

Recently, Vasan et al. (3) published a study of 1.5 million blood donors in which they investigated the association between ABO blood groups and incidence of first and recurrent venous thromboembolic and arterial events. They concluded that the study added strong evidence of a consistent association between non-O blood groups and both venous thromboembolism and cardiovascular events. Taking into account that non-O blood groups confer an overall increased risk of incident, recurrent, and provoked thromboembolism, ABO blood group may have a role in thrombosis risk assessment and could potentially be added to available clinical prediction systems (3).

In the last 4 decades, several studies have investigated the importance of ABO blood group as risk factor of occurrence of arterial thrombosis. In general, a significant increase in the incidence of ischemic heart diseases and cardiovascular mortality in patients with non-O blood groups has been observed (4). One mechanism proposed to explain the association between ABO blood type and ischemic heart disease is elevated serum fibrinogen level. Elevated fibrinogen level constitutes a valuable marker in diagnosis of PVT (5).

It is a fact that patients with non-O blood group have a higher risk of developing venous and arterial thromboembolic events than members of the O blood group.

This report is the first that shows the association between the ABO blood types and occurrence of PVT.

Despite its limitations and need to occur more evidence on this topic, think that non-O blood groups can convert is in an attractive biomarker prognosis for in development of PVT.

In agreement with the authors, I suggest using ABO blood types as a new factor in the stratification of risk of thrombosis in patients with prosthetic heart valve.

Fidel Manuel Caceres-Loriga

Department of Cardiology, University Central Hospital; Lubango-Angola

References

1. Astarcioglu MA, Kalçık M, Yesin M, Gürsoy MO, Şen T, Karakoyun S, et al. ABO blood types: impact on development of prosthetic mechanical valve thrombosis. *Anatol J Cardiol* 2016 Jul 21. Epub ahead of print. [Crossref](#)
2. Franchini M, Lippi G. Relative risks of thrombosis and bleeding in different ABO blood groups. *Semin Thromb Hemost* 2016; 42: 112-7.
3. Vasan SK, Rostgaard K, Majeed A, Ullum H, Titlestad KE, Pedersen OB, et al. ABO Blood Group and Risk of Thromboembolic and Arterial Disease: A Study of 1.5 Million Blood Donors. *Circulation* 2016; 133: 1449-57. [Crossref](#)
4. Zhou S, Welsby I. Is ABO blood group truly a risk factor for thrombosis and adverse outcomes? *World J Cardiol* 2014; 6: 985-92.
5. Aykan AC, Gökdeniz T, Gündüz S, Astarcioglu MA, Gürsoy OM, Ertürk E, et al. Value of serum fibrinogen levels in the assessment of mechanical prosthetic valve thrombosis. *J Heart Valve Dis* 2014; 23: 222-7.

Address for Correspondence: Fidel Manuel Caceres-Loriga, MD, PhD
Department of Cardiology, University Central Hospital,
Lubango-Angola



E-mail: dr.caceres10@hotmail.com

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Author's Reply

Authors of this mentioned article did not send any reply for this Letter to Editor, in spite of our insistently request.

Heart rate variability in Eisenmenger syndrome and its correlation with echocardiographic parameters and plasma BNP, high sensitivity troponin-I level

To the Editor,

Eisenmenger syndrome (ES) is the latest stage of congenital heart disease associated pulmonary arterial hypertension (PAH) and is more common in our daily practice lately. Despite all improvements, there are several limitations to determining prognosis of these patients (1). Therefore, different parameters with high prognostic values are needed. Heart rate variability (HRV) and autonomic dysfunction can be early prognostic markers in patients with ES.

Twenty patients with ES (12 female, 8 male) and 20 healthy matched volunteers were enrolled in the study. Plasma brain natriuretic peptide (BNP) and troponin-I levels were measured. HRV parameters were calculated from 24-hour Holter electrocardiogram recordings. HRV parameters were compared with those of 20 healthy subjects. Bivariate analysis was performed to evaluate correlation between echocardiographic parameters and plasma BNP, high sensitivity (hs)-troponin-I levels. Mean age was 29.25±12.53 years and patients were clinically stable. All patients were receiving specific pulmonary hypertension treatment. Eight patients (40%) were receiving combination therapy, while 12 patients (60%) were receiving single agent.

There were significantly lower time-domain HRV parameters [SD of all RR intervals (SDNN): 125.8±36.96 vs. 173.30±34.47 (p<0.0001); mean of SD of all RR intervals for all 5-minute segments over the entire recording (SDNNi): 48.30±14.65 vs. 71.65±19.74 (p<0.0001); SD of averaged normal RR intervals calculated for all 5-minute periods (SDANN): 116.15±37.22 vs. 157.00±31.18 (p<0.0001); 32.25±14.32 vs. 39.05±14.98 (p=0.151); triangular index (TI): 40.31±20.05 vs. 48.45±14.16 (p=0.150)] in ES patients compared to healthy controls. Root-mean-square of successive normal sinus RR interval difference (RMSSD) and TI were lower in ES patients, but without statistical significance (p=0.151).

There was no statistically significant correlation between HRV parameters and 6-minute walking test, functional capacity, right ventricular systolic function, BNP and hs-troponin-I levels.

Most common cause of death in these patients is arrhythmia, and autonomic dysfunction may be triggering factor (2). HRV parameters are now being used for prognostic evaluation in PAH patients. There are also studies suggesting HRV reduction may be associated with mortality and need for transplant in children and poor prognosis in adults with idiopathic PAH (3). Considering the fact that our patients were clinically stable and were also under appropriate treatment, guideline-recommended prognostic markers were not severely affected, despite significantly reduced HRV parameters. As a result, HRV parameters may be an early marker of prognosis even before deterioration of currently suggested markers. These data suggest that HRV parameters can be utilized as an early marker of poor prognosis in ES patients, but additional prospective studies are needed.

The limited number of patients and lack of long-term follow up are the major limitations of this study. Frequency-domain parameters would also provide additional benefit.

Burak Sezenöz, Gülten Aydoğdu Taçoy¹, Serkan Ünlü¹, Belma Taşel², Sedat Türkoğlu¹, Yakup Alsancak³, Gökhan Gökalp¹, Atiye Çengel¹
Department of Cardiology, Gazi Mustafa Kemal State Hospital; Ankara-Turkey

¹Department of Cardiology, Faculty of Medicine, Gazi University; Ankara-Turkey

²Department of Cardiology, Mersin Anamur State Hospital; Mersin-Turkey

³Department of Cardiology, Atatürk Education and Research Hospital; Ankara-Turkey

References

- Galie N, Humbert M, Vachiery JL, Gibbs S, Lang I, Torbicki A, et al. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension: The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS): Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT). *Eur Respir J* 2015; 46: 903-75. [Crossref](#)
- Rich S, Dantzker DR, Ayres SM, Bergofsky EH, Brundage BH, Detre KM, et al. Primary pulmonary hypertension. A national prospective study. *Ann Intern med* 1987; 107: 216-23. [Crossref](#)
- Engelfriet PM, Duffels MG, Moller T, Boersma E, Tijssen JG, Thaulow E, et al. Pulmonary arterial hypertension in adults born with a heart septal defect: the Euro Heart Survey on adult congenital heart disease. *Heart* 2007; 93: 682-7. [Crossref](#)

Address for Correspondence: Dr. Burak Sezenöz
 Gazi Mustafa Kemal Devlet Hastanesi
 Kardiyoloji Bölümü, Yenimahalle, Ankara-Türkiye
 E-mail: drburaksezenoz@gmail.com

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CRT-D or CRT-P in CRT-indicated patients?

To the Editor,

Cardiac resynchronization therapy with defibrillator (CRT-D) has demonstrated advantages over implantable cardioverter defibrillator (ICD) in terms of morbidity, symptom reduction, and survival. But there is no exact data indicating benefit of adding an ICD in CRT-indicated patients, despite theoretically decreased risk of death due to arrhythmia with this combination (1). Despite the lack of evidence, CRT-D is preferred over cardiac resynchronization therapy with pacemaker (CRT-P) without any strict recommendation. Here we would like to share our experience, which also favors CRT-D over CRT-P, but for another reason: pacing site-dependent arrhythmia.

Pacing site-dependent arrhythmia, first described by Medina-Ravell et al. (2) in 2003, can be defined as an arrhythmia due to non-physiological, simultaneous pacing of right ventricle (RV) endocardium and left ventricle (LV) epicardium. Normal ventricle activation starts at the endocardium and spreads through the myocardium to the epicardium. Due to longer duration of action potential of endocardium; repolarization wave starts at the epicardium and ends in the endocardium. This sequence of activation and repolarization makes an upright T wave with the same polarity as the QRS (3). LV epicardial pacing alters ventricle activation and repolarization dynamics, which in turn ends up with prolongation of QT interval, leaving ventricle vulnerable to extrasystoles that result in R on T phenomenon, Torsades des Pointes (TdP), or non-sustained or sustained polymorphic ventricular tachycardia (VT). The basic mechanism of formation and progression of TdP and polymorphic VT is the same as long QT syndromes. The incidence of this condition was reported to be between 3.4% and 4% and most were ischemic cardiomyopathy patients (4).

As a tertiary cardiovascular hospital, our institution has performed more than 250 CRT implantations over the course of 10 years. During this time, we observed 1 incessant electrical storm in TdP patient (5), and 2 monomorphic ventricular tachycardia (MMVT) patients soon after starting biventricular pacing (BiVP) mode with CRT. The first patient was a 59-year-old woman, suffering from ischemic cardiomyopathy who went from functional class I to III (New York Heart Association) over time and had electrocardiogram of sinus rhythm with left bundle branch block morphology and QRS duration of 160 ms. Decreased ejection fraction (EF) to 20% with increased functional class led us to consider CRT for symptom relief and ICD for primary prevention (no prior episodes of syncope or tachycardia). When CRT was activated in the operating room, incessant electrical storm of TdP started. After failed anti-tachycardia pacing attempts, defibrillation was used to stop the TdP. Device was switched off and considered a possible cause since this patient had not experienced tachycardia attack before. Pacing from RV endocardium and right atrium did not trigger the arrhythmia, but every attempt to pace