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Research Journal of Life Sciences, Bioinformatics, Pharmaceutical and Chemical Sciences

Journal Home page http://www.rjlbpcs.com/



Original Case Report

DOI - 10.26479/2018.0403.03

NSTEMI REVEALING A GIANT CORONARY ARTERY ANEURYSM

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ABSTRACT: Giant coronary artery aneurysms are rare, with a reported prevalence of 0.02% to 0.2%. Causative factors of CAAs include atherosclerosis, Takayasu arteritis, congenital disorders, Kawasaki disease, syphilitic aortitis, scleroderma, systemic lupus erythematosus, Behçet disease, fibromuscular dysplasia and percutaneous coronary intervention. Surgical correction is generally accepted as the preferred treatment for giant coronary artery aneurysms. When surgical treatment is not possible, several authors to reduce the risk of in situ thrombus or distal embolization have supported the use of antiplatelet or antithrombotic medication (or both). We present an illustrative case of a giant (50/45 mm) coronary artery aneurysm in a 48-year-old man. In addition, we provide a review of the medical literature on giant coronary artery aneurysms.

KEYWORDS: Angina pectoris, aneurysm, dilatation, coronary angiography, antithrombotic, embolization, percutaneous intervention

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1.INTRODUCTION

Coronary artery aneurysms (CAAs) are seen in 0.3% to 5,3 % and is defined as a localized dilatation of a coronary artery segment more than 1.5-fold compared with adjacent normal segments [1]. Rarely, coronary artery aneurysms (CAAs) are large enough to be called giant CAAs [1]. Causative factors of CAAs include atherosclerosis, Takayasu arteritis, congenital disorders, Kawasaki disease, syphilitic aortitis, scleroderma, systemic lupus erythematosus, Behçet disease, fibromuscular © 2018 Life Science Informatics Publication All rights reserved

Peer review under responsibility of Life Science Informatics Publications 2018 May - June RJLBPCS 4(3) Page No.40 Kossir et al RJLBPCS 2018 www.rjlbpcs.com Life Science Informatics Publications dysplasia and percutaneous coronary intervention [1]. We present an illustrative case of a giant (50/45 mm) coronary artery aneurysm in a 48-yearold man. In addition, we provide a review of the medical literature on giant coronary artery aneurysms.

2. CASE REPORT



Figure 1:

A: Coronary angiography shows a circumflex artery aneurysm(arrow) and giant

LAD aneurysm (arrow).

B. Coronary angiography shows a giant (50/45mm) mid LAD aneurysm (arrows)

C. Coronary CT scan shows a giant LAD aneurysm (arrow).

A 48-year-old man with a history of end-stage renal disease and high blood pressure presented at the emergency department for the evaluation and management of angina pectoris. The clinical examination was normal. The electrocardiogram performed had objectified a subshift of the ST segment in anterior. An ultrasensitive troponin cycle was made, the first troponin was positive at 47 times the normal value, the second was positive at 65 times the normal value. Transthoracic electrocardiography performed showed akinesia of the anterior wall, anterolateral, anteroseptal with © 2018 Life Science Informatics Publication All rights reserved

Peer review under responsibility of Life Science Informatics Publications 2018 May - June RJLBPCS 4(3) Page No.41 Kossir et al RJLBPCS 2018 www.rjlbpcs.com Life Science Informatics Publications LVEF 30%. Emergency coronary angiography performed showed a subocclusive proximal LAD lesion, a giant mid LAD aneurysm (Fig.1A), and circumflex artery aneurysm (Fig.1B). To further evaluate the angiography findings, we ordered a coronary CT scan; this showed a giant LAD aneurysm (Fig.1C). The patient was evaluated by our cardiothoracic surgery department, which recommended surgical repair of the aneurysm. However, the patient refused further intervention. After receiving a dialysis treatment, he was discharged from the hospital on warfarin and could not be contacted for a follow-up appointment. Unfortunately, we have no idea about the clinical evolution; our patient was lost to follow-up.

3. DISCUSSION

We define CAAs as a local dilatation in the coronary artery that are 1.5-fold greater than normal adjacent segments [1]. The overall incidence of CAAs ranges from 0.3 to 5.3% [1,2]. There is an entity of CAAs, called giant CAAs. A universally accepted definition of a giant CAA does not exist but some authors describe dilatations that exceed 4 cm in diameter as giant coronary aneurysms [3]. The published data on giant CAAs report a prevalence of 0.02% to 0.2% [4,5]. The incidence of CAAs is higher in men than in women, 2.2 versus 0.5%, respectively [6,7]. However, although CAAs can be seen at any age, those related to atherosclerosis usually appear later in life compared to CAAs related to other causes [8]. The most frequent cause of CAAs and of giant CAAs is atherosclerosis, which has been linked to 50% of CAAs diagnosed in adults [9]. In a large angiographic series, the Coronary Artery Surgery Study (CASS) registry [10, 11], investigators reported a prevalence of 4.9% [12], which exceeded the rates of CAAs in many other angiographic studies (0.37–2.53%) [13]. The other causes of giant CAAs include congenital heart disease, trauma, Ehlers-Danlos syndrome, Kawasaki disease, Marfan syndrome, Takayasu arteritis, polyarteritis nodosa, syphilitic aortitis, scleroderma, systemic lupus erythematosus, Behçet disease, fibromuscular dysplasia and percutaneous coronary intervention [14,35]. In this case, our patient had no history of connective-tissue disease, vasculitis, trauma, or infectious processes; therefore, atherosclerotic disease was considered to be most likely. Histological examination of atherosclerotic CAAs include hyalinization, lipid deposition, disruption of intima and media, focal calcification and fibrosis, cholesterol crystals, intramural hemorrhage, and foreign-body giant-cell reaction to the atherosclerotic process [15, 16–21]. Such features weaken the vessel wall and decrease its elasticity, ultimately reducing the vessel's tolerance to intraluminal pressures of blood flow, thus predisposing it to subsequent dilatation and aneurysm formation. It is very important to note that the chronic transmural inflammation seen in atherosclerotic disease aggravates this process of vessel wall weakening [5,16-21]. The right coronary artery is the most frequently affected, and it is involved in 40-70% of giant CAAs [22, 23,24]. According to the study carried out by Keyser and colleagues on 28 cases of giant CAA, the vast majority (89%) occurred in the right coronary artery (85.7% were

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Kossir et al RJLBPCS 2018 www.rjlbpcs.com Life Science Informatics Publications found in the proximal portion thereof) [5]. One giant CAA involved both the left and right coronary artery systems. Giant aneurysms from the proximal LAD are exceedingly rare [25]. It should be noted that CAAs of atherosclerotic or inflammatory origin are often multiple and involve more than one coronary artery, whereas the CAAs resulting from other causes generally affected a single artery [15]. Our illustrative case is unusual because the aneurysm occurred in the left system and was markedly large, at 50 × 45 mm. Clinical manifestations of CAAs vary and are often related to the cause. In most cases they are asymptomatic, but sometimes patients present with with angina pectoris, myocardial infarction, sudden death, hemopericardium, fistula formation, tamponade, of compression surrounding structures. or congestive heart failure [9,15,23,24,26,27,28,29,30,31,32]. Patients with giant CAAs can also present with mediastinal mass or with superior vena cava syndrome [23]. There are several methods of imaging techniques used to view giant CAAs, but the coronary angiography remains the gold standard, it provides information about the size, location, number and shape of the aneurysm. However, it is difficult to determine by coronary angiography if it is a true or false aneurysm, hence the important of intravascular ultrasound which allows in addition to the differentiation between a true and a false aneurysm, to give information on the composition of the lumen and the arterial wall structure [14]. Surgical treatment is generally preferred for Giant CAAs. However, the intervention necessitates median sternotomy, cardiopulmonary bypass, and myocardial revascularization. Therefore, certain patients will not be candidates for such an invasive procedure [23, 24, 33,34]. In the study done by Keyser and colleagues, 19 of 28 were treated surgically. 5 patients were treated with antiplatelet or anticoagulant medication; Only one of the 5 patients treated conservatively had an unventful course. One was treated by percutaneous intervention and the 3 patients who received no therapy died [5]. When Surgical treatment is not possible, the use of antiplatelet or antithrombotic medication (or both) has been supported by several authors to reduce the risk of in situ thrombus or distal embolization [14]. A study conducted by Banerjee and colleagues who presented a case of a giant right CAA partially filled with thrombus treated with caution and success with warfarin [30]. It is now possible to treat patients with less invasive percutaneous techniques. The choice of treatment takes into consideration the immediate and long-term risks associated with percutaneous procedures or surgery compared with the complications that can occur during prolonged antithrombotic therapy [35]. When percutaneous exclusion has been decided. Therapeutic options include include stent placement with or without coil embolization [15,28,36]. Therefore, the less invasive percutaneous approach of giant CAAs seems reasonable for aneurysms greater than 5 mm but less than 10 mm [14]. In our case, the patient refused further intervention, so the decision of a conservative medical treatment was adopted. Unfortunately we have no idea about the clinical evolution; our patient was lost to follow-up.

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Giant CAAs are rare, with a reported prevalence of 0.02% to 0.2%. The most frequent cause is atherosclerosis. Surgical treatment is generally preferred for Giant CAAs. When surgical treatment is not possible, the use of antiplatelet or antithrombotic medication (or both) reduces the risk of in situ thrombus or distal embolization.

5. CONFLICT OF INTEREST

The authors report no relationships that could be construed as a conflict of interest.

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