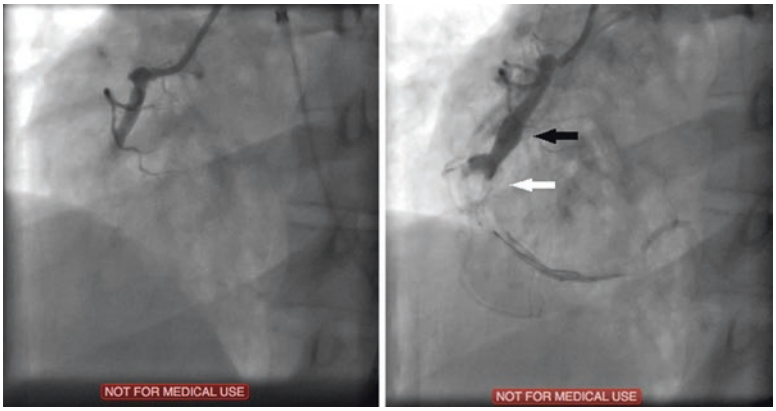

Fig. 31.2 The angiogram of RCA revealed totally thrombotic cut off at mid part (left figure). After wiring, the angiogram showed ectasia in proximal part (black arrow), a large coronary aneurysm (white arrow) in mid part with huge thrombus burden in mid to distal part segment (right figure)

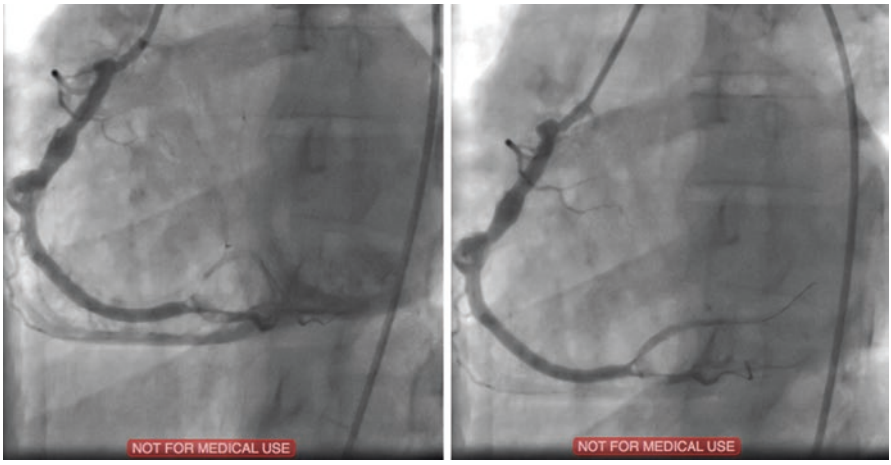

Fig. 31.3 After several manual thrombectomy, acceptable TIMI flow was achieved. The final injection showed some thrombus in the coronary aneurysm at the mid part of RCA and also some in the ostio-proximal part of posterior descending artery (PDA)

Conclusion

Aneurysmal dilation of coronary arteries is ranged from 0.3% to 5% among patients undergoing coronary angiography. Irrespective of the presence of concomitant atherosclerotic coronary artery disease, the presence of coronary aneurysm or ectasia has been associated with poor long-term outcomes. Clinical presentations range from incidental findings on cardiac imaging to acute coronary syndrome. There are criteria that distinguish between two phenotype types of coronary aneurysm and ectasia [1]. By these criteria, a focal dilation of coronary segments of at least 1.5 times the adjacent normal segment is described as a coronary aneurysm; whereas the term coronary ectasia is used to define similar, but more diffuse lesions where the length of the dilated segment is more than 50% of the diameter [1, 2].

Coronary aneurysms are most commonly found in the RCA (68%), followed by the proximal left anterior descending (60%) and left circumflex (50%). Aneurysms in the left main stem are exceedingly rare and occur in only 0.1% of the population [3]. Interestedly, the prevalence of coronary aneurysms is more in the men and also, in proximal than to distal segments of the coronary bed [4].

The most common cause of coronary aneurysms in adults is atherosclerosis, whereas Kawasaki disease is responsible for the majority of cases in children. Other etiologies such as inflammatory disorders, infectious, connective tissue disorders, drugs (cocaine and amphetamines), trauma, and iatrogenic are a less common cause of aneurysm formation [3]. Coronary aneurysms secondary to atherosclerosis or vasculitis usually affect more than one artery, whereas congenital and iatrogenic CAAs are typically confined to a single vessel [1].

Most coronary aneurysms are clinically silent and are only detected incidentally during coronary angiography or computed tomography. However, in some cases the clinical symptoms and presentation can develop due to one of the following reasons:

1. The presence of concomitant stenotic atherosclerotic disease can result in both effort angina or acute coronary syndrome.
2. Local thrombosis in the lumen of large aneurysms may lead to distal embolization and myocardial infarction.
3. Massive enlargement of some coronary aneurysms can result in compression of adjacent structures.
4. Stress-induced myocardial ischemia due to microvascular dysfunction has been documented even in the absence of significant coronary stenosis.
5. Myocardial ischemia due to the turbulence of flow and energy loss as blood passes through the coronary aneurysm in absent of significant coronary stenosis, especially in giant aneurysms.
6. Other complications such as sudden cardiac death, fistula formation, rupture, hemopericardium, tamponade are very rare [1, 3].

The most common imaging modality to assess ectatic or aneurysmal coronary arteries is coronary angiography. However, delayed antegrade contrast filling, segmental back flow, and contrast stasis in the dilated coronary segment often hamper optimal imaging during angiography [1, 4]. So, a forceful and prolonged contrast injection may be necessary to avoid misinterpreting slow aneurysmal filling as in situ thrombosis, especially in giant aneurysms. In some cases, intravascular ultrasound (IVUS) can be extremely helpful for better distinguishing of vessel wall structures (between true aneurysm, pseudoaneurysm) and also, selecting proper stent size if percutaneous coronary intervention is planned. Although, optical coherence tomography (OCT) in assessing coronary aneurysms is limited by the small scan diameter of the infrared light [1, 5]. Computed tomography is superior to invasive angiography for evaluation of coronary aneurysm size and degree of thrombus and calcification, particularly in those with giant aneurysm [6].

Our case was bodybuilder and used anabolic steroids and hGH, additional to smoking. Anabolic-androgenic steroids, including testosterone and its numerous derivatives that have been modified to improve anabolism, are usually used to boost protein synthesis, muscle growth, and erythropoiesis. The anabolic-androgenic steroids by a decrease in high-density lipoprotein, an increase in low-density lipoprotein, and an increase in homocysteine levels in the blood lead to increase the risk of Atherosclerosis development and cerebrovascular disease. Also, by direct effect on the coagulation/fibrinolysis system, can lead to an increased risk of thrombus formation. Other effects of anabolic-androgenic steroids on the cardiovascular system include hypertension, coronary spasm, myocardial apoptosis, cardiac hypertrophy, dilated cardiomyopathy, arrhythmia, and sudden cardiac arrest [7]. Additionally, hGH abuse contributes to increased heart rate and cardiac output, consequently leading to concentric ventricular hypertrophy and diastolic dysfunction. In some cases, hGH abuse may even promote ischemia/necrosis and heart failure associated with impairment of the systolic function [8].

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