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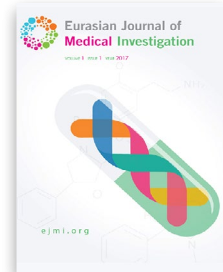

Research Article

Relationship Between Shunt Size and Right Lead Electrocardiographic Findings in Patients with Secundum Atrial Septal Defect

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Abstract

Objectives: Secundum atrial defect (ASDs) causes changing in cardiac hemodynamics dependent on the defect size and shunt volume including volume overload, stress extends to tissues in the cardiac conduction system and manifests as intra-atrial or intra-ventricular conduction delay. The aim of the study is to evaluate that ECG findings in patients with atrial septal defect can help determine the shunt rate.

Methods: This retrospective study included 74 patients with ASDs. Standard 12-lead ECG and right precordial electrocardiographic recordings (V3R, V4R, V5R, and V6R) were obtained from all patients. QT intervals were measured manually from ECG on paper prints. According to shunt ratio (Qp/Qs), the patients divided into two groups as <1.5 group 1; 1.5> Group 2.

Results: The mean ages of Group1 and Group2 were similar (p>0.05). There were no significant differences between the two groups between RA (right atrium) diameters, RV (right ventricle) diameter (p>0.05). V4RQTmax were 321.2±26.4 msec; 351.1±33.6 msec, respectively (p<0.05) and V5RQTmax were 319.2±22.6 msec; 350.1±32.3 msec, respectively, (p<0.05), which were found to be significantly different.

Conclusion: Were obtained that the right precordial leads could be used to estimate the shunt ratio of patients with secundum ASD.

Keywords: Electrocardiography; right precordial lead; secundum atrial defect

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Atrial septal defect (ASD) encompassed 10% of all congenital heart lesions[1] and represent the third most common congenital cardiac defect seen in adults.[2] Secundum ASD causes shunting of blood between the systemic and pulmonary circulations. Most young patients with ASD are asymptomatic during childhood and adolescence, even if the shunt volume is large.[3, 4] ASD is associated with the development of pulmonary hypertension, congestive heart failure, atrial arrhythmias, right heart volume overload and decompensation.[5–7]

ASD causes changing in cardiac hemodynamics dependent on the defect size and shunt volume.[3] In the myocardium of an atrium and ventricle continuously exposed to volume overload, stress extends to tissues in the cardiac conduction system and manifests as intra-atrial or intra-ventricular conduction delay. Furthermore, the degeneration of cardiac cells, including fibrosis by myocardial remodeling, pathologically modifies myocardial repolarization.[8, 9] The first report on cardiac mechano-electrical feedback in 1943.[10] The interaction between cardiac mechanics and elec-

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trophysiology has been well established and it is considered to be an important contributor to arrhythmogenesis in conditions with pathologically altered hemodynamics.[11] Prolonged repolarization in ASD patients who typically lack significant depolarization changes may be a probable model of the effect of volume overload on repolarization.[12, 13] The aim of the study is to evaluate that ECG findings in patients with atrial septal defect can help determine the shunt rate.

Methods

This retrospective study included 74 patients with secundum ASD diagnosed by echocardiography and catheter study in Türkiye Yüksek İhtisas Hospital. This study was carried out with the approval of the chief of clinic. All of the patients mean age had 42.05±15 581 (40 females and 34 males). All the patient information was de-identified and the study was purely descriptive and retrospective in nature with no clinical relevance for the involved patients.

Exclusion criteria had pulmonary venous return anomaly, left persistent superior vena cava, primary pulmonary hypertension, chronic obstructive pulmonary disease, essential hypertension, atrial fibrillation, pacemaker, left bundle branch block and systemic disease. Additionally, the patients taking QRS prolonging medications were also excluded.

ECGs had been readable with clearly defined monophasic T waves and disappearing or different U waves.

Standard 12-lead ECG and right precordial electrocardiographic recordings (V3R, V4R, V5R, and V6R) were obtained from all patients. QT intervals were measured manually from ECG on paper prints. QT interval was determined visually from the beginning of the earliest onset of the QRS complex to the end of T wave defined as the point where the tangent drawn at the steepest slope of the descending limb of the T wave intersects the isoelectric line. The longest QT distance is determined as maximum QT.

Echocardiography

Right ventricular (RV) dimensions were obtained from echocardiographic M-mode tracings and subsequently their Z-scores were calculated.

Maximum right and left ventricular (LV) inlet dimensions at end-diastole were obtained from apical four chamber images. Shunt ratio (Qp/Qs) was calculated by two-dimensional and Doppler echocardiographic measurements.

According to shunt ratio (Qp/Qs), the patients divided into two groups: <1.5 group 1; 1.5> Group 2. Both groups age, RA (right atrium), RV (right ventricle), SPAP (systolic pulmonary artery pressure and V1, V2, V3R, V4R, V5R, V6R maximum QT values were compared.

Statistics

Categorical (nonparametric) data were compared with Mann-Whitney U test in evaluations. Values in the same group were compared with the paired t test. Statistical data are given as mean value. P values <0.05 were considered significant.

Results

The mean ages of Group 1 and Group 2 were similar (p>0.05). There were no significant differences between the two groups between RA (right atrium) diameter, RV (right ventricle) diameter, V1QTmax, V2QTmax, V3RQTmax and V6RQTmax (p>0.05).

Between Group 1 and Group 2; systolic pulmonary artery pressure were 32.5±6 mmHg; 43.4±11.3 mmHg, respectively, p<0.05).

V4RQTmax were 321.2±26.4 msec; 351.1±33.6 msec, respectively p<0.05 and V5RQTmax were 319.2±22.6 msec; 350.1±32.3 msec, respectively p<0.05 which were found to be significantly different (Table1).

In the ROC Curve analysis, it was observed that the V4RQTmax value was above 321 ms and determined that the shunt rate was 1, 5 and above in these patients with 76, 7% sensitivity and 58.1% specificity (r=0.634).

Discussion

RBBB pattern is frequently encountered in patients with atrial septal defect. RBBB pattern has been suggested to be due to RV volume load and RV hypertrophy.[14]

RBBB may be an electrocardiographic indicator of right ventricular pressure load.[15] It was concluded that the right axis deviation seen in secundum ASD is dependent on the volume and pressure load of the right ventricle.[16] Atrial arrhythmias, first degree atrioventricular block, and right ventricular hypertrophy are other electrocardiographic findings in patients with ASD.[17] These findings on electrocardiography are not specific for ASD and are not sufficient to predict the disease in clinical practice.

Electrocardiographically, right precordial leads are more sensitive than standard leads in showing problems of the right ventricle.

In addition to the standard leads used in the exercise test, right lead leads were added in the thought that it would increase the sensitivity in the diagnosis of coronary artery disease. It was observed that the sensitivity of the exercise test performed in this way to detect right coronary lesions increased.[18]

In our study, there was a significant correlation and positive correlation between V4RQTmax and V5RQTmax

Table 1. Comparison of age, RA diameter, RV diameter, SPAP and V1QTmax, V2QTmax, V3RQTmax, V4RQTmax, V5RQTmax and V6RQTmax values for those with a shunt ratio below 1.5 (Group 1) and those above 1.5 (Group 2)

	Group1(n=14)	Group2(n=60)	p
Age	35.7±11	43.5±16.2	0.259
RA diameter (cm)	2.9±0.7	3.4±1.1	0.35
RV diameter (cm)	2.9±0.8	3.1±1.0	0.662
SPAP (mmHg)	32.5±6	43.4±11.3	**0.0116**
V1QTmax (msec)	341.1±35.4	357.6±31.2	0.276
V2QTmax (msec)	343.1±30.8	371.6±35.2	0.077
V3RQTmax (msec)	338.1±29.6	354.4±27	0.227
V4RQTmax (msec)	321.2±26.4	351.1±33.6	**0.029**
V5RQTmax (msec)	319.2±22.6	350.1±32.3	**0.013**
V6RQTmax (msec)	324±27	347.3±31.6	0.109

RA: Right atrium; RV: Right ventricle; SPAP: Systolic pulmonary artery pressure

values with shunt ratio over 1,5 (respectively $p<0.05$, $r=0.634$; $p<0.05$, $r=0.616$). There was a significant relationship between systolic pulmonary artery pressure and shunt ratio ($p<0.05$). Pulmonary artery pressure was high in patients with shunt ratio above 1,5 this finding. It shows that RV pressure load increases with increasing shunt ratio in patients with ASD. Shahim W. et al informed that the prolongation of the QRS duration in the left precordial leads has been shown to be due to an increase in the pressure load rather than an increase in the volume load of the left ventricle.[19]

Our study; we could not spate a significant relationship between the shunt ratio and the change in RA and RV dimensions ($p>0.05$). However, we observed a significant relationship between systolic pulmonary artery pressure and RA and RV dimensions ($p<0.001$). These results showed that the diameter of RA and RV in patients with ASD is associated with a change in systolic pulmonary artery pressure, not shunt rate.

Conclusions

Were obtained that the right precordial leads could be used to estimate the ratio of patients with ASD. Clinician will be able to comment on shunt rate according to V4RQTmax and V5RQTmax values. In patients who are considered to have ASD as a preliminary diagnosis with symptoms and examination findings, the right lead will be useful in demonstrating the severity of the disease by electrocardiographic evaluation. Moreover, increased shunt ratio leads to increased systolic pulmonary artery pressure and systolic loading occurs in the right ventricle. This pathophysiological change might show itself in length on the right lead electrocardiography (V4R and V5R) at QT.

Disclosures

Ethics Committee Approval: This study was carried out with the approval of the chief of clinic.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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References

1. Dickinson DF, Arnold R, Wilkinson JL. Congenital heart disease among 160 480 live-born children in Liverpool 1960 to 1969: implications for surgical treatment. Br Heart J 1981;46:55–62.
2. Hijazi ZM, Hellenbrand WE. The right ventricle in congenital heart disease. Cardiology Clin 1992;10:91–102.
3. Geva T, Martins JD, Wald RM. Atrial septal defects. Lancet 2014;383:1921–32.
4. Kuijpers JM, Mulder BJ, Bouma BJ. Secundum atrial septal defect in adults: a practical review and recent developments. Neth Heart J 2015;23:205–11.
5. Gatzoulis MA, Freeman MA, Siu SC, et al. Atrial arrhythmia after surgical closure of atrial septal defects in adults. N Engl J Med 1999;340:839–46.
6. Konstantinides S, Geibel, Olschewski M, et al. A comparison of surgical and medical therapy for atrial septal defect in adults. N Engl J Med 1995;333:169–73.
7. Gatzoulis MA, Redington AN, Somerville J, Shore D. Should atrial septal defects in adults be closed? Ann Thorac Surg 1996;61:657–9.
8. Chubb H, Whitaker J, Williams SE, Head CE, Chung NA, Wright MJ, O'Neill M. Pathophysiology and management of arrhythmias associated with atrial septal defect and patent foramen ovale. Arrhythm Electrophysiol Rev 2014;3:168–172.
9. Morton JB, Sanders P, Vohra JK, Sparks PB, Morgan JG, Spence SJ, et al. Effect of chronic right atrial stretch on atrial electrical remodeling in patients with an atrial septal defect. Circulation 2003;8:1775–82.
10. Bolzer E. Tonus changes in cardiac muscle and their significance for the initiation of impulses. Am J Physiol 1943;139:477–80.
11. Lab MJ. Mechanoelectric feedback (transduction) in heart: concepts and implications. Cardiovasc Res 1996;32:3–14.
12. Eckhardt L, Kirchhof P, Breithardt G, Haverkamp W. Load-induced changes in repolarization: evidence from experimental and clinical data. Basic Res Cardiol 2001;96:369–80.
13. McCulloch AD, Pfeiffer E, Tangney J, Omens J. Biomechanics of Cardiac Electromechanical Coupling and Mechanoelectric Feedback. J Biomech Eng 2014;136.
14. Barbosa HT, Brandenburg RO, Swan HJC. Atrial septal defect: the electrocardiogram and its hemodynamic correlation in 100 proven cases. Am J Cardiol 1958;2:698–703.

15. Hutchinson RG. Complete right bundle branch block in right ventricular volüme averload. J Miss State Med Assoc 1969;10:88–91.
16. Boineau JP, Moore EN, Patterson DF. Relationship between the ECG, ventricular activation and the ventricular conductionsystem in ostium primum ASD. Circulation 1973;48:556–64.
17. Hanrath P, Schluter M, Langenstein BA, Poster J, Engel S, Kremer P, et al. Detection of ostium secundum atrial septal defects by transoesophageal cros-sectional echocardiography. Br Heart J 1983;49:350–8.
18. Ueshima K, Kobayashi N, Kamata J, Saitoh M, Yamazaki T, Chiba I, Hiramori K. Do the Right Precordial Leads during Exercise Testing Contribute to Detection of Coronary Artery Disease? Clin Cardiol 2004;27:10–105.
19. Shamim W, Yousufuddin M, Cicoria M, Gibson DG, Coats AJS, Henein MY. Incremental changes in QRS duration in serial ECGs over time identify high risk elderly patients with heart failure. Heart 2002;88:47–52.