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Original Research Article

ECG Changes of Sinus Tachycardia in young Patient is the Manifestation of Atrial Septal Aneurysm - An Observational Study

Authors
Dr Suman Gupta M.D[1], Dr Bhanu Choudhary M.D[2]
[1]Anaesthesia. Associate Professor, Department of Anesthesia, G.R.M.C. Gwalior (M.P) India
[2]Professor and Head of Department of Anaesthesia G.R.M.C, Gwalior (M.P) India
Corresponding Author
Dr Suman Gupta
Address: 11-Helipad Colony, Lashkar, Gwalior M.P. India Pin 474009
Email: *sumangupta02@rediffmail.com*, Phone NO. 09981962454

Abstract
Atrial septal aneurysm is one the cardiac congenital malformation which either confines itself to the thin fibrous remnant sheet of Fossa ovalis or involve whole of the septum between right and left atrium .This aneurysmal septum looms out either towards right or left atrium during cardiac cycle. Once considered to be a rare now being more frequently diagnosed in patient with wide spread use of 2-D Transthoracic Echocardiography for screening the preanesthetic patient. It is usually asymptomatic, but may be associated with other congenital anomaly like Patent Foramen Ovale, Patent Ductus Arteriosus, or may present with atrial arrhythmias or cryptogenic strokes.
Keywords: *Sinus Tachycardia, Atrial Septal Aneurysm, Transthoracic Echocardiography, Congenital Anomaly, Anaesthesia.*

Background

Atrial septal aneurysm is one the cardiac congenital malformation which either confines itself to the thin fibrous remnant sheet of Fossa ovalis or involve whole of the septum between right and left atrium .This aneurysmal septum is saccular defect that thrusts out either towards right or left atrium or in both directions during cardiac cycle[1] .

Although it was considered to be of rarest, however, with the advent of 2-D transthoracic echocardiography (TTE) and the widespread use of transesophageal echocardiography (TEE) for screening preoperatively it is being more and more diagnosed in patients.[2] The TTE delineate around 2% of patients are having ASA[1]. It is commonly present along with other congenital heart diseases like Patent foramen ovale(PFO), Patent ductous arteriosus (PDA), Atrial septal defect(ASD), Ebstein's anomaly Ventricular septal defect (VSD), Valvular prolapse (VP), and Tricuspid atresia, as well as with acquired heart diseases i.e, cardiomyopathy, systemic and pulmonary hypertension, ischemic heart disease, valvular disease arrhythmias and thrombus formation. [2]

Material and Methods

A young female aged 17 year came for pre anaesthetic consultation scheduled for excision of fibroadenoma breast bilaterally. Apart from surgical problem patient did not have any other significant history related to cardiovascular, respiratory or central nervous system .i.e palpitation, breathlessness, headache or syncopal attack. Clinical examination reveals regular pulse but tachycardia with Heart rate of 132bpm, blood pressure of 110/70mmHg. Peripheral pulses were normal. On auscultation normal Heart sounds S1 S2 were heard, unable to hear any audible systolic click in pulmonary area pathognomic of ASA, may be due to prevailing sinus tachycardia. Routine lab rotary examination i.e hemoglobin 10.4gm%,, serum urea 14mg/dl%, serum creatinine 0.7mg/dl coagulation profile were all within normal range EKG reveal sinus tachycardia with T, wave inversion in lead v1, v2 ,v3 as shown in(figure1,2..) Chest Xray shows normal cardiac silhouette, no cardiomegaly, with clear lung fields. Patients was referred to cardiologist for expert review.TTE done reveals normal size chambers ,valves, intravenicular septum was intact, intra atrial septum showing a bulge in right atrial cavity, signaling TYPE 1R ASA according to the new classification of ASA as shown in figure. 3. No evidence of thrombus in any of the chamber, or associated other congenital anomaly was evident.

Knowledge of the anatomy and physiology of the congenital anamoly is important for choosing appropriate monitoring and inducing agents. Such patient usually come for repair of septum or closure device. As such in this case with TYPE 1R ASA, without any other congenital, we need to administer anaesthesia for non cardiac surgical procedure of excision of fibroadenoma. Induction done with inj. Midazolam 1mg, Fentanyl 2mcg/kg, Propofol 2mg/kg, Anaesthesia was maintained with variable concentration of Sevoflurane 1-4%. Patients received Oxygen 33% and Nitrous Oxide 67%in spontaneously breathing patients, with LMA in situ. Monitoring was done with NIBP, ECG, RR and HR. SPO2 Continuous capnography.

After the surgery, Sevoflurane was stopped. LMA was removed after eye opening and mouth opening to command. After removal of LMA patient was observed in recovery, then shifted to ward, and finally discharged on very next day.

Table 1.New Classification of ASA

Serial No.	Type of septal Aneurysm	Direction of movement of Aneurysmal Atrial Septum
1	Type 1R	ASA projects from the midline of the atrial septum to the RA throughout the entire cardiorespiratory cycle
2	Type 2L	the ASA protrudes from the midline of the atrial septum to the LA throughout the entire cardiorespiratory cycle
3	Type 3RL	the maximum deflection of the ASA is toward the RA and lesser toward the LA
4	Type 4LR	the maximal excursion of the ASA is toward the LA
5	Type 5	the ASA movement is bidirectional

ASA: Atrial Septal Aneurysm, LA : Left Atrium, RA: Right Atrium.

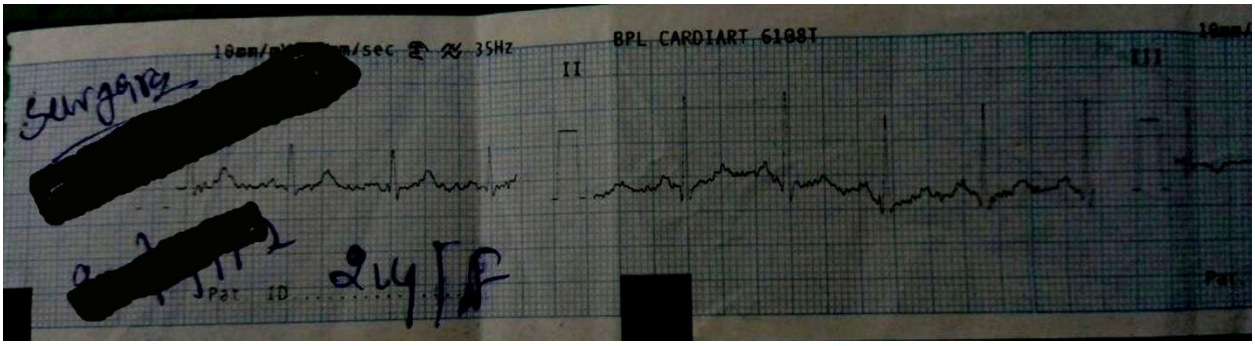

Figure 1 ECG leads I,II,III revealing sinus tachycardia

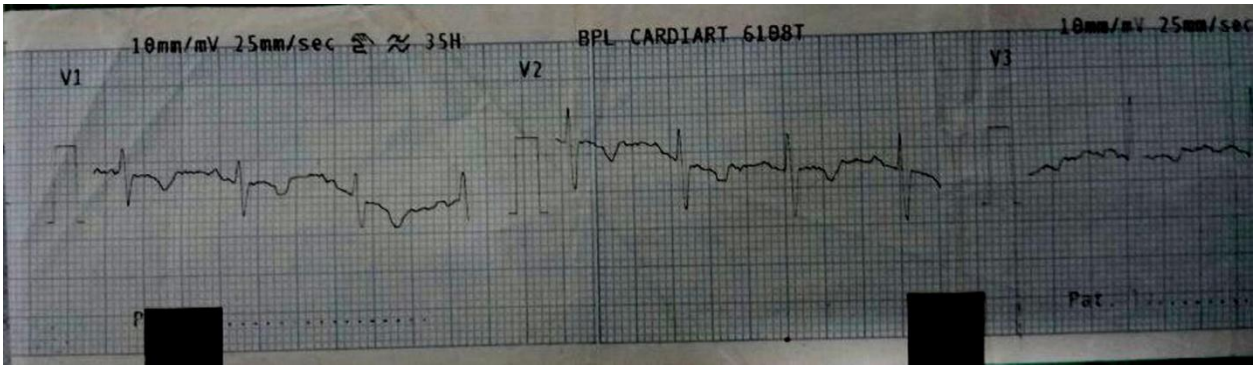

Figure 2 ECG showing T WAVE INVERSION in leads v1, v2, v3

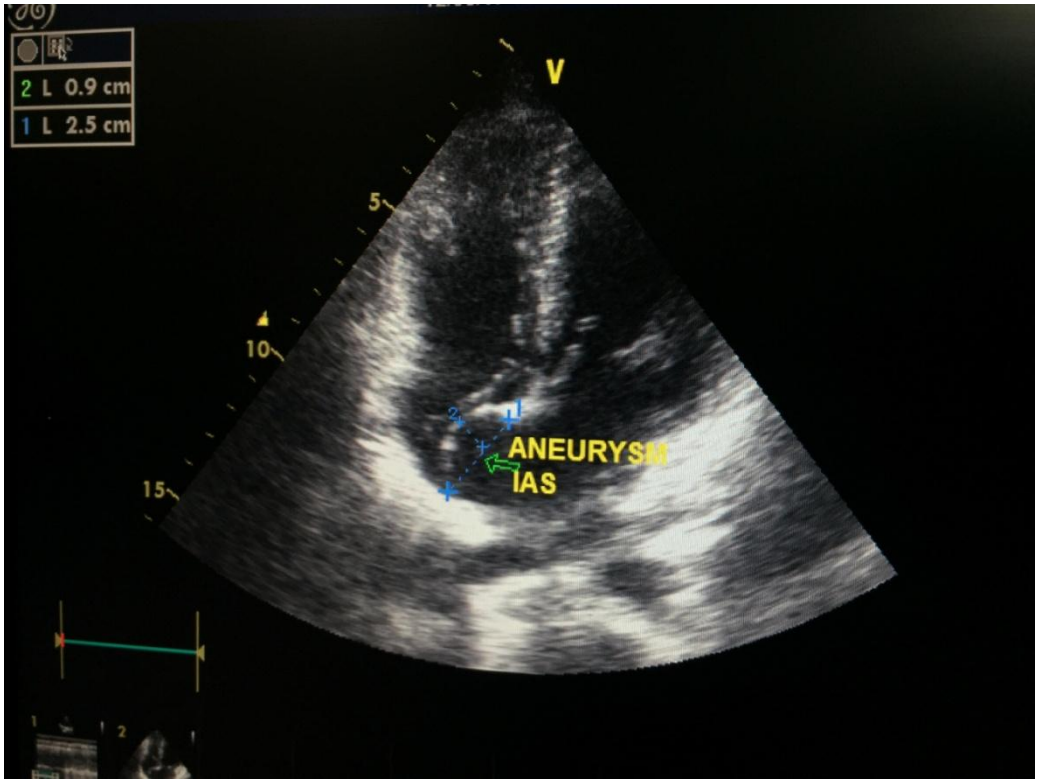

Figure.3.ASA bulging into right atrium (Type 1R) during cardiorespiratory cycle as seen in 2-Dimentional Transthoracic Echocardiography

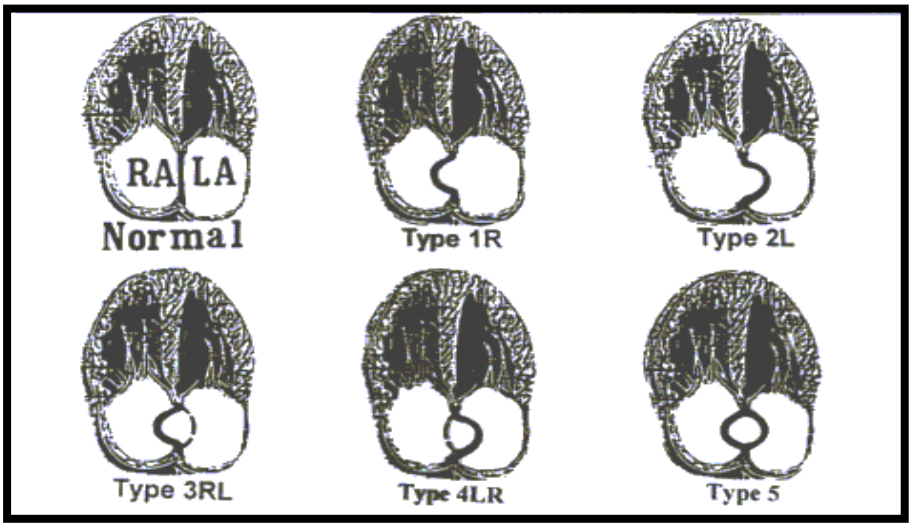

Figure 4.describing New classification of ASA by Alexander Olivares-Reyes et al[12]

Discussion

Atrial septum aneurysm (ASA) is a rare cardiac clinical entity that was first reported by Lang and Posselt in 1934[3]. It is an isolated deformity "saccular" like at the level of Fossa Ovalis which usually balloons out either to right or left atrial chambers, or to both sides at a time during the cardiac cycle. It is a noteworthy cardiac malformation, which is serendipitously diagnosed, and is a well known causative factor for cardioembolic stroke albeit there is no focus of thrombus in aneurysm itself or in left and right atrium when screened via TTE.[4]

Patients with ASA are generally asymptomatic and do not present with any of the electrocardiogram or clinical findings implicative of ASA.[5]. On auscultation a non ejection SYSTOLIC click may sporadically be audible possibly produced, as the Inter ASA protrudes and tenses within LA/RA chambers during cardiorespiratory cycle.[6]

Sometimes it may be associated with clinical variables like Cardiovascular embolism, Hypertension, Coronary Artery diseases, Diabetes Mellitus, valvular prolapses, arrhythmias, valvulopathies, PFO, ASD, [2, 5]

Scholz EP, Zitron E, Katus HA *et al.* reported a case of ASA with in electrocardiogram finding of right atrial enlargement and thereby suggests echocardiography even in asymptomatic patients with abnormal 12-lead electrocardiogram. This reported case presented with asymptomatic tachycardia when further reviewed by cardio-logist, shows T wave inversion in lead v_1 v_2 v_3 in ECG (Figure 2.), and ASA in TTE.(Figure 3)

Clinical Manifestations attributed to ASA are usually atrial arrhythmias, ventricular tachyca-rdia's[7]. These primary congenital malformations at fossa ovalis or involving the entire interatrial septum result in interatrial pressure difference generating tachyarrhythmia's.[7.8] At times Interatrial septal aneurysm itself behave as an arrhythmic focus, generating focal atrial tachycardias [1,9] In a study delineated by Hanley et al it was observed out of 80 patients 25% of them had atrial arrhythmias. Majority of the reported cases are congenital in nature and are detected incidentally. [1, 9]

ASA is also known to cause complications like arterial embolism, the presence of aneurysmal septum physically obstructs the blood flow during cardiac systole and advocates stasis of blood flow, generating a focus of minute LA clots and predisposes to systemic thrombo embolism.[9,8]

Mechanism of cardioembolic stroke could be right to left shunting or the thrombogenic properties of aneurysm itself.[4]

Transthoracic Echocardiography is the imaging modality of preference for ASA diagnosis, for routine echocardiography screening or during the event of cardioembolic cerebrovascular stroke and peripheral embolism. In Comparison to TTE, TEE is more sensitive in picking up ASA [8.9]

The presence of isolated and Uncomplicated ASA usually requires assurance and follow up apart from any specific treatment. A thorough evaluation need to be executed to rule out presence of thrombus in patients with aneurysm. Various Therapeutic disciplines for prevention of recurrent stroke in patients with atrial septal aneurysm are commencing medical therapy with antiplatelet agents, anticoagulants and surgical or percutaneous closure of the defect [4]

Various classification has been laid out since 1985, Longhini et al [10]., then by Hanley et al[1], In 1989 Roudaut et al.[11] In 1991 Pearson et al[9], to describe ASA, later In 1997 Alexander Olivares-Reyes et a,l[12] based on the direction and movement of ASA proposed a new classification in to 5 types as described in Table 1.

Summary and Conclusion

Thus the patient reported above had TYPE 1R type of ASA, without any prior thromboembolic events or headache. It was not possible to rule out the cause of rise in left atrial pressures leading to the development of ASA. No Clear guidelines exist regarding management of ASA, incidentally found ASA without any symptoms do not require any treatment. Though debate continues on

antiplatelet therapy with aspirin alone versus oral anticoagulation therapy with warfarin in patients with PFO. The Lausanne Study, observed hardly any difference in primary end points of recurrent stroke or mortality at the end of 2 years in aspirin alone versus warfarin alone treated groups [13]

Most reported cases of ASA are incidental findings [9]. ASA is increasingly gaining clinical significance as it is attributed cardio-embolic stroke and arrhythmias.

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Consent

Here we report a case of ASA in young patient without any symptomology, a written well informed consent was given by the patient for this purpose of publication.

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Conflict of Interest: Nil

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