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EDITORIAL

Effects of atrial septal defects on the cardiac conduction system

Jin-Hua Kang, Hong-Yan Wu, Wen-Jie Long

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Abstract

The case report presented in this edition by Mu *et al*. The report presents a case of atrial septal defect (ASD) associated with electrocardiographic changes, noting that the crochetage sign resolved after Selective His Bundle Pacing (S-HBP) without requiring surgical closure. The mechanisms behind the appearance and resolution of the crochetage sign remain unclear. The authors observed the disappearance of the crochetage sign post-S-HBP, suggesting a possible correlation between these specific R waves and the cardiac conduction system. This editorial aims to explore various types of ASD and their relationship with the cardiac conduction system, highlighting the diagnostic significance of the crochetage sign in ASD.

Key Words: Atrial septal defects; Cardiac conduction system; Crochetage sign; Electrocardiogram; Selective His bundle pacing

Core Tip: Atrial septal defect (ASD) is characterized by its slow clinical progression, often remaining asymptomatic in children and young adults until diagnosed later in life. Early screening and timely treatment for ASDs are essential. The heightened sensitivity of the crochetage sign in ASDs emphasizes the need for cost-effective, rapid, and non-invasive body surface electrocardiography for early screening, particularly in underserved areas. This editorial seeks to illustrate the impact of ASD on the cardiac conduction system, specifically focusing on the crochetage sign, through a discussion of the recently published case report.

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INTRODUCTION

The case report presented in this edition by Mu *et al*[1], highlights the presence of a crochetage sign in atrial septal defect (ASD) potentially associated with the His-Purkinje system within the cardiac conduction system. This editorial reviews the different types of ASDs, their electrocardiographic features, and their relationship to the cardiac conduction system.

ASD is characterized by an underdeveloped or abnormal secondary or primary septum in the atrial septum, creating an abnormal passageway between the left and right atria. This results in atrial-to-horizontal shunts of varying severity and direction. ASD is primarily congenital and represents a common adult congenital heart disease, accounting for approximately 10% of congenital heart diseases and 30% to 40% of adult congenital heart diseases. The occurrence of ASD is more prevalent in females than in males, and there is a noted familial tendency, as first-degree relatives have a relatively high risk ranging from 2% to 6%[2-4]. Although most ASDs are sporadic, genetic syndromes such as Holt-Oram and Ellis van Creveld have shown a genetic association with these atrial defects. Mutations in the *NKX2-5* gene, located on the cardiac homologous chromosome and essential for normal cardiac development, have been linked to autosomal dominant familial ASD and atrioventricular conduction block[5,6].

DIAGNOSIS AND CLASSIFICATION OF ASD

ASD is characterized by slow clinical progression and often remains asymptomatic in children and young adults. Electrocardiograms, cardiac ultrasound, and right heart catheterization are primary methods for diagnosing ASD. As individuals age, symptoms such as exercise intolerance, arrhythmias, right ventricular dysfunction, and pulmonary hypertension become more prevalent. Symptoms like fatigue, shortness of breath, palpitations, and chest tightness may occur, along with manifestations of heart failure, which can lead to a reduced survival rate. The severity of ASD is determined by hemodynamic alterations associated with the size of the defect, left and right ventricular compliance, as well as the resistance within the systemic and pulmonary circulatory systems[7,8]. In instances of considerable left-to-right shunting, an increase in pulmonary blood flow may result in pulmonary congestion along with enlargement of the right atrium and ventricle, attributed to the heightened volume load on the right side of the heart. Sustained high blood flow within the pulmonary circulation can elevate pulmonary artery pressure and lower pulmonary vascular compliance, ultimately leading to persistent pulmonary hypertension. Should the pressure within the right atrium or ventricle surpass that of the left side, the left-to-right shunt may reverse to a right-to-left shunt, leading to symptoms such as cyanosis[7,9,10].

ASD can be classified into four main types: Ostium secundum, ostium primum, sinus venosus, and coronary sinus. The type that occurs most frequently is ostium secundum ASD, comprising 75%-80% of cases, followed by ostium primum ASD (15%-20%), sinus venosus ASD (5%-10%), and the less common coronary sinus defect ASD (< 1%). Primum, sinus venosus, and coronary sinus ASDs often result in significant hemodynamic shunts and may require surgical closure at an early age[11]. ASDs of the ostium secundum type are situated in the atrial septum's central fossa ovalis and can be further classified into central, inferior, superior, and mixed defects. These defects may vary in size and shunt volume over time. The size of the defect, along with the diastolic filling properties or compliance of both the left and right ventricles, influences the direction and volume of blood flow across the ASD[7].

ASD AND CARDIAC CONDUCTION SYSTEM ABNORMALITIES

ASD is a congenital heart condition characterized by structural abnormalities in the heart chambers that disrupt the normal positioning of the heart's electrical conduction system. Typically, the right ventricle and right bundle branch are affected, leading to abnormal electrocardiograms, such as conduction blocks. Secundum ASDs, usually located higher in the heart, primarily impact the interjunctional bundles. Over time, these changes can also affect the His-Purkinje system, resulting in right axis deviation and incomplete right bundle branch block[12]. In contrast, ostium primum ASDs are located in the lower section of the atrial septum and can exhibit a left axis deviation. They can cause delays in signal transmission to the atrioventricular (AV) node due to involvement of the anterior mitral leaflet cleft or tricuspid septal cleft[13]. Sinus venosus ASDs may exhibit atrial ectopic pacing and P-wave inversion in leads II, III, and aVF, suggesting sinus node abnormalities[14,15]. If ASDs involving the coronary sinus extend to the opening area of the coronary sinus, the position of the AV node may shift posteriorly, potentially leading to conduction disturbances[16]. These pathological changes may result from developmental disorders and structural abnormalities in the conduction system caused by ASDs, as well as hemodynamic abnormalities in the septum and central fibrous body[17].

Due to the mild early symptoms and subtle signs of the disease, many patients may not recognize the need for timely treatment. Early diagnosis and prompt atrial septal repair are crucial for improving patient outcomes. Thus, there is a critical need for early screening and diagnostic markers for atrial septal defects. Cardiac ultrasound is essential for

diagnosing ASD as it offers vital details regarding the size and location of the defect, the direction of shunting, and dilation of the right heart, as well as diastolic septal inversion indicating hemodynamic significance. Additionally, cost-effective and accessible body surface electrocardiography plays a vital role in early screening.

ASD AND CROCHETAGE SIGN

Electrocardiograms of patients with ASDs often display various abnormalities, including incomplete or complete right bundle branch block, prolonged PR interval, atrioventricular block, atrial arrhythmia, right atrial and right ventricular hypertrophy, and right deviation of the frontal electrical axis. However, electrocardiography alone is not sufficiently sensitive or specific for diagnosing ASDs. In 1959, Rodriguez-Alvarez *et al*[18] observed tangential tracings on the apical part of the QRS wave cluster in 11 patients with ASDs, introducing the concept of the Crochetage R wave, though it initially received little attention. Since Heller's description of the Crochetage R wave as a distinctive electrocardiography (ECG) diagnostic marker for ASD, this feature has gained recognition among clinicians. The Crochetage R wave is characterized by a notch in the ascending branch or apex of the R wave in the inferior wall leads, typically appearing within 80 ms following the initiation of the QRS complex and creating a distinctive M-shaped pattern in the ascending branch or apex of the R wave[19]. The Crochetage sign in leads II, III, and aVF has a sensitivity of 57% and a specificity of 92% for diagnosing ASD[12]. These Crochetage R waves can manifest in a single inferior wall lead or in 2-3 inferior wall leads simultaneously. Sensitivity and specificity are particularly high when Crochetage R waves are present in all three inferior wall leads or in conjunction with incomplete right bundle branch block. After the surgical repair of ASDs, R-wave patterns frequently occur before the pattern of incomplete right bundle branch block fades in 35% of individuals[19,20]. Furthermore, the presence of the Crochetage sign is significantly correlated with ASD diameter and defect area, with patients exhibiting these waves often having larger defect areas compared to those without[12,21].

S-HBP AND THE DISAPPEARANCE OF CROCHETAGE SIGN

Due to the patient's ASD, who developed heart failure and exhibited atrial fibrillation witha risk of prolonged R-R interval, second-degree atrioventricular block, and incomplete right bundle branch block, selective His bundle pacing (S-HBP) was performed as a preliminary intervention. Currently, His bundle pacing (HBP) is considered the most physiological method for pacing, since it achieves synchronization of ventricular excitation by directly stimulating the His bundle. This stimulation ensures that the heart's electrical activity is predominantly conducted through the His-Purkinje fiber system, facilitating synchronized biventricular contractions. In certain patients with conduction abnormalities, HBP can correct established conduction issues[22,23]. There are two types of HBP: S-HBP and non-selective His bundle pacing (NS-HBP). S-HBP differs from NS-HBP in that while NS-HBP captures both the right and left portions of the His bundle and the membranous septal ventricular muscle simultaneously through high-output pacing, S-HBP operates at a lower threshold than that of the peripheral ventricular myocardium. By using low-output pacing, S-HBP eliminates the involvement of the membranous septal ventricular muscle, correcting the block solely by stimulating either the right or left aspect of the His bundle[23].

Considering the potential risks associated with implanting an atrial septal occluder device, which could further impact the cardiac conduction system, the attending physician opted for S-HBP as the initial treatment approach. Following this procedure, the postoperative surface electrocardiogram showed the disappearance of the crochetage sign. The mechanism underlying the crochetage sign in ASDs remains unclear. However, most scholars suggest it may be related to excessive cardiac pressure and volume load, as well as the anatomical changes observed in individuals diagnosed with ASD. The resolution of the crochetage sign indicates improved patient prognosis and a reduction in symptoms. S-HBP has the potential to enhance synchronized ventricular contraction, correct hemodynamics, alleviate cardiac pressure and volume load, and restore the functionality of the cardiac conduction system in ASD patients. Thus, the implementation of S-HBP may lead to the resolution of the crochetage sign, accompanied by a reduction in patient symptoms and an improvement in long-term prognosis. The late follow-up of the disappearance of the crochetage sign after S-HBP, as reported by Mu *et al*[1], supports this hypothesis. Nevertheless, additional studies on the effects of S-HBP on ASD are still needed.

CONCLUSION

Early screening for ASDs is crucial, along with subsequent treatment. The high sensitivity of the crochetage sign in ASDs highlights the importance of using cost-effective, rapid, and non-invasive body surface ECG for early screening, particularly in disadvantaged areas. However, it is important to note that the crochetage sign may yield false positives and has limitations in assessing the specific type of defect in patients with ASD. Therefore, a definitive diagnosis and accurate classification of ASD require further clarification through additional modalities, such as echocardiography. Moreover, the application of S-HBP in ASD patients who have experienced cardiac conduction blockages and congestive heart failure has the potential to significantly benefit many individuals with ASDs, improving their quality of life, functional status, and overall survival. Further research is warranted to explore the direct relationship between the presence and resolution of the crochetage sign in ASDs and cardiac conduction disorders.

FOOTNOTES

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