Progress in Cardiovascular Diseases 62 (2019) 265–271

Contents lists available at ScienceDirect

Progress in Cardiovascular Diseases

journal homepage: www.onlinepcd.com

Non-arrhythmic causes of sudden death: A comprehensive review☆,☆☆

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ARTICLE INFO

Keywords:
Sudden cardiac death
Nonarrhythmic
Coronary artery disease

ABSTRACT

Sudden cardiac death (SCD) is a major public health issue in the United States and worldwide. It is estimated to affect between 1 and 1.5 million patients worldwide annually, with the global burden expected to rise due to the concomitant rise in coronary artery disease in the developing world. Although arrhythmic causes of SCD such as ventricular tachycardia and ventricular fibrillation are common and well-studied, non-arrhythmic causes are also important, with diverse etiologies from ischemia-related structural heart disease to non-ischemic heart diseases, non-atherosclerotic coronary pathologies, and inflammatory states. Recent research has also found that risk factors and/or demographics predispose certain individuals to a higher risk of non-arrhythmia-related SCD. This review discusses the epidemiology, mechanisms, etiologies, and management of non-arrhythmic SCD.

Contents

Abbreviations: CAD, coronary artery disease; ICD, implantable cardiac defibrillator; LV, left ventricle or ventricular; LVEF, left ventricular ejection fraction; MI, myocardial Infarction; NA SCD, non-arrhythmic sudden cardiac death; NSTEMI, nonsca-ST elevation myocardial infarction; SAD, sudden arrhythmic death; SCAD, spontaneous coronary dissection; SCA, sudden cardiac arrest; SCD, sudden cardiac death; STEMI, ST elevation myocardial infarction.

☆ Conflict of interest: None.

☆☆ The Authors have no disclosures.

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https://doi.org/10.1016/j.pcad.2019.05.004
0033-0620/

Introduction

Sudden cardiac arrest (SCA) can be defined as an immediate and unexpected cessation of cardiac activity, often leading to sudden cardiac death (SCD). The duration of symptoms (if any) is important to this definition: The World Health Organization defines SCD as unexpected death within 1 h of symptom onset if witnessed, or within 24 h of the person having been observed alive and symptom-free if unwitnessed.[1]

There are multiple causes of SCD (Fig. 1). The most common cause of SCD is SCA due to a lethal arrhythmia, frequently occurring in the presence of coronary artery disease (CAD), with or without reduced left ventricular ejection fraction (i.e., LVEF <40%). However, a significant percentage of patients are believed to suffer non-arrhythmic SCD (NA-SCD). For example, a recent study assessing autopsy-proven causes of death estimated that 8% of witnessed and 3% of unwitnessed SCD were nonarrhythmic in origin.[2] For example, pulmonary embolism and drug overdoses are often mistaken for SCD, and usually are differentiated only on blood testing or at autopsy.

In this review, we discuss the incidence, pathophysiology, risk factors, and prevention strategies for NA-SCD.

Epidemiology

The reported incidence of SCD varies widely, due to varying definitions and methods of adjudicating cause of death. The estimates range from 180,000 to 450,000 cases annually in the United States, accounting for approximately 5–20% of the total annual mortality.[1,3,4]

Without a complete autopsy, however, noncardiac and non-arrhythmic conditions leading to SCD often cannot be correctly identified.[5] Current autopsy rates are quite low in the United States for out-of-hospital natural deaths (around 10%).[6] A recent study that did perform autopsies concluded that arrhythmic deaths made up 56% of SCDs, cardiac non-arrhythmic causes of death (e.g., heart failure, tamponade) accounted for 4%, and non-cardiac sudden death made up the remaining 40% (Fig. 2).[2] Interestingly, despite the advances in the management of cardiovascular disease, the incidence of SCD has remained approximately the same over time.

Post-myocardial infarction (MI) NA-SCD

SCD remains a serious complication post MI[7] despite significant advancements in treatment, including immediate coronary revascularization,[8]

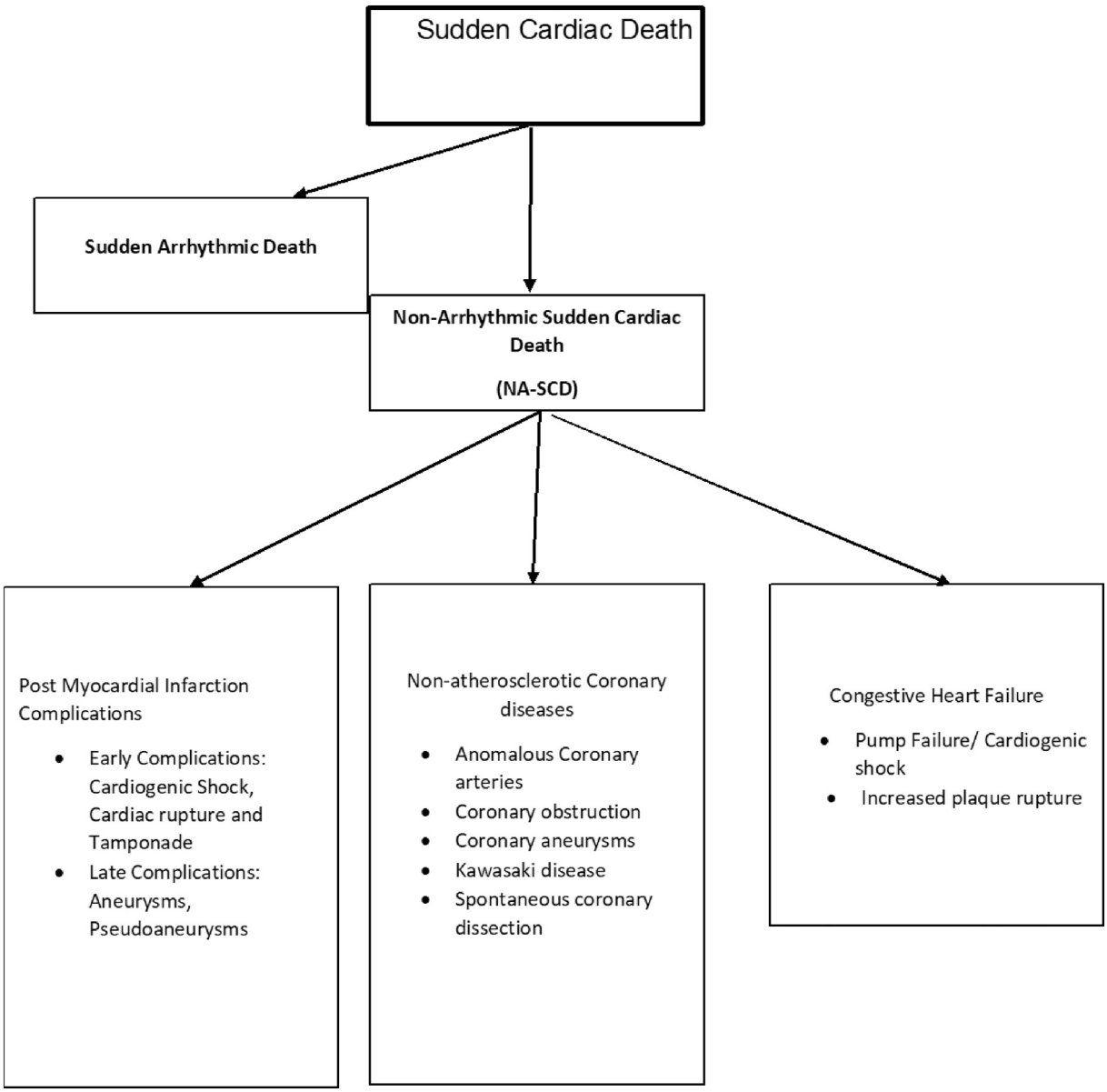

Fig. 1. Classification of sudden cardiac death.

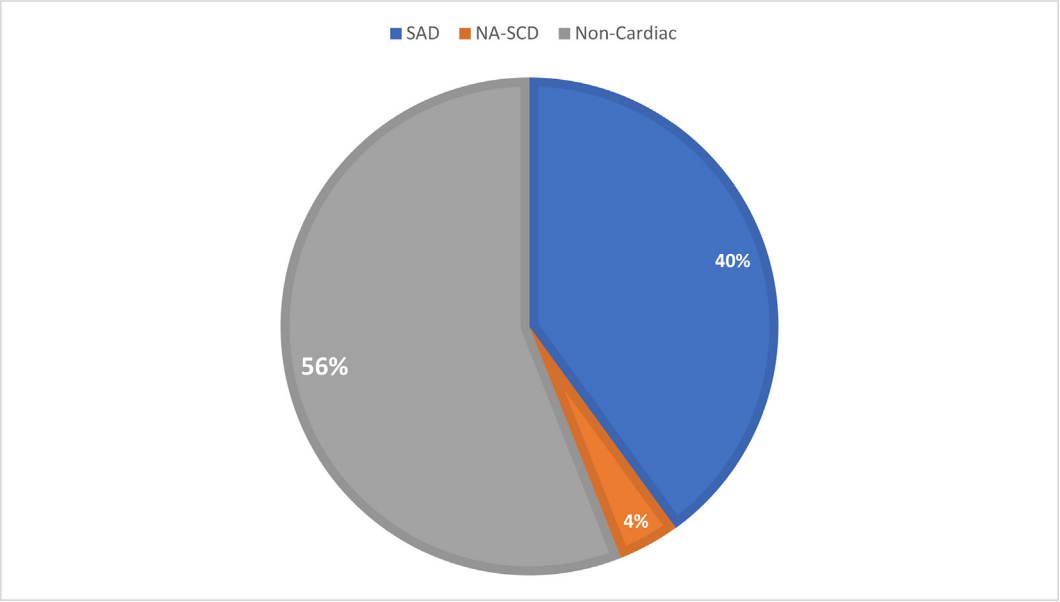

Fig. 2. Causes of sudden cardiac death in autopsy patients.

dual antiplatelet therapy,[9] and guideline directed medical therapy[10] resulting in increased survival.[11] Despite these advancements, post -MI patients remain at significant risk, with SCD accounting for approximately half of the overall mortality[12,13] that occurs in the early post-MI period.[14,15]

In patients at moderate to high risk of SCD, clinical trials have been performed[16]; the results of which have established implantable cardioverter-defibrillators (ICDs) as beneficial in post-MI patients who have a persistently low LVEF. However, a sizeable percentage of post-MI mortality is due to NA-SCD, most often caused by mechanical complications of damage to the myocardium.

Epidemiology and natural history of NA-SCD Post MI

SCD following MI has been extensively studied. The best studies are population-based, with autopsies performed to accurately adjudicate the cause of death. In an Olmsted county population surveillance study,[14] there was a steady decrease in post-MI SCD between 1979 and 2005, likely due to improvement in therapy and early revascularization. Post-MI risk factors for SCD included older age, female sex, history of hypertension or diabetes mellitus, and higher Killip class. In addition, a large study examining autopsy records of patients from the VALIANT trial analyzed time-related causes of death in MI patients.[17] That study found that 54 patients with no specific findings were presumed to have suffered sudden arrhythmic death (SAD), while 51 had specific pathologic findings indicating nonarrhythmic death. Of these 51 patients, 44 died of a fatal MI (28 recurrent MI, 13 cardiac rupture, 3 initial MI), while 4 died of pump failure or cardiogenic shock. The other 3 patients died of non-cardiac causes (stroke, drug overdose, pulmonary embolism). Significant differences between the SAD group and the NA-SCD group in this study included lower LVEF in the SAD group ($31.5 \pm 8.2\%$ vs. $35.9 \pm 11.8\%$, $p = 0.035$). Time to death was also markedly different, with most NA-SCD's occurring in the first month after the index MI, while arrhythmic deaths occurred more than twice as long after NA-SCDs (306 ± 246 days vs. 137 ± 194 days, $p < 0.001$). This high frequency of early NA-SCD may provide insight into the failure of ICD to reduce all-cause mortality when implanted early post-MI.[1819]

The prevalence of NA-SCD causing half of SCD after acute MI is mirrored in other landmark studies.[20,21] In all these trials, a low percentage of patients (15–20%) received autopsies. Similar results were found in another study examining SCD following Non-ST elevation MI (NSTEMI) in 4 major trials.[22] Those investigators found that following NSTEMI, SCD occurs relatively infrequently (~1% per year). However, the incidence of SCD in this population increases with time and increases further following recurrent NSTEMI.

Etiology of NA-SCD post MI

The etiology of NA-SCD in the immediate post-MI period can be classified into early complications (defined as happening a few hours to a few days post MI) and late complications occurring weeks to months post MI.

Early complications of MI leading to NA-SCD

Cardiogenic shock

Cardiogenic shock is the most common cause of death in patients with acute MI[23,24] and is more common in STEMI compared to NSTEMI patients, likely due to complete coronary arterial occlusion and higher likelihood of developing large areas of myocardial necrosis.[25]

The most common causes of cardiogenic shock leading to NA-SCD in acute MI include sudden severe LV dysfunction, ventricular septal defect from myocardial rupture, or papillary muscle rupture leading to severe mitral regurgitation and flash pulmonary edema.[26–29] In these cases, NA-SCD occurs due to a vicious cycle of decreased cardiac output from LV dysfunction due to the above causes, drop in systolic blood pressure, with sympathetic overdrive and activation of the renin-angiotensin system leading to vasoconstriction, sodium and water reabsorption, tissue hypoxia, lactic acidosis, and further compromise of myocardial function, all culminating in death.[27] Risk factors such as advanced age, diabetes mellitus, and large anterior infarctions predispose these patients to cardiogenic shock.[30,31]

Treatment for those who are resuscitated includes mechanical circulatory support with an intra-aortic balloon pump or percutaneous LV assist device (e.g., Impella or Tandem Heart) with emergent percutaneous intervention or surgical revascularization combined with repair of the ruptured myocardium, papillary muscles, or both.[32–34]

Cardiac tamponade

Another early complication that leads to NA-SCD occurring 4–7 days post-MI is cardiac tamponade from free wall rupture of the myocardium. This most often follows a large MI, or infarction resulting in ventricular septal rupture or papillary muscle rupture that extends to the

epicardium.[35,36] Cardiac tamponade leads to SCD, due to sudden increase in intrapericardial fluid pressure that exceeds central venous pressures, ultimately impeding cardiac venous return. If acute, only a small amount of pericardial blood (200–300 cc) is required to cause this phenomenon.[37]

Risk factors for developing structural complications leading to tamponade post-MI include age > 60 years, female sex, pre-existing hypertension, and lack of LV wall hypertrophy.[35] The lateral and inferior aspects of the LV are known to be most susceptible to post-infarct rupture.[36] Patients who are resuscitated from pericardial tamponade usually are managed with emergent pericardiocentesis and surgical repair as needed.

In rare cases, late tamponade may occur from Dressler's syndrome,[28] which is post-MI secondary pericarditis that also may cause a pericardial effusion. Dressler's syndrome is most often encountered some weeks after a large infarction. It is presumed to be triggered by an immune response from pericardial and myocardial cells.[38]

The preferred agent to treat acute pericarditis caused by MI is aspirin in a dosage of 650 to 1000 mg four times per day with a taper over four weeks.[39,40] In addition, colchicine may be used to treat recurrent pericarditis or pericarditis that does not respond to conventional treatment.[41] Glucocorticoids should not be used as first-line therapy for patients with acute pericarditis, because this practice increases the risk of recurrence and infection.[39]

Late complications of MI

LV aneurysm

An aneurysm may develop after a transmural MI due to scar thinning and infarct expansion; LV aneurysms include epicardium, myocardium and endocardium, and have a wide neck with smooth margins[42] (Fig. 3). Other causes of LV aneurysms, such as Chagas disease, sarcoid, blunt chest trauma, and congenital malformations, are exceedingly rare, but do occur. The incidence of true LV aneurysm in autopsy studies is approximately 3.5–5%.[43]

Although LV aneurysms are well known to predispose to ventricular arrythmias and consequently sudden arrhythmic death,[44] they also can predispose to NA-SCD in the form of late wall rupture.[45] Another cause of LV aneurysm is the LV apical aneurysm that can develop in hypertrophic cardiomyopathy (HCM) patients. These are commonly associated with an adverse clinical course, including SCD.[46]

LV pseudoaneurysm

A pseudoaneurysm is a rare complication (incidence ~0.3%)[47] that usually develops between a few days to 2 weeks after a large transmural acute MI complicated by ventricular free wall rupture that is fortuitously contained by localized pericardial adhesions. As depicted in Fig. 4, it is characterized by a narrow neck that communicates freely between the pericardium and the LV. Pseudoaneurysms tend to expand and rupture, which differentiates them from the more stable true LV aneurysm described above. Other rare causes of pseudoaneurysm include trauma, endocarditis, and iatrogenic (e.g., resulting from surgery).

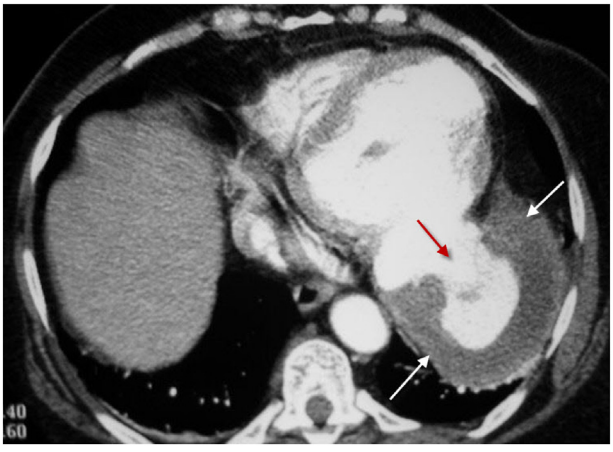

Fig. 3. Cardiac computed tomography demonstrating a left ventricular aneurysm (adapted with permission from Herring W: Learning Radiology (ed 4) Philadelphia, PA, Elsevier, 2015.)

Patients with pseudoaneurysms are at high risk of SCD due to rapid expansion and sudden rupture of unstable pericardial adhesions in communication with the LV via the myocardial defect. The incidence of SCD as the presenting symptom of LV pseudoaneurysm is 3%.[48] Patients may also die suddenly from ventricular arrhythmias due to arrhythmogenic foci around the pseudoaneurysm, or related myocardial electrical reentry. Similar risk factors that predispose to LV aneurysms may play a role in pseudoaneurysms. These include advanced age, female sex, hypertension, transmural MI, and lack of revascularization or late presentation.[49]

Pseudoaneurysms are usually fatal if ruptured. If diagnosed before rupture via echocardiography or other imaging modalities, early surgical intervention by patch closure, or percutaneous closure (e.g., via Amplatzer device) in high operative risk patients, is recommended[49,50].

NA-SCD related to coronary artery conditions other than acute coronary syndrome (ACS)

Coronary obstruction

Total, or subtotal, occlusion of coronary arteries most often results from thrombotic occlusion due to atherosclerotic disease and ruptured vulnerable plaques. Arterial occlusion also may occur as a result of external compression. Though in cases of such obstruction the proximal cause of death may be arrhythmia, the ultimate cause is ischemia rather than primarily arrhythmic. Although rare, case reports have described coronary compression by large aneurysms of the sinuses of Valsalva resulting in SCD.[51] Another rare cause in this category is flow limitation by tumors, such as papillary fibroelastomas occurring on the aortic valve causing ostial coronary artery occlusion, or external coronary compression due to growing neoplasms.[52]

With the advent of transcatheter aortic valve replacement, a new category of external coronary occlusion has become evident. Poor patient selection, malpositioning of the prosthesis, or inaccurate measurement of the sinus of Valsalva can lead to misplacement of the valve prosthesis and ultimately to delayed coronary obstruction and resultant SCD. The incidence of such obstruction has been reported at 0.2% and is more common in patients undergoing valve-in-valve procedures within stentless surgical valves with valve leaflets external to the sewing ring (0.9%).[53,54]

Coronary aneurysms

Coronary aneurysms in adults

Coronary arterial aneurysms can be defined as a >1.5-fold dilatation of the coronary artery compared to adjacent segments.[55] They are rare, with an estimated lifetime incidence of 0.3–5%.[55] Atherosclerosis is responsible for almost half of coronary aneurysms.[56,57] Rare causes include Percutaneous Coronary Intervention-related,[58] infectious,[59] and congenital (see below). Sudden cardiac death can be caused by rupture of the aneurysm, or thrombus formation due to turbulent flow within the aneurysm[60,61].

Coronary aneurysms in infants and young adults - Kawasaki disease

Kawasaki disease is an acute, self-limited inflammatory vasculitis of unclear etiology.[62] It affects small to medium sized arteries.[63] It is the most common cause of acquired coronary artery pathology in infants and young children,[64,65] with a prevalence in those of Asian (especially Japanese) descent.[66] There is a slight male predominance. Serious

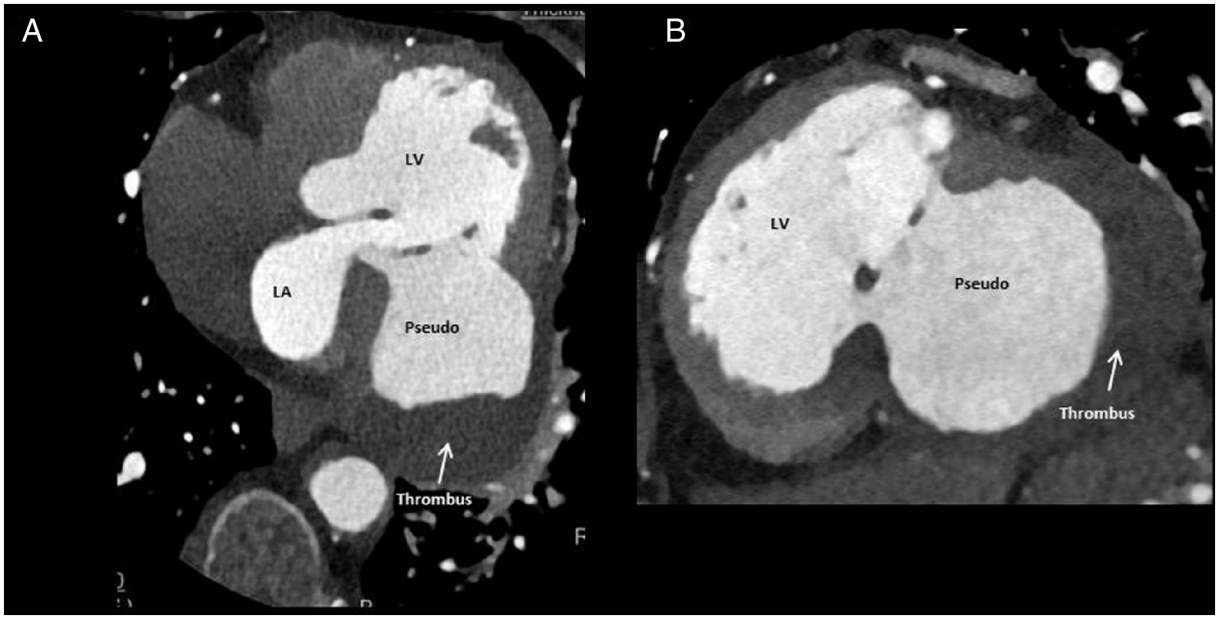

Fig. 4. Cardiac computed tomography demonstrating a left ventricular pseudoaneurysm (adapted with permission from Reyaldeen et al., Multimodality Imaging in a Case of Chronic Massive Left Ventricular Pseudoaneurysm CASE (Phila). 2018 Jun; 2(3): 95–98.)

coronary and myocardial complications are fortunately relatively rare (3%), and mostly seen in older children.[67]

If not promptly treated with intravenous immunoglobulins and aspirin, about a quarter of those with Kawasaki syndrome develop coronary artery aneurysms, which can lead to SCD from occlusion and rupture.[68–71]

SCD associated with spontaneous coronary artery dissection(SCAD)

SCAD is a condition defined angiographically by dissection of the coronary intima or media, with or without adventitial hematoma formation.[72] The frequency of SCAD varies between 0.1% and 4%[73–76] as a cause of ACS and accounts for approximately 0.4% of cases of SCD.[77] The demographics vary depending on the cause of SCAD. While atherosclerotic SCAD is often seen in a patient with risk factors for CAD, most often it is related to typical acute coronary syndrome (ACS). Non-atherosclerotic (or "true") SCAD is most often seen in the young female patients. It is especially associated with the peripartum state[75,78] and has been associated with some connective tissue diseases, such as fibromuscular dysplasia.[79,80]

The gold standard for diagnosis of SCAD is invasive coronary angiography. Newer imaging modalities such as computed tomography angiography, intravascular ultrasound, and optical coherence tomography are used as an adjunct to increase sensitivity.[72,81,82] Due to the underdiagnosis or misdiagnosis of SCAD, together with the common initial presentation of SCAD as STEMI or unstable ventricular arrhythmia in more than half of patients,[83] it is probably an underappreciated cause of SCD in the community.

In the Massachusetts General Hospital SCAD registry of 102 patients presenting with SCAD or referred after a SCAD episode, 14% experienced aborted SCD, and half underwent ICD placement.[84] The SCD rate was likely higher in this registry than the usual incidence, due to MGH's being a referral center for SCAD patients. Of the aborted SCDs, 7 (50%) occurred out of hospital, and 4 (29%) in the emergency department. In that series, risk factors for SCD included the peripartum state, tobacco use, and presentation with STEMI. ICD implantation in patients who did not suffer aborted SCD and who do not meet other criteria for ICD placement is not currently supported by literature, and carries some risk.[84]

Anomalous coronary arteries

Coronary arterial anomalies are found in 0.9% of patients in large angiographic data series[85] and are responsible for approximately 12% of SCD in athletes in the United States.[86] There are multiple variants of anomalous coronary arteries. Many classification schemes have been developed, although no single classification scheme is universally employed. As seen in Fig. 5, one frequently used method of classification classifies anomalies by the potential paths the coronary artery that arises from the contralateral sinus of Valsalva may take to its perfusion territory: pre-pulmonic, retro-aortic, inter-arterial, trans-septal, retro-cardiac.[87]

Most coronary artery anomalies are not hemodynamically significant, and often are benign findings usually discovered during cardiac catheterization for acute coronary syndrome or stable atherosclerotic disease. In contrast, some anomalies are life threatening. Hemodynamically significant coronary artery anomalies include coronary origin from the pulmonary artery (which is quite serious) or from the opposite sinus of Valsalva or opposite coronary artery (i.e., left or right anomalous coronary) with an intra-arterial or intramural course.[88]

Episodic myocardial ischemia resulting in fatal arrhythmias from scar formation due to chronic ischemia is believed to be the main cause of SCD in coronary artery anomalies.[89] However, in a study of 30 patients with left or right anomalous coronary arteries diagnosed at post-mortem examination, a myocardial substrate for arrhythmia was identified in only a minority of individuals with SCD, and hence a single episode of acute ischemia from obstruction or acute angle closure may lead to SCD in many such patients.[90] An example of this was famed basketball player "Pistol" Pete Maravich, who had a single coronary artery and suffered sudden cardiac death despite playing for many years as an elite athlete.[91]

Treatment of patients with coronary anomalies who survive aborted SCD is usually accomplished via surgery, which varies depending on the course of the anomalous vessel. Some surgical techniques include unroofing of an intramural segment of the artery, reimplantation elsewhere in the aortic root, and coronary artery bypass grafting for symptomatic intra-arterial left main or intramural right coronary arteries.[92]

Conclusion

NA-SCD is an important but commonly underestimated cause of death. Early and late post - MI complications play an important role in its etiology. Other less common causes are coronary aneurysms, spontaneous dissections, and congenitally anomalous coronary anatomy. More research is needed to further delineate other etiologies, predictors, risk

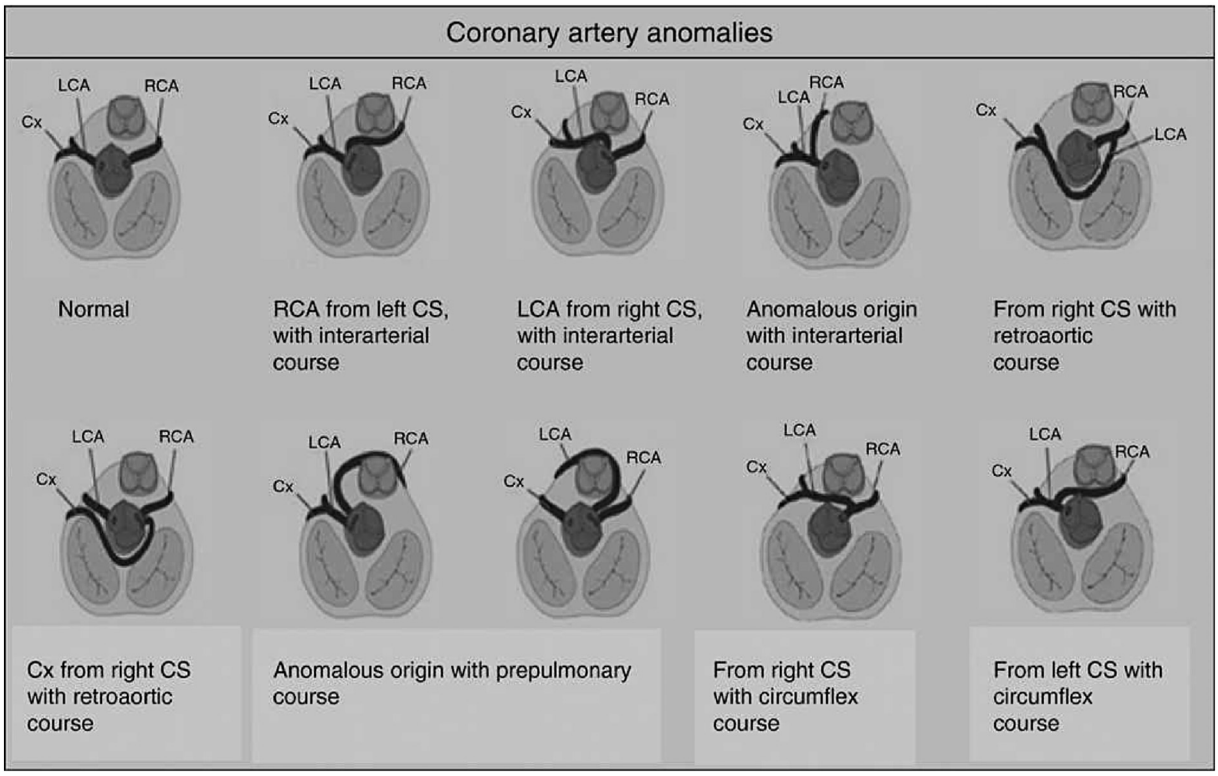

Fig. 5. Schematic representation of the possible paths by which the coronary arteries can connect with the opposite coronary cusps (Adapted with permission from Machado M et al., Benign and pathological electrocardiographic changes in athletes [Article in English, Portuguese] Rev. Port Cardiol. 2015 Dec;34(12):753–70.). CS = Coronary sinus, Cx = Circumflex artery, LCA = Left coronary artery, RCA = Right coronary artery.

factors, screening techniques, and preventive/abortive strategies for NA-SCD.

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