Int J Cardiovasc Pract. **Original Article**

July 2016, Volume 1, Issue 2 (33-35)

Effect of Nocturnal Oxygen Therapy on Electrocardiographic Changes among Patients with Congestive Heart Failure

Mehdi Pishghahi [1,*], Rama Bozorgmehr [2], Masoud Rastgari [2], Mohammad Amin Abbasi [2]

[1] Department of Cardiology, Faculty of Medicine, Shohada Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[2] Department of Internal Medicine, Faculty of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran

** Corresponding author: Mehdi Pishghahi, Department of Cardiology, Faculty of Medicine, Shohada Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran. Tel: +98-2125719, E-mail address: mp_cr1@yahoo.com*

DOI: 10.20286/ijcp-010204

Submited: 06.10.2016

Accepted: 06.27.2016

Keywords:

Heart Failure

Oxygen Inhalation Therapy

Arrhythmias, Cardiac

Abstract

Introduction: Nocturnal hypoxia is an important factor in increasing the risk of mortality in patients with chronic heart failure and is associated with atrial and ventricular arrhythmias. In addition, QT dispersion (QTd) is used as a prognostic sign in determining future malignant arrhythmias and sudden cardiac death. In the current study, we investigated the effect of nocturnal oxygen therapy (NOT) on electrocardiographic changes among patients with chronic heart failure.

Methods: In this study, a consecutive of 154 patients (87 males and 67 females) known with chronic heart failure (EF ≤ 40%) were enrolled. The patients were administered NOT (oxygen flow of 2 L/min for 8 hours during sleeping). Electrocardiography was taken before and after the NOT, and RR interval, PR interval and QTd were measured each time.

Results: The mean age of the participants was 61.3 ± 11.4 years. Our results revealed significant reduction in QTd (55.8 ± 7.5 vs. 61.4 ± 9.1 msec, P = 0.001) and heart rate (79.6 ± 4.7 vs. 76.8 ± 4.3, P = 0.001) in a patient's electrocardiogram after NOT.

Conclusions: In this study, NOT decreased heart rate and QTd in patients with chronic heart failure, but not PR interval, which could consequently decrease the risk of malignant arrhythmias and sudden cardiac death.

INTRODUCTION

Chronic heart failure (CHF) is one of the most important problems in cardiovascular diseases. About 2% of the adult population suffers from heart failure, with 50% mortality during five years and with a high economic cost due to frequent and prolonged hospitalizations for hemodynamic decompensation [1, 2]. The prognosis of patients with CHF remains poor, despite the introduction of many effective drugs in clinical settings. One of the cardiac pathophysiologic abnormalities that worsens heart failure is activation of the sympathetic nervous system [3]. Among patients with CHF, sudden cardiac death occurs in 50% as of fatal ventricular arrhythmias, particularly ventricular tachycardia and fibrillation [4].

QT dispersion (QTd) is defined as the difference between the longest and the shortest QT intervals as measured in the 12-lead electrocardiogram (ECG). It can be assessed computerized and manually. QTd as a marker of ventricular repolarization inhomogeneity may be a potential prognostic sign in the detection of future ventricular tachyarrhythmic events and death [5]. Many factors may affect QTd. Nocturnal hypoxia is associated with atrial and ventricular arrhythmias in patients with chronic heart failure and is an important factor increasing the risk of mortality. Furthermore, smoking may acutely increase QTd, which may be due to hypoxia or other harmful changes in the electrical function of myocardial cells [6, 7]. Despite significant advances in the treatment of chronic heart failure, mortality and morbidity remain high [8]. Therefore, in this study we aimed to investigate the effect of nocturnal oxygen therapy (NOT) on QTd in patients with CHF.

METHODS

The protocol for this study was approved by the Medical Ethics Committee of Shahid Beheshti University of Medical Sciences. Between June 2015 and October 2016, a consecutive of 154 patients with CHF with etiologies including coronary artery disease, idiopathic dilated cardiomyopathy, valvular heart disease, or hypertensive heart disease were enrolled in this prospective study. Patients were excluded if they had atrial fibrillation, pacemaker dependency, electrolyte imbalance, class I or class III antiarrhythmic drug treatment, acute congestive HF, or renal failure requiring hemodialysis. Patients were administered NOT, nasal oxygen thrapy with the oxygen flow of 2 L/min for

8 hours during sleeping. ECG was taken before and after NOT.

Evaluation of Electrocardiogram

Standard 12-lead ECGs were analyzed at a paper speed of 50 mm/s. The R-R (interval between two QRS), PR and QT intervals were measured in sinus rhythm in all 12 leads, recorded simultaneously by two observers. The QT intervals were measured from the onset of the QRS to the end of the T wave, defined as a return to the baseline. QTd was defined as maximum minus minimum QT interval.

Statistical Analysis

All the data were presented as the mean ± SD. Statistical analyses were performed by SPSS statistical software (version 16.0 for Windows; SPSS Inc., Chicago, IL, USA). Statistical analysis was performed using paired t-test for data before and after the oxygen therapy. P values less than 0.05 were considered statistically significant.

RESULTS

A consecutive of 154 patients (87 males and 67 females) known with chronic heart failure was enrolled in this study, of which 87 (56.4%) had ischemic, 43 (28%) hypertensive, 15 (9.8%) valvular and 9 (5.8%) non-ischemic dilated cardiomyopathy as the underlying cause of CHF. The mean ± SD age of the participants was 61.3 ± 11.4 years. The demographic characteristics of patients are represented in Table 1. The mean ± SD ages of males and females were 61.45± 10.49 and 60.71± 11.31 years, respectively; there was no significant deference between gender (P = 0.3).

Our results revealed that after nocturnal oxygen therapy, QTd (55.8 ± 7.5 vs. 61.4 ± 9.1 msec, P = 0.001) and heart rate (79.6 ± 4.7 vs. 76.8 ± 4.3, P = 0.001) significantly reduced. Despite reduction in QTd and heart rate, here in we did not reveal any significant changes in PR interval before and after the NOT (17.6 ± 2.4 vs. 18.4 ± 2.1 respectively, P = 0.2).

Table 1: Baseline Characteristics

	Values, (n=154)
Age, years	61.4 ± 11.2
EF, %	48.3 ± 8.5
Male	87 (56.49)
Diabetes, %	76 (22.1)
Hypertension, %	43 (28)
Ischemic heart disease, %	87 (56.4)
Dilated Cardiomyopathy, %	9 (5.8)
Valvular heart disease	15 (9.8)

Data in table are presented as Mean ± SD or number (%).

DISCUSSION

Our study demonstrated that NOT decreased QTd in patients with CHF, which could consequently decrease the risk of malignant arrhythmias and sudden cardiac death. To our knowledge, few reports have investigated the effect of NOT on electrocardiogarphic changes among patients with CHF. The results of our study were along with previous studies, which have demonstrated significantly increased QTd values in patients with CHF [9].

It is known that the renin-angiotensin-aldosterone system as a mediator of neurohormonal activation and the sympathetic nervous system are the two most important factors in the progression of CHF [10]. Activation of these systems is suggested as relating to a poor prognosis in patients with CHF [11]. Sympathetic activation or vagal withdrawal at the level of the sinoatrial node will increase the heart rate and shorten the RR interval, whereas an increase in sympathetic outflow to the ventricular myocardium will decrease the QT interval. A decrease in sympathetic outflow or an increase in vagal outflow to the atrio-ventricular node will result in PR interval prolongation [12].

Dispersion of ventricular repolarization may represent as the substrate for re-entry tachycardia and can be masured by QTd. An increased repolarization heterogeneity has been approved in the genesis of re-entry and malignant ventricular arrhythmias in many studies [13]. The greater the QTd, the greater the variability in the timing of electrical recovery within the heart. Many studies have suggested that increased Qd can predict sudden death and ventricular arrhythmias in patients with CHF or hypertrophic cardiomyopathy [4].

It is also a predictor for developing ventricular arrhythmias in patients with different cardiovascular pathologies, including myocardial infarction, vasospastic angina, systemic hypertension, long QT intervals, or idiopathic dilated cardiomyopathy [4, 13]. In a retrospective case control study, Bonnar et al. compared the QTd and corrected QTd values in patients and left ventricular systolic dysfunction with control subjects. The QTd and corrected QTd values were significantly higher in patients with impaired left ventricular systolic function than controls [14].

In addition, it is known that in patients with obstractive sleep apnea, hypoxia significantly increases the heart rate and shortens the QT interval, and these changes are greater compared with healthy controls [15]. Our findings were similar to a previous study by Piranfar et al., demonstrating that NOT significantly decreased QTd in patients with CHF [16].

In this study, NOT decreased heart rate and QTd in patients with CHF, which could consequently decrease the risk of malignant arrhythmias and sudden cardiac death.

Table 2: Electrocardiographic Values before and after Nocturnal Oxygen Therapy

	Before NOT	After NOT	P value
Heart Rate	79.6 ± 4.7	76.8 ± 4.3	0.001
PR interval	2.1 ± 18.4	2.4 ± 17.6	0.2
QTd	61.4 ± 9.1	55.8 ± 7.5	0.001

NOT: Nocturnal Oxygen Therapy

Data in table are presented as Mean ± SD.

CONFLICTS OF INTEREST

All authors declared that this study has no conflict of interest.

REFERENCES

1. McMurray JJ, Stewart S. Epidemiology, aetiology, and prognosis of heart failure. Heart. 2000;83(5):596-602. DOI: 10.1136/heart.83.5.596 PMID: 10768918
2. Writing Group M, Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, et al. Heart disease and stroke statistics--2010 update: a report from the American Heart Association. Circulation. 2010;121(7):e46-e215. DOI: 10.1161/CIRCULATIONAHA.109.192667 PMID: 20019324
3. Thomas JA, Marks BH. Plasma norepinephrine in congestive heart failure. Am J Cardiol. 1978;41(2):233-43. DOI: 10.1016/0002-9149(78)90162-5 PMID: 203177
4. Yildirir A, Sade E, Tokgozoglu L, Oto A. The effects of chronic carvedilol therapy on QT dispersion in patients with congestive heart failure. Eur J Heart Fail. 2001;3(6):717-21. DOI: 10.1016/S1388-9842(01)00191-X PMID: 11738224
5. Day CP, McComb JM, Campbell RW. QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. Br Heart J. 1990;63(6):342-4. DOI: 10.1136/hrt.63.6.342 PMID: 2375895
6. Kautzner J, Malik M. QT interval dispersion and its clinical utility. Pacing Clin Electrophysiol. 1997;20(10 Pt 2):2625-40. DOI: 10.1111/j.1540-8159.1997.tb06112.x PMID: 9358510
7. Akbarzadeh MA, Yazdani S, Ghaidari ME, Asadpour-Piranfar M, Bahrololoumi-Bafruee N, Golabchi A, et al. Acute effects of smoking on QT dispersion in healthy males. ARYA Atheroscler. 2014;10(2):89-93. PMID: 25161676
8. Malik M, Farbom P, Batchvarov V, Hnatkova K, Camm AJ. Relation between QT and RR intervals is highly individual among healthy subjects: implications for heart rate correction of the QT interval. Heart. 2002;87(3):220-8. DOI: 10.1136/heart.87.3.220 PMID: 11847158
9. Kuo CS, Reddy CP, Munakata K, Surawicz B. Mechanism of ventricular arrhythmias caused by increased dispersion of repolarization. Eur Heart J. 1985;6 Suppl D:63-70. DOI: 10.1093/eurheartj/6.suppl_D.63 PMID: 2417854
10. Francis GS, Benedict C, Johnstone DE, Kirlin PC, Nicklas J, Liang CS, et al. Comparison of neuroendocrine activation in patients with left ventricular dysfunction with and without congestive heart failure. A substudy of the Studies of Left Ventricular Dysfunction (SOLVD). Circulation. 1990;82(5):1724-9. DOI: 10.1161/01.CIR.82.5.1724 PMID: 2146040
11. Smith JH, Baumert M, Nalivaiko E, McEvoy RD, Catcheside PG. Arousal in obstructive sleep apnoea patients is associated with ECG RR and QT interval shortening and PR interval lengthening. J Sleep Res. 2009;18(2):188-95. DOI: 10.1111/j.1365-2869.2008.00720.x PMID: 19645965
12. Nalivaiko E, Catcheside PG, Adams A, Jordan AS, Eckert DJ, McEvoy RD. Cardiac changes during arousals from non-REM sleep in healthy volunteers. Am J Physiol Regul Integr Comp Physiol. 2007;292(3):R1320-7. DOI: 10.1152/ajpregu.00642.2006 PMID: 17110530
13. Barr CS, Naas A, Freeman M, Lang CC, Struthers AD. QT dispersion and sudden unexpected death in chronic heart failure. Lancet. 1994;343(8893):327-9. PMID: 7905146
14. Bonnar CE, Davie AP, Caruana L, Fenn L, Ogston SA, McMurray JJ, et al. QT dispersion in patients with chronic heart failure: beta blockers are associated with a reduction in QT dispersion. Heart. 1999;81(3):297-302. PMID: 10026356
15. Roche F, Gaspoz JM, Court-Fortune I, Costes F, Geyssant A, Duverney D, et al. Alteration of QT rate dependence reflects cardiac autonomic imbalance in patients with obstructive sleep apnea syndrome. Pacing Clin Electrophysiol. 2003;26(7 Pt 1):1446-53. PMID: 12914620
16. Asad PourPiranfar M. Evaluation of overnight O2 therapy on QT dispersion in patients with chronic heart failure. Res Med. 2013;2013(37):1.