

Membranous Ventricular Septal Aneurysm: to Be or not to Be



Aneurisma del septo membranoso ventricular: ser o no ser

To the Editor,

We have read with interest the article published by Guerrero Márquez et al¹. However, in the wake of that reading, we would like to make a few comments that we believe to be important.

Perimembranous ventricular septal defect (VSD) is commonly associated with an aneurysm of the membranous septum. However, the term *aneurysm of the membranous septum* is usually a misnomer, since the tissue surrounding the defect is usually derived from the tricuspid valve leaflets rather than from the membranous septum,² as it seems to be in the case reported by the authors. This mechanism tends to decrease the functional size of a perimembranous VSD, closes about 50% of them before the patient is aged 10 years, and smaller defects are more likely to be closed than larger ones. For this reason, it would have been interesting to rule out the existence of early systolic ejection sounds or an unusual decreasing systolic murmur in childhood as a way to screen for underlying congenital heart disease.

In contrast, a membranous septum tissue is defined as a well-developed and thickened fibrous tissue. The absence of myocardium of this structure may lead to aneurysm-like behavior and bulging into the right ventricle. Unlike the perimembranous VSDs associated with a restrictive tricuspid tissue, the membranous septum has no attachments to the tricuspid valve or the tricuspid valve mechanism and may be or not be associated with perforations and therefore interventricular shunts. Such aneurysms may promote tricuspid insufficiency, aortic valve prolapse, right ventricle outflow tract obstruction, embolic events, and bacterial endocarditis.³ In contrast, complications are rare when an excess of tricuspid fibrous tissue acts as a closure mechanism of a perimembranous VSD.²

The bundle of His begins at the atrioventricular node, located at the inferior interatrial septum, and passes into the subendocardium through the right fibrous trigone to the interventricular septum, after which it divides into right and left limbs that pass along either side of the septum, branching to the walls of the ventricles. The course of the bundle of His in patients with perimembranous VSDs passes down the posterior edge of the defect and bifurcates on its inferior border.⁴ Meanwhile, in patients with true aneurysms of the membranous septum, the bundle of His does not differ from that of normal individuals.⁵ The mechanism of the conduction disorders, seen in patients with perimembranous VSDs, may be related to the fibrotic response that occurs when the VSD closes with the tricuspid tissue. In contrast, a true aneurysmal sac may favor mechanical strain, traction and

compression on the conduction pathways at the level of the neck of the aneurysm, leading to fibrosis and progressive destruction of the conduction system.⁶ Both conditions may promote conduction disturbances or the occurrence of bundle branch blocks.

Regarding clinical presentation, congenital complete heart blocks may be intermittent when first detected, but usually become persistent in later childhood or adulthood, escaping notice because of a high ventricular rate and the absence of symptoms.⁷ Despite the very low frequency of complete atrioventricular block seen in patients with “aneurysms of the membranous septum”, as stated by the authors, we must keep this entity in mind, especially in young patients with unexplained syncope.

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