Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: www.saspublishers.com

OPEN ACCESS

Urology

Parasympathetic System in Heart Failure: A Case Series

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| **Received:** 06-05.2019 | **Accepted:** 13.05.2019 | **Published:** 21.05.2019
DOI: 10.36347/sjams.2019.v07i05.008

Abstract **Original Research Article**

In heart failure, it has been recognized that the sympathetic nervous system (SNS) is activated, with imbalance of its activity leading to vagal interactions. Measuring vagal response as a daily practice in heart failure patients would be challenging. The aim of this study is to report the vagal response. We conducted a prospective study including a group of symptomatic cardiac failure subjects with ejection fraction EF <40% with sinus rhythm, able to perform the deep breathing and valsalva test. All of them had clinical examination, ECG, echocardiography and evaluation of autonomic nervous system function. The results obtained were analyzed using SPSS software. We included 100 patients; their mean age was 54 years. The exploration of the autonomic nervous system in our patients was based essentially on two tests which are the deep breathing and the Valsalva maneuver. The results showed a vagal deficit among 80% of our patients with deep breathig test and 69% with the Valsalva maneuver. This results objectived a significant vagal deficiency response. Furthermore, it has recently been demonstrated that vagal stimulation has beneficial effects on heart failure patients.

Keywords: Vagal nerve, heart failure, deficiency.

INTRODUCTION

Patients with chronic heart failure (HF) have features of autonomic dysfunction characterized by excessive sympathetic activation and concomittant parasympathetic withdrawal. Autonomic imbalance, which manifests as a reduction of heart rate [HR], variability and baroreflex sensitivity, is associated with worsening HF and an increased risk of mortality independently of ejection fraction and ventricular arrhythmias. Vagal stimulation, a very new promising management of heart failure is under investigation. Measuring vagal response as a daily practice in cardiac failure patients would be interesting. The objective of this study is to report the vagal response amongst heart failure patients.

METHODS

We conducted a prospective study including a group of symptomatic cardiac failure subjects with ejection fraction EF <45% with sinus rhythm. All of them had clinical examination based on heart rate and blood pressure, ECG, echocardiography, deep breathing test (that will evaluate changes in the instant heart rate by deep breathing at a rythm of 6 breaths/mn performed in a supine position) and Valsalva maneuver (consisting on a forced expiration against a closed glottis which leads to changes in intrathoracic pressure). The results obtained were analyzed using SPSS software.

RESULTS

We included 100 patients; extreme ages were ranging from 16 to 89 years, with a mean age of 54 years [figure 1]. Sex ratio was 1.9 with 66% of women and 34% of men [figure 2].

Associated cardiovascular risk factors noted were chronic smoking (34% of the cases), followed by sedentary (32% of the cases), 29% of the cases of android obesity, 28% of patients suffered from diabetes, 26% of the cases from high blood pressure and 19% of the cases had dyslipidemia. However, we didn't notice any of an inherited coronary artery desease [figure 3].

According to the NYHA classification of dyspnea, 13 patients had class I, 45 patients had class II, 31 patients had class III, and 12 patients had class IV.

In our series, acute decompensated heart failure was found in 59 patients, while 41 patients had a compensated heart failure. 61 patients showed no signs of HF, 12 patients showed signs of right-sided HF, and 14 patients had signs of congestive HF [figure 4]. Heart failure was complicated with ischemic heart disease in 42 cases, followed by valvulopathies in 26 cases

[figure5]. The exploration of the autonomic nervous system in our patients was based essentially in two tests, the deep breathing test and the Valsalva maneuver [Table 1].

Concerning the deep breathing test, amongst the 36 patients that were under 50 years, 27 ones had a vagal deficiency. For the rest of the patients (64 aged more than 50 years), 28 had a vagal deficiency. Coming to the Valsalva maneuver, its p value below 1.21 is considered abnormal with a vagal deficit in 69%in our patients [figure 6].

Table-1: Vagal reponse to the deep breathing test

	Vagal deficiency	Normal response (Ratio>1,2)	Total
Age < 50 years	27	9	36
	Vagal deficiency	Normal response (Ratio>1,1)	Total
Age > 50 years	28	36	64

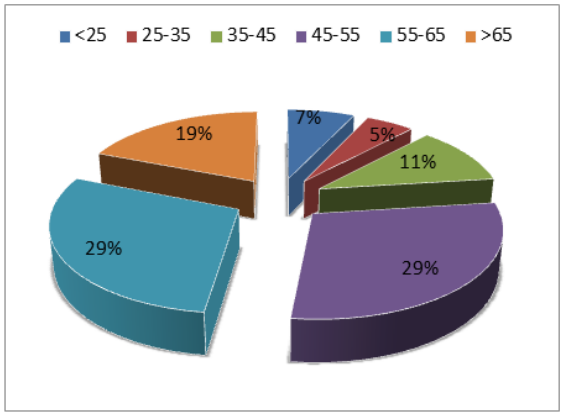

Fig-1: Patient distribution by Age

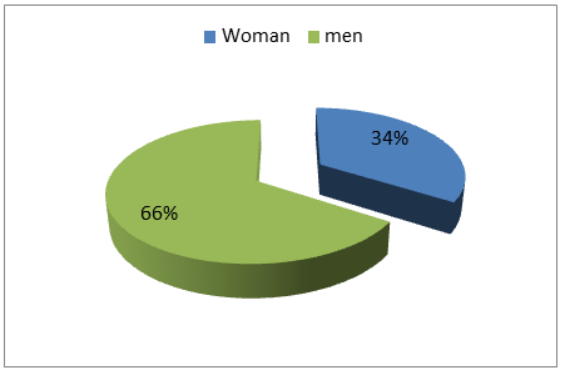

Fig-2: Patient Distribution by sex

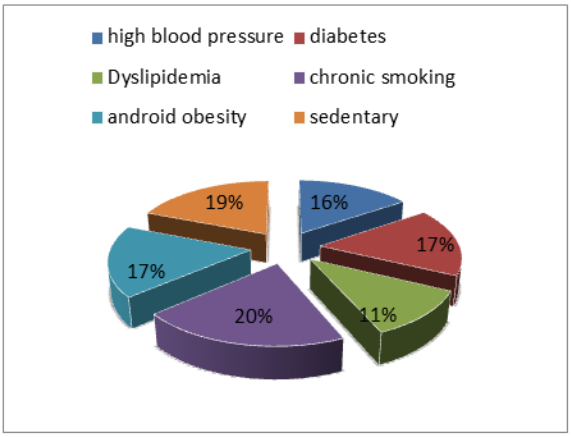

Fig-3: Distribution of cardiovascular risk factors

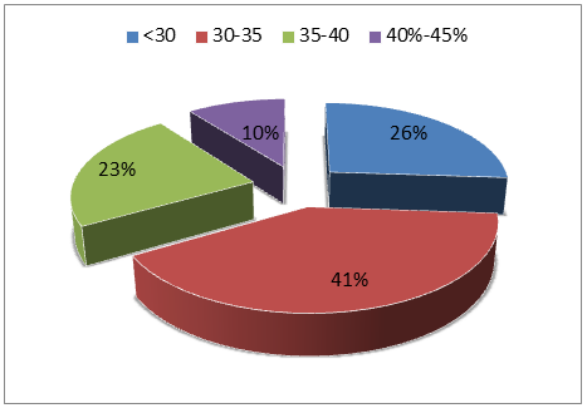

Fig-4: Ejection fraction in our patients

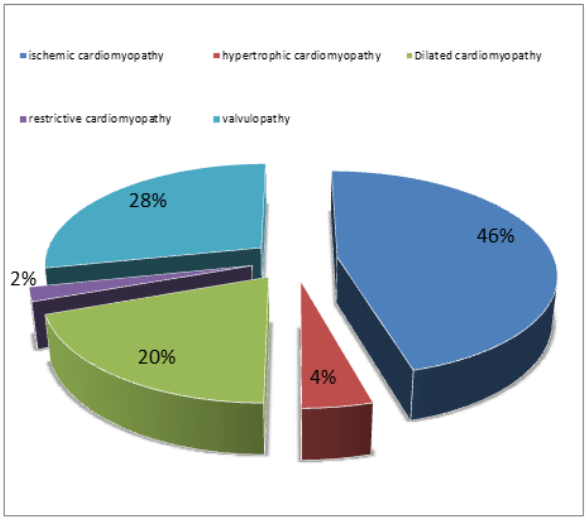

Fig-5: Etiology of heart failure

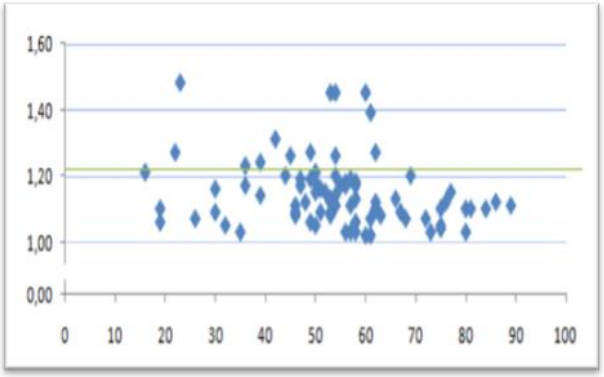

Fig-6: Vagal response to the Valsalva maneuver

DISCUSSION

These results have shown a significant vagal deficiency response in cardiac failure patients, and this concords with the study conducted N.Mouine who included 34 patients, 10 patients [group 1] compared to 24 patients [group 2]. Preliminary results showed vagal response of $7,8 \pm 1,7\%$ in group 1 versus $27 \pm 7,4\%$ in group 2 [$p < 0,05$] [1].

Parasympathetic control of the heart via the vagus nerve is the primary mechanism that regulates beat-to-beat control of heart rate [2]. Additionally, the vagus nerve exerts significant effects at the AV node, as well as effects on both atrial and ventricular myocardium. Vagal control is abnormal in heart failure, occurring at early stages of left ventricular dysfunction, and this reduced vagal function is associated with worse outcomes in patients following myocardial infarction and with heart failure. While central control

mechanisms are abnormal, one of the primary sites of attenuated vagal control is at the level of the parasympathetic ganglion [3]. It remains to be seen whether or not preventing or treating abnormal vagal control of the heart improves prognosis [4].

Acute myocardial ischemia represents a very useful model for a clear representation of what may appear as a complex relationship between sympathetic and vagal activity [5]. The sensory endings of both vagal and sympathetic afferent fibers are mechanoreceptors and are thereby stimulated by the mechanical stretching associated with cardiac dilatation. Indeed, when the heart dilates, vagal and sympathetic afferent cardiac fibers increase their firing, and this afferent sympathetic excitation leads to the tonic and reflex inhibition of cardiac vagal efferent activity [2].

This phenomenon can be expected to occur whenever the heart dilates in heart failure probably due to the systolic dysfunction. In cases of diastolic dysfunction in which the heart does not dilate, the mechanisms in which the vagal activity is reduced have not been fully determined. The study failed to demonstrate an improvement in left ventricle remodeling [LV] parameters, LV function, or circulating biomarkers following 6 months of chronic vagal stimulation [VNS] at the prescribed stimulation settings [11].

NECTAR-HF did however demonstrate significant improvements in the subjective endpoints of NYHA functional class and heart failure related quality-of-life measures [12-14]. Chronic beta-blocker therapy improves left ventricular performance and reverses left ventricular remodeling, reduces risk of hospitalization, and improves survival [6-8]. We must recognize that heart failure is a complex syndrome with an autonomic nervous system dysfunction, and that the autonomic imbalance with the activation of SNS and the reduction in vagal activity should be focused more in the aspects of treatment of heart failure. In this aspect, conservative pharmacological therapy is not sufficient, and device therapy and/or non-pharmacological therapy (exercise training, Waon-therapy) are necessary [5, 9, 10].

CONCLUSION

These results showed a significant vagal deficiency response in cardiac failure. It has been recognized that autonomic nervous system dysfunction occurs. In the treatment of heart failure, the inhibition of the activated SNS, such as with beta-blockers and/or exercise training is important. Furthermore, it has recently been demonstrated that vagal stimulation has beneficial effects on heart failure.

Authors' contributions

All the authors contributed to the writing of this manuscript

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