Journal of Cardiology xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Review

Idiopathic ventricular arrhythmias
Relevance to the anatomy, diagnosis and treatment

Takumi Yamada (MD, PhD)*

Division of Cardiovascular Disease, University of Alabama at Birmingham, Birmingham, AL, USA

ARTICLE INFO

Article history:
Received 22 May 2016
Accepted 24 May 2016
Available online xxx

Keywords:
Idiopathic
Ventricular tachycardia
Premature ventricular contraction
Electrocardiogram
Treatment

ABSTRACT

Idiopathic ventricular arrhythmias (VAs) are ventricular tachycardias (VTs) or premature ventricular contractions (PVCs) whose mechanisms are not related to myocardial scar. Idiopathic VAs occur most commonly without structural heart disease, but can occur with structural heart disease. Imaging tests, such as echocardiography, nuclear test, and cardiac magnetic resonance imaging, are helpful for excluding any association of an idiopathic VA occurrence with myocardial scar. Since catheter ablation emerged, the sites of idiopathic VA origins, commonly endocardial but sometimes epicardial, have been increasingly recognized. Idiopathic VAs usually originate from specific anatomical structures, and exhibit characteristic electrocardiograms based on their anatomical background. Idiopathic VAs are basically benign, but they require medical treatment or catheter ablation when idiopathic VAs are symptomatic, incessant, or produce left ventricular dysfunction. This review describes the up-to-date information on the prevalence of idiopathic VA origins relevant to the anatomy, and diagnosis, and treatment of idiopathic VAs.

© 2016 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Contents

Introduction

Idiopathic ventricular arrhythmias (IVAs) are ventricular tachycardias (VTs) or premature ventricular contractions (PVCs) whose mechanisms are not related to myocardial scar. IVAs occur commonly without structural heart disease (SHD), but can occur with SHD [1,2]. Classically, VTs originating from the right ventricular outflow tract (RVOT) and left posterior fascicle are well known as IVAs. However, since catheter ablation emerged, IVAs originating from other endocardial and also epicardial sites have been increasingly recognized (Fig. 1). IVAs usually originate from the specific anatomical structures, and exhibit characteristic electrocardiograms based on their anatomical background. Basically, IVAs are not life threatening, but are often symptomatic and also can cause tachycardia-induced cardiomyopathy [3,4]. Therefore, it is important for cardiologists to update their knowledge about IVAs. This review describes the up-to-date information on the prevalence of IVA origins relevant to the anatomy, and diagnosis, and treatment of IVAs.

* Correspondence to: Division of Cardiovascular Disease, University of Alabama at Birmingham, FOT 930A, 510 20th Street South, 1530 3rd Ave S, Birmingham, AL 35294-0019, USA. Tel.: +1 205 975 2404; fax: +1 205 996 5857.
E-mail address: takumi-y@fb4.so-net.ne.jp

http://dx.doi.org/10.1016/j.jjcc.2016.06.001
0914-5087/© 2016 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Please cite this article in press as: Yamada T. Idiopathic ventricular arrhythmias. J Cardiol (2016), http://dx.doi.org/10.1016/j.jjcc.2016.06.001

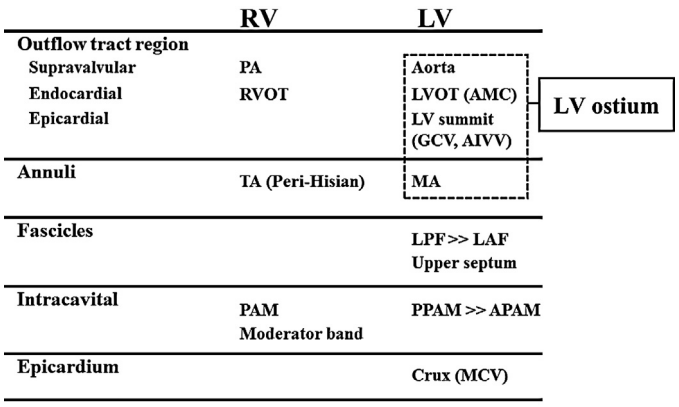

Fig. 1. Idiopathic ventricular arrhythmia origins. AIVV, anterior inter-ventricular vein; AMC, aorto-mitral continuity; APAM, antero-lateral papillary muscle; GCV, great cardiac vein; LAF, left anterior fascicle; LPF, left posterior fascicle; LV, left ventricle; LVOT, LV outflow tract; MA, mitral annulus; MCV, middle cardiac vein; PA, pulmonary artery; PAM, papillary muscle; PPAM, postero-medial papillary muscle; RV, right ventricle; RVOT, RV outflow tract; TA, tricuspid annulus.
Source: This figure was modified from Yamada [9], with permission.

Prevalence of IVA origins relevant to the anatomy

The sites of IVA origins have been identified by electrophysiological mapping and confirmed by successful catheter ablation. The most common site of IVA origins is the ventricular outflow tract [1,5]. IVAs originate more often from the RVOT than the left ventricular outflow tract (LVOT). In the RVOT, the septum is a more common site of IVA origins than the free wall. The most common site of IVA origins in the LVOT is the aortic root followed by the sites underneath the aortic coronary cusps (ACCs) (Fig. 2A) [6,7]. In particular, the site underneath the left coronary cusp (LCC) is termed the aorto-mital continuity (AMC). The mitral annulus (MA) is also one of the major sites of IVA origins [8,9]. The antero-medial aspect of the MA may overlap with the AMC. Anatomically, the aortic and mitral valves are in direct apposition and attach to the elliptical opening at the base of the left ventricle (LV) known as the LV ostium [10,11] (Fig. 2A). Because there is no myocardium between the aortic and mitral valves (fibrous trigone), most idiopathic LV VAs can originate from along the LV ostium. The LV myocardium comes in direct contact with the aorta at the base of the ACCs (Fig. 2A). When IVAs arise from the most superior portion of the LV ostium (the aortic sinus of Valsalva), they can be ablated within the base of the ACCs. It has been reported that some IVAs can be ablated from the junction (commissure) between the left and right coronary cusps (L-RCC) [12]. In these VAs, catheter ablation from underneath the ACCs is often required for their elimination. Anatomically, the superior end of the LV myocardium makes a semicircular attachment to the aortic root at the bottom of the right and left coronary cusps. However, because of the semilunar nature of the attachments of the aortic valvular cusps, the superior end of the LV myocardium is located underneath the aortic valves at the L-RCC (Fig. 2A). Therefore, IVAs that can be ablated at the L-RCC should be classified into the same group as the VAs that can be ablated within the ACCs. In this setting, these IVAs may be defined as IVAs arising from the aortic root [7]. It has been reported that IVAs can rarely be ablated from within the non-coronary cusp of the aorta (NCC) [7,13,14]. Spatially, the aortic root occupies a central location within the heart, with the NCC anterior and superior to the paraseptal region of the left and right atria close to the superior atrioventricular junctions (Fig. 2B) [11]. In healthy human hearts, the NCC is adjacent to the atrial myocardium on the epicardial aspect and the NCC does not directly come in contact with the ventricular myocardium (Fig. 2B). Indeed, atrial tachycardias can be ablated from within the NCC. However, the clinical observation that a non-coronary sinus of Valsalva aneurysm can rupture into the right ventricle (RV) as well as the right atrium supports the assumption that the NCC may be attached to the ventricular myocardium where IVAs can arise from [13]. IVAs can arise from the pulmonary artery with a ventricular myocardial extension from the RVOT [15]. It should be noted that ventricular myocardial extensions never occur in the aorta [11].

IVAs can originate from the atrioventricular annuli including the MA [8,9] and tricuspid annulus (TA) [16]. IVAs originating from the MA and TA account for 5% and 8% of all IVAs, respectively. MA VAs can originate from any of the regions along the MA, but the antero-lateral and postero-septal aspects of the MA are the most common and second most common sites of MA VA origins, respectively [8,9]. TA VAs can originate from any regions along the TA, but more often originate from the septal aspect, especially in the antero-septal or para-Hisian region than the free wall [16].

IVAs can arise from the intra-cavital structures including the papillary muscles (PAMs) [17–21] and moderator band (MB) [22]. PAM VAs account for approximately 7% of patients with IVAs [17–21]. LV PAM VAs are known to arise more commonly from the postero-medial PAM than the antero-lateral PAM [19]. The sites of the PAM VA origins are limited to the base of the PAMs. IVAs can rarely originate from the PMs in the RV [21]. IVAs can arise from all 3 RV PAMs, but half of them arise from the septal PAM [21]. It has been recently reported that the MB, although rarely, can be a source of IVAs including PVCs, VTs, and ventricular fibrillation [22]. Anatomically, the MB is considered to be a part of the septomarginal trabeculation, crossing from the septum to the RV free wall and supporting the anterior PAM of the tricuspid valve (Fig. 3A) [22].

IVAs can arise from the Purkinje network, most commonly from the left posterior fascicle followed by the anterior and septal fascicles [20,23,24]. The left anterior fascicle runs along the MA. The peripheral Purkinje network extends to the surface of the PAMs and MB. Therefore, these VAs have to be differentiated from IVAs originating from the PAMs, MB, and atrioventricular annuli.

IVAs arise commonly from the endocardial side, but can arise from the epicardial side [25] and rarely from the intramural site [26]. There are two major sites of origin of idiopathic epicardial VAs, such as the crux of the heart [27] and LV summit [28]. Anatomically, the crux of the heart is formed by the junction of the atrioventricular groove and the posterior inter-ventricular groove and corresponds roughly to the junction of the middle cardiac vein and coronary sinus, near the origin of the posterior descending coronary artery (Fig. 2C) [27]. A region of the LV epicardial surface that occupies the most superior portion of the LV has been termed the LV summit by McAlpine (Fig. 2D) [10,28]. The LV summit is bounded by the left anterior descending coronary artery and left circumflex coronary artery. This region near where the great cardiac vein (GCV) ends and the anterior inter-ventricular cardiac vein begins is one of the major sources of epicardial IVAs. The LV summit is bisected by the GCV into an area lateral to this structure that is accessible to epicardial catheter ablation (the *accessible area*) and a superior region that is inaccessible to catheter ablation due to the close proximity of the coronary arteries and thick layer of epicardial fat that overlies the proximal portion of these vessels (the *inaccessible area*) [28]. The prevalence of LV summit VAs has been reported to account for 12% of idiopathic LV VAs. Among these VA origins, 70%, 15%, and 15% of them have been identified within the GCV, accessible area, and inaccessible area, respectively.

Please cite this article in press as: Yamada T. Idiopathic ventricular arrhythmias. J Cardiol (2016), http://dx.doi.org/10.1016/j.jjcc.2016.06.001

Download English Version:

https://daneshyari.com/en/article/5614847

Download Persian Version:

https://daneshyari.com/article/5614847

Daneshyari.com