

# Atrial Septal Aneurysm and Atrial Arrhythmia: A Review of The Literatures

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## ABSTRACT

This paper is a brief review of literatures about the relationship between atrial septal aneurysm and atrial arrhythmia.

**Keywords:** Atrial Septal Aneurysm; Atrial Arrhythmia

## Introduction

Atrial Septal Aneurysm (ASA). is a saccular deformity located in the atrial septum [1]. The first report on ASA was published by Lang and Posselt in 1934. It is a localized bulging of the interatrial septum and the diagnostic criteria is usually a protrusion >6 mm into the right atrium or left atrium or both atriums [2]. The pathogenesis of atrial septal aneurysm might be explained by an abnormal structure of the interatrial septum, by a change in the normal inter-atrial pressure gradient, or by both. Although ASA is a well-recognized cardiac anomaly, the clinical significance of ASA has not yet been fully elucidated. The relationship between ASAs and cardiac arrhythmias have been evaluated in many studies and case reports [3-7]. The prevalence of supraventricular tachycardia in a 50 cases study has been reported to be 40% by Schneider [5]. While the prevalence of supraventricular tachycardia has been reported to be 57% by Longhini [7] and 45% by Morelli [6] in the smaller series.

It is unknown whether the ASA itself or associated structural and functional abnormalities are related to the pathogenesis of atrial arrhythmias [4-6,8]. There are only few case reports or retrospective studies that analyse the association between ASA and atrial arrhythmias [9-16]. Some studies [6,15,16] showed a positive association between ASA and supraventricular arrhythmias, whereas others [5,17,18] did not confirm the relationship. Deveci

[15] reported that the prevalence of supraventricular arrhythmias was significantly higher in a group of 66 young patients with echocardiographic diagnosis of ASA without comorbidities in comparison to a control group of 62 healthy volunteers (43.9 vs. 8.1%,  $P<0.001$ ). In the study by Morelli [6], the group of 20 patients with ASA showed a higher prevalence of supraventricular arrhythmias with respect to the control group of 19 healthy individuals. On the contrary, the prospective study by Schneider [5], including 50 consecutive patients with ASA, did not show a clear association between supraventricular arrhythmias and the presence of ASA.

Similar results were also obtained in the study by Miga [17], in which the prevalence of ASA was not significantly different in a group of 30 infants with supraventricular arrhythmias compared with a control group of 30 infants without any arrhythmias (13% vs. 7%, difference not significant;  $n=30$ , respectively). Vincenti [19] have noted that nearly half of the paroxysmal atrial fibrillation attacks were induced following a supraventricular ectopic beat. The study of Deveci [15] showed that the most common supraventricular tachycardias in patients with ASA are frequent supraventricular extrasystoles, which enrolled greater than 2000 supraventricular extrasystoles were detected in 27.3% of ASA patients within 24 hours. It is confirmed that ASA can act as an arrhythmic focus,

generating focal atrial tachycardias [20]. What is the proarrhythmia mechanism of ASA? Evidence given by two studies that sinus impulses are conducted in a heterogeneous and anisotropic manner within the atrial tissue due to an irregular physical structure and differentiated microstructure of the atrial myocardium [21,22]. In ASA patients right and left atrial appendage function are impaired, the biatrial dysfunction may cause arrhythmia [23].

Morelli [6] confirmed a clear etiopathogenetic relationship between a large ASA and re-entry related atrial tachycardia, they believed the re-entry mechanism could be dependent on an electro-anatomical barrier and/or different electrophysiological properties between ASA and the remaining atrial septum. Deveci [15] believed that the heterogeneity of atrial macro- and microgeometry caused by ASA may lead to changes in electrophysiological dynamics of the atrial myocardium, which in turn leads to more frequent atrial extrasystoles and induces atrial fibrillation attacks. Deveci [15] also found that the P-wave dispersion was significantly increased in patients with ASA compared to the control group. Prolongation of conduction has been documented both by 12-lead surface electrocardiogram and by a signal-averaged electrocardiogram recording to have a longer P-wave duration [24]. In the opinion of Muser D and colleague, the presence of ASA is indeed a structural element favoring the occurrence of atrial tachycardia and this is probably due to the abnormalities of both functional and anatomical characteristics of the atrium, including dispersion of the action potential and appearance of re-entrant circuits [16]. In all, although the relationship between ASA and supraventricular tachyarrhythmias is also controversial, more and more evidences tend to support the proarrhythmia mechanism of ASA.

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