CASE OF THE WEEK
LIFE WINS

TOPIC : SUPER-GIANT CORONARY ANEURYSMS AFTER KAWASAKI DISEASE

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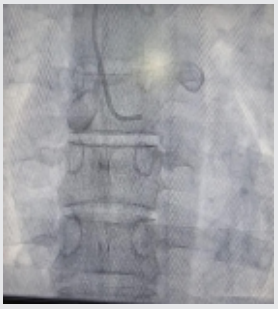

Fig - 1

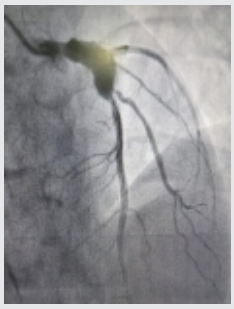

Fig - 2

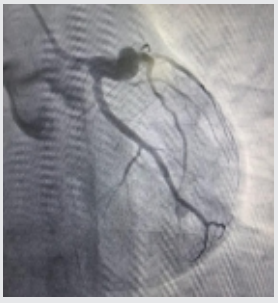

Fig - 3

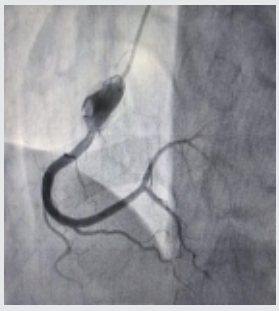

Fig - 4

A 26-year-old male, Doctor by profession suffered acute onset rest angina. He had no symptoms of heart failure and had no history of cigarette smoking or illicit drug use. He could not recall any severe childhood illness and was not aware of a family history of chronic disease. On clinical evaluation, he had no significant positive physical findings.

Laboratory blood tests showed normal values of complete blood count, serum chemistry, fasting blood glucose, lipid panel, thyroid function, coagulation panel, erythrocyte sedimentation rate, and C- reactive protein level. Serology tests for hepatitis B and C, human immunodeficiency virus, Venereal Disease Research Laboratory, rheumatoid factor, and antinuclear antibody were all negative. His erect poster anterior chest X-ray was normal. Transthoracic echocardiography showed a mildly dilated right atrium, midly increased PA pressure of 37 mmg Hg, and a left ventricular ejection fraction of 35%-40%

His CPKMB and TROP I was very high.

Coronary angiogram (Fig. 1 to 4) revealed a giant RCA saccular aneurysm immediately adjacent to the ostium of the RCA and extending to the proximal and mid-RCA. The distal RCA was normal in caliber and course. The LMCA was normal. The left anterior descending (LAD) coronary artery ostium was aneurismal with distal LAD and Diagonal plaque. The left circumflex coronary artery (LCX) was normal. Aneurysms showed whirling pattern of contrast staining inside it.

Differential diagnosis

In adults, giant coronary artery aneurysm (gCAA) is predominantly atherosclerotic in origin. Other less common causes include connective tissue diseases, infections, vasculitis, Kawasaki disease, and congenital conditions. Our patient had no risk factors for atherosclerosis, history of connective tissue disease or vasculitis, and no evidence of systemic infection clinically or on workup. In addition, no supporting evidence for these etiologies of gCAA was revealed by serology tests and coronary angiography. Coronary angiography presented no findings suggestive of a congenital source such as associated fistulous connections.

Although the patient reported no childhood illness suggestive of Kawasaki disease, this disease can occasionally be manifested as a mild febrile episode, and the more characteristic mucocutaneous inflammation does not occur in all cases. Kawasaki disease is considered the most likely cause in our patient because of the multiple coronary arteries affected by aneurysms.

Treatment

Patient was asked to undergo CABGs.