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Mini Review

Coronary Artery Aneurysms: Review with Emphasis on Etiology

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ABSTRACT

Coronary Artery Aneurysm (CAA) belongs to vessels malformations with low frequencies, approximately 0.35%. It is observed different causes of CAAs: Congenital and acquired, inflammatory and iatrogenic. CAAs are independent risk factor of myocardial infarction, bleeding, heart failure, death. In time detection of arteries aneurysms, especially silent, preserve from development of such dangerous complications. The better understanding of CAAs pathogenesis and etiology can improve diagnosis. In the review it was summarized the etiological causes of CAA; described clinical presentations and diagnostic investigations of CAA; assessed the options of medical and surgical management of CAA; and estimated the prognosis of this rare pathology.

Keywords: Coronary artery aneurysm; Pathogenesis of coronary artery aneurysm; Management of coronary artery aneurysm; Molecular and genetic changes

INTRODUCTION

Due to the low incidence of this pathology, more often CAA have been described in the clinical cases or analyzed in small studies. The biggest prospective research was conducted from 2004 to 2016 [1]. Among 436,467 patients from 9 countries, 32 hospitals, more than 1500 were included in the Coronary Artery Aneurysm Registry (CAAR). The frequency of CAA was 0.35% according to the data of this study. But CAAs impact on mortality is more seriously. The outcomes of CAA were evaluated in several studies. The data from a long standing prospective study showed a 12.8% mortality rate (cardiac causes were observed in 26.4%) and a 42% incidence of cardiovascular events. The presence of CAA was considered an independent risk factor for both mortality (Hazard Ratio: 3.1, CI 95% 1.8-5.6; p=0.000) and MACE (Hazard Ratio: 2.3, CI 95% 1.4-3.8; p=0.000), as calculated using various Cox multivariate models [2]. Similar results were revealed in CAAR, mortality and MACE incidents were 15.3% and 31%, respectively [1]. Baman et al. described coronary artery aneurysm as an independent predictor of death, an overall 5-year survival for CAA patients was 71% [3].

CAAs are extremely heterogeneous in etiology causes, pathogenesis, clinical manifestations. The deep appreciation of the nature of CAA results in proper management of such pathology.

LITERATURE REVIEW

Coronary artery aneurysm is characterized as abnormal vessel dilatation, exceeding the 1.5-fold diameter of the adjacent normal segment [4]. It is distinguished different types of CAA based on their sizes, forms, and structures shown in Table 1 [5-7].

Several studies have assessed the prevalence of CAAs localization. It was observed the controversial results. The data of the most representative research pointed the Left Anterior Descending Artery (LAD) as the place of CAA (48.6%), followed by the Right Coronary Artery (RCA) (31.8%), and the Circumflex (Cx) (28.1%) [1]. Aneurysms were also revealed in the left main artery. In some studies results are similar, although other studies demonstrated a higher frequency of CAA in RCA [2,7-9]. According to CAAR isolated CAA is more common (83%), followed by both coronary artery aneurysms (12.8%) [1].

Although CAAs are developed due to weakening of the vessel wall and subsequent dilation, the histological and pathogenetic changes in the aneurysms differ depending on the etiological causes. All the underlying pathological processes are illustrated shown in Figure 1.

DISCUSSION

CAAs manifestations vary from asymptomatic status to sudden cardiac death, including stable angina, myocardial infarction (STEMI or NSTEMI), syncope, sudden cardiac death, fistula formation, rupture, compression of surrounding structures, or congestive cardiac failure [4, 6, 24]. It is considered that stable angina is the most common presentation [2,7].

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CAAs thrombosis, due to the irregular endotelium of the aneurysm or distal embolization, are the causes of acute coronary syndrome. The highest risk of thrombosis is observed, when aneurysm diameter exceeds 5 mm [25]. It can progress to ST-elevation myocardial infarction or non-ST elevation myocardial infarction or sudden cardiac death [1,2].

In some studies, it was described that CAAs lead to the development of chronic heart failure [2,6].

Giant CAAs manifest by sins of the surrounding structures compression, more common vena cava superior [6]. Patients suffer also of dysphagia. Giant CAAs should be differentiated from cysts, cardiac tumors, and other masses [26].

In recent large comparative study, focus on the patient with Coronary Artery Fistula (CAF), was revealed that CAF was complication of CAA in 74.83%. Combination of CAF and CAA associates with larger fistulous diameter and coronary-cardiac chamber arterial fistulas [27].

Life-threatening CAAs complication is aneurysm rupture followed by hemopericardium or heart tamponade [24].

In rare cases aneurysm can be localized multivessels. Li-Cheng Jiang et al conducted systemic review on PubMed and Embase to assess frequencies and typical placements of concomitant vascular aneurysms. The study analyzed the data of 61 articles with a total of 76 patients (average age: 37.4±26.5 years; male: 58 [76.3%]). According to their results, CAAs co-existed more common with abdominal aorta aneurysms (n=40, 52.6%) and common iliac artery (n=38, 50%). Other localizations of vascular aneurysms included all medium-sized arteries [28]. The most common etiology of multiple aneurysms was Kawasaki Diseases (KD) (43.3%) and atherosclerotic disease (16.4%).

Diagnosis of aneurysms

There aren't specific changes in common laboratory tests. In novel studies the researchers try to identify the potential molecular biomarkers of CAAs and reveal the gene predispositions to the CAAs development regarding the early and proper prevention and diagnosis of such disorder.

MicroRNAs (miRNAs) are suggested as new biomarkers of cardiovascular diseases, useful for both diagnosis and prognosis. Despite the roles of some types of miRNA were described in atherogenesis, myocardial infarction, aortic aneurysms and many another cardiology diseases the potential effect of miRNA on CAAs formation aren't studied properly [29]. In recent study it was assessed the expression of five preselected miRNA markers (miR-125b-5p, miR-210-3p, miR-328-3p, miR-425-3p, and miR-483-5p), and three reference miRNAs (miR-16-5p,miR-30d-5p, miR-320d) in 3 groups (CAAs group, Coronary Artery Disease (CAD) group, control group) to determinate the typical changes for CAAs. The results showed that miR-451a can serve as biomarker of CAAs surround the patients with CAD and miR-328-3p predicts CAAs in general population [30].

The pathogenesis of atherosclerosis CAAs is intricately linked to the overexpression of matrix metalloproteinasesMMPs (enzymes which destroy the vessel connective tissue). It was revealed an elevation in MMP-2, MMP-3, MMP-9, and MMP-12, alongside a reduction in TIMPs, such association resulted in extensive arteries remodeling, dilatation and CAAs development [16]. In recent study it was presented that the plasma levels of MMP8 were significantly higher in the patient with CAAs than in the CAD group and control groups [31]. The marker of endothelial dysfunction and stress responses fibronectin 1, are dercreased in both CAD and CAA [32].

Kawasaky disease, vasculaties, is characterized by activation of inflammatory cytokines. It was observed increased TNF-alpha levels, elevated secretion of vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) Complement system dysfunction are confirmed by decreased levels of complement factor H and Mannose-Binding Lectin 2 in KD with CAAs [15, 32]

The genetic predispositions of KD are the most studied among all types of aneurysms. Several researches were performed to find typical genes changes for CAAs. Early it was observed a connection between CAAs formation and variations in MMP-9 gene polymorphisms, the genetic variant rs2833195 in the intron of the TIAM1 gene [16, 33]. At recent study an association of CAAs and changes in the intergenic region on chromosome 20q13 have been found [34]. Korea researchers detected by sex-stratified Genome-Wide Association Studies (GWAS) 6 male-specific (PDE1C, NOS3, DLG2, CPNE8, FUNDC1, and GABRQ) and 2 female-specific (SMAD3 and IL1RAPL1) susceptibility loci in patients with CAA due to KD [35].

The mutation of fibrillin 1 (FBN1) gene, results in heightened TGF-β activity, have been revealed to be associated with arterial aneurysms in Marfan syndrome [22,23]. There were the attempts to discover the gene predisposition of CAD by using GWAS. It wes observed that the changes of HLA-E and MMP-3 gene, chromosome 9p21.3, as well as insertion/deletion polymorphisms of the angiotensin-converting enzyme (ACE DD genotype), SRC-1 and GRIN3A genes have been linked to CAD, abdominal aortic and intracranial aneurysms [17,18]. Further studies of such genes are needed to detect the association with CAAs formation. At new published study at first it was observed the associating a single nucleotide variant p.P517R in exon 22 of COL3A1 with the CAAs without previous arteries disorders [36]. Imaging techniques used to diagnose CAA consist of coronary angiography, Intravascular Ultrasound (IVUS), Computed Tomography Angiography (CT angiography), coronary MR Angiography (MRA), and echocardiography. All of them supply information about aneurysms location, shape and frequencies; the presence of thrombi, coronary arteries stenosis and occlusion. Coronary angiography remains the most commonly performed method for revealing CAA, with limitations in assessing the actual aneurysm size and in differentiating between a true aneurysm and a pseudoaneurysm [37-39]. IVUS, as the "Gold Standard" for diagnosing CAA, provides essential information about arterial wall structure and luminal composition, with the option to differentiate between various types of aneurysms and to identify the association of CAAs with previously implanted stents [40]. CT angiography, non-invasive method, becomes more often used for the diagnosis of CAA [41,42]. However, limitations include exposure to radiation and the use of iodinated contrast media, which may be relevant for patients with kidney failure. Magnetic Resonance Angiography (MRA) is performed for diagnosing CAAs in the case of contraindication to CT angiography [35]. Transthoracic or transesophageal echocardiography is preferable for diagnosing CAAs due to Kawasaki disease in children [43].

Management of coronary artery aneurysms

In current studies it was proposed different therapy algorithms and

strategies, but there are no standardized guidelines for the diagnosis and management of patients with CAA [1,2,7,44]. The treatment of CAA depends on clinical presentations (silent or symptomatic), etiologies, CAAs morphology (size, location, shape), patient comorbidities and cardiovascular risk factors. Different options include medical treatment, percutaneous coronary intervention and surgical treatment.

Medical treatment

Medical management of CAAs consists of risk modification and antithrombotic/anticoagulant therapy [5,45,46] . In the studies, the antiplatelet medications were common used in the case of atherosclerosis etiology of CAAs [1,45,46]. In the Coronary Artery Aneurysm Registry (CAAR) it was observed that aspirin being the most commonly prescribed, 90.2% of cases; dual antiplatelet therapy, for 12 months period, was used in 64.8%. In 13.4% of cases, for management CAAs with co-existing aneurysms at multiple locations or in high thrombotic risk, longer dual therapy or a combination with anticoagulants was proposed [1]. The antiplatelet medicine is recommended as single therapy for asymptomatic patient with CAA [7]. It was recommended anticoagulants, specifically vitamin-K antagonists for treatment the coronary artery ectasia. Doi et al. demonstrated a positive effect of warfarin in such patients. Another indication for anticoagulants was multivessel aneurysms or disorders with high prothrombotic risk [47] Figure 1.

The treatment of CAAs in the patients with KD also includes warfarin, especially for large or rapidly progressing aneurysm. According to autoimmune inflammation pathogenesis of KD, intravenous immunoglobulin was recommended [48]. As pathogenetic therapy, statins and angiotensin-converting enzyme inhibitors were proposed due to their ability to inhibit matrix metalloproteinases [49,50]. Immunosuppressive therapy should be prescribed for autoimmune CAAs management. It is recommended glucocorticoids, cyclophosphamide, methotrexate for treatment polyarteritis nodosa, glucocorticoids, rituximab- for IgG4-related disease [51,52].

Surgery managment

Operative treatment is represented by Percutaneous Coronary Intervention (PCI) or surgical management. The intervention type depends on clinical manifestations, CAAs morphology and surgeon experience due to the absent of the official guideline. The indications for PCI and surgery, the intervention options are shown in Tables 1 and 2.

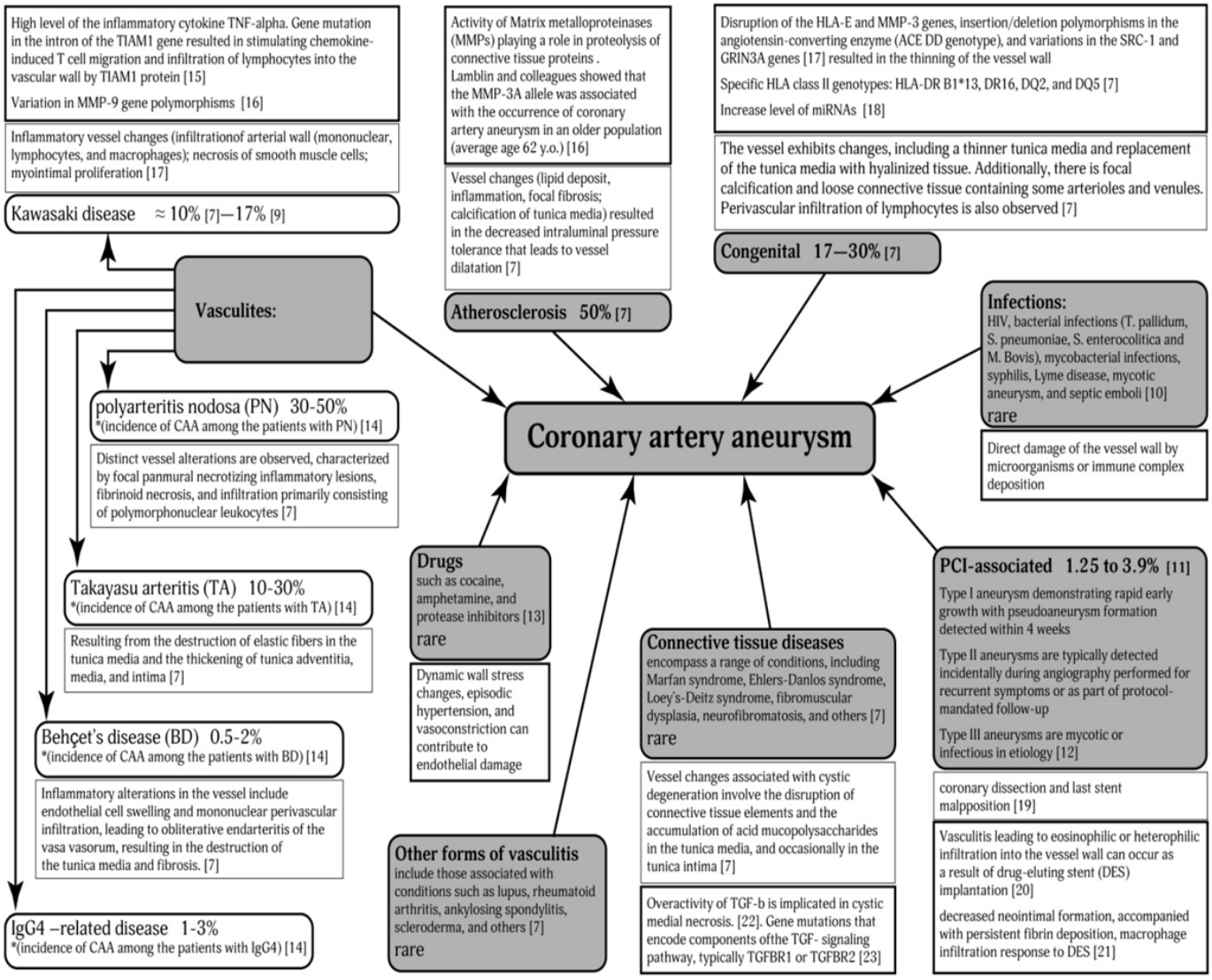

Figure 1: The etiologies of CAA.

Table 1: The types of CAA.

Aneurysmal dilatation of coronary arteries			
1. Coronary aneurysm three vessel layers are exicted	2. Coronary artery ectasia	3. Giant coronary aneurysm	4. Pseudoaneurysm loss of vessel wall integrity
Focal enlargement more than 1.5 times the adjacent normal segment	Diffuse enlargement more than 1.5 times the adjacent normal segment	Diameter >20 mm	
Saccular aneurysm: Longitudinal diameter < Transverse diameter	Fusiform aneurysm: Longitudinal diameter > transverse diameter		

Table 2: The indications and types of interventional therapy of CAA.

	procedure	indication
PCI	Stent implantation (DES, PTFE stent graft); Stent-assisted coiling ; Coil embolization [7,53,54]	-Single-vessel or focal multivessel disease; - No left main coronary artery involvement; - No mechanical complications; - Suitable anatomy for PCI - ACS [6]
Surgery	Resection of the aneurysm associated with CABG; Proximal and/or distal ligation associated with CABG; Aneurysmal thrombectomy; Marsupialization with interposition graft; CABG [55-57]	-CAAs in the LMS -CAAs near the bifurcation of large branches -CAA complicated by fistula formation -Compression of cardiac chambers -Giant CAA (dilatation exceeding the reference vessel -diameter by > four times) -CAA complicated by embolization of distal part of -Coronary artery -CAA progressive enlargement [5]

CONCLUSION

Despite CAAs are low frequency pathology, they can lead to fatal outcomes in patient. The etiologies of aneurysms are very heterogeneous: atherosclerosis, congenital changes, vasculitis, connective tissue diseases, infections, drugs, PCI association. The better knowledge of CAAs causes, clinical implementation of molecular biomarkers and gene predisposition of CAAs can improve early diagnosis of such vascular pathology and allow to avoid dangerous complications. When enough prospected researches will be perform, the guideline for management CAAs can be created.

CONFLICT OF INTEREST

The author declare that there is no conflict of interest.

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