International Journal of Innovation Scientific Research and Review
Vol. 04, Issue, 10, pp.3442-3444, October 2022
Available online at http://www.journalijisr.com
SJIF Impact Factor 4.95

ISSN: 2582-6131

Case Report

PARADOXICAL SINUS NODE DECELERATION DURING DOBUTAMINE STRESS ECHOCARDIOGRAPHY; CASE STUDY

[2]Shahad AlRaimi, [1]Yahya M AlZahrani, [1]Zainalabdin Assadigi, [1,3] *Muneera AlTaweel, [4]Sarah AlMukhaylid

[1]King Abdulaziz Hospital, MNGHA, Al Ahsa, Saudi Arabia.
[2]Echocardiography Technologist, King Abdulaziz Hospital, MNGHA, AlAhsa, Saudi Arabia.
[3]King Abdullah International Medical Research Center (KAIMRC), Al Ahsa, Saudi Arabia.
[4]College of Applied Medical Sciences (CoAMS-A), King Saud Bin Abdulaziz University of Health Sciences, AlAhsa, Saudi Arabia.

Received 13th August 2022; Accepted 14th September 2022; Published online 20th October 2022

ABSTRACT

Dobutamine stress echocardiography (DSE) is a well-established non-invasive testing procedure for diagnosing myocardial ischemia and coronary artery disease. As a result of dobutamine stress testing, side effects such as ventricular tachycardia, fatal ventricular fibrillation, cardiogenic shock, free wall rupture, and transient ischemic attacks have been reported. However, a paradoxical heart rate response during dobutamine stress echocardiography (DSE) is not necessarily associated with abnormal test results, nor does it imply coronary artery disease. Herein we report a 54-year-old female who presented with paradoxical sinus node deceleration of heart response to maximum dobutamine dose resulting in stress test interruption in the absence of obstructive coronary artery disease.

Keywords: dobutamine stress Echocardiography, paradoxical heart rate response to stress.

INTRODUCTION

A dobutamine stress echocardiogram combines normal echo with the drug dobutamine; it's an alternative method to detect coronary artery disease if a patient cannot perform a treadmill test. Dobutamine Infusion increases myocardial contractility and heart rate, increasing myocardial oxygen demand (Attenhofer *et al.,* 1997). Normal coronaries respond by dilation to increase blood flow, while stenosed coronaries cause myocardial ischemia, manifested by ventricle dysfunction (Elhendy *et al.,* 2002). Paradoxical sinus deceleration is an infrequent dose-dependent complication observed on high-dose dobutamine infusion by activating a phenomenon called bezold-jaricshreflex. Many generated studies investigated whether dobutamine-induced nodal deceleration is due to the right coronary artery ischemia and whether it can be an accurate marker of right coronary artery disease (Attenhofer *et al.,* 1997).

CASE REPORT:

A 54-year-old female is known to have Dyslipidemia, Diabetes mellitus, newly diagnosed Hypertension, bilateral Osteoarthritis with status post left total knee replacement and gastro esophageal reflux disease. Her condition started with retrosternal heaviness associated with mild Shortness of breath for two weeks. The chest pain is precipitated by minimal exertion, relieved by rest, radiating to her back and neck, and is not associated with palpitation, dizziness, vomiting, sweating, or syncope. However, before presenting to our hospital, the patient sought medical advice at a private clinic, was found to have uncontrolled hypertension, and started on medication with no improvement. Therefore, the patient went to the emergency department for evaluation to rule out coronary artery disease. Thus, routine investigation of electrocardiography, Troponin-I, CBC, and Biochemical lab results were all normal. An echocardiogram revealed an ejection fraction of 60-65% with no regional wall motion abnormalities. The patient underwent a Dobutamine Stress Echocardiogram test (DSE) to exclude underlying ischemic heart disease. The patient achieved 70% of the predicted heart rate, but the test was terminated due to Paradoxical Sinus node deceleration at the max dose of dobutamine. Then, she entered a junctional rhythm, requiring small atropine dose. Test at 70% THR was negative for Exercise-induced ischemia with appropriate wall thickness and movement with no chest pain and Hemodynamic normal response, maintained throughout the recovery period. The following table shows the heart rate and blood pressure response during a dobutamine stress Echocardiogram (Table 1). Lastly, the patient underwent Cardiac Computed Tomography Angiography, which was negative for obstructive coronary artery disease. Therefore, the patient presenting symptoms are likely hypertension-related, and the DES heart rate response is a cardio inhibitory vagal reflex.

Table 1: Heart rate and blood pressure response to dobutamine stress Echocardiogram

Time	Dosage (µg/kg/min)	Heart rate (Bpm)	Blood pressure (mmHg)
Bassline	-	85	155/79
3 min	5	95	168/83
6 min	10	95	194/71
9 min	30	100	188/71
12 min	40	121 then dropped to (81Bpm)	175/68
+ Atropine	0.25mg	100	130/56
Recovery	-	82	119/65

***Corresponding Author: Muneera AlTaweel,**
1 King Abdulaziz Hospital, MNGHA, Al Ahsa, Saudi Arabia.
3 King Abdullah International Medical Research Center (KAIMRC), Al Ahsa, Saudi Arabia.

DISCUSSION:

Stress Echocardiography combines echocardiography with physical or pharmacological stress (Sicari *et al.,* 2009). The dobutamine stress echocardiography test (DSE) isa diagnostic well-tolerated modality that evaluates impaired coronary vascular function (Attenhofer *et al.,* 1997; Hossain *et al.,* 2020; Eroglu *et al.,* 2015). DSE has a sensitivity of 77% and a specificity of 75% (Kamperidis *et al.,* 2017). Regional wall motion abnormality in stress echocardiography test is the diagnostic endpoint for myocardial ischemia. The imbalance between blood supply and oxygen demand usually results in myocardial ischemia (Sicari *et al.,* 2009). High doses of dobutamine results in both positive inotropic and chronotropic responses that increase heart rate and cardiac output. The Increment in heart rate and myocardial contractility increase oxygen demand. In normal coronary arteries, it will contribute to the dilatation of the coronary arteries and increase blood flow (Elhendy *et al.,* 2002). However, it has been noticed that some patients during high dobutamine dose infusion have hada paradoxical decrease in heart rate (Attenhofer *et al.,* 1997). The first documentation of a rapid decline in heart rate after dobutamine drug administration occurred during horses 'therapy (Hofmeister *et al.,* 2005). Initiation of myocardial ischemia during DSE depends on increased heart rate. At the same time, under-detection of coronary artery disease could be caused by the paradoxical decrease in heart rate during DSE. However, it has also been reported in coronary artery disease patients (Attenhofer *et al.,* 1997).

The consequences of having sinus node deceleration during DSE cannot be equated with right coronary artery disease due to the high incidence of false-positive results (Takeuchi *et al.,* 1998). It could be observed in patients with normal coronary artery disease, without angiographic coronary artery disease, or inducible myocardial ischemia proposing the presence of cardio inhibitory reflex (Attenhofer *et al.,* 1997;4). In a study comprised of 181 consecutive patients,112 were men and 69 were women, only 14 patients had a paradoxical decrease in heart rate after dobutamine infusion, and a total of 8% of the patients had paradoxical sinus deceleration. Of the 14 patients, 57% had hypotension with sinus deceleration (Attenhofer *et al.,* 1997). Another study reported 7.1% of 757 patients that developed sinus node deceleration induced by Dobutamine. It had been assumed that the mechanism of sinus deceleration during DES is by the cardio inhibitory vagal reflex (Takeuchi *et al.,* 1998). Bezold-Jarisch reflex manifests this cardio inhibitory and vasodepressor response that activates the increase of parasympathetic activity and withdrawal of sympathetic activity, promoting bradycardia, hypotension, and vasodilation (Attenhofer *et al.,* 1997; Hossain *et al.,* 2020; Kamperidis *et al.,* 2017). The receptors with vagal afferent that deal with cardio inhibitory, and vasodepressor responses are mainly located in the anteroposterior wall (Holland and Stouffer, 2022). Sensory receptors located at the anteroposterior LV wall are activated by chemical drugs or forceful myocardial contraction (Kamperidis *et al.,* 2017). In some reported cases, sinus deceleration has been reported with blood pressure that is hypertensive or normotensive. (Kamperidis *et al.,* 2017) Reported a case of a 55-year-old male known to have coronary artery disease and had RCA percutaneous intervention. Baseline Echocardiography revealed normal LV systolic function. ECG was normal sinus rhythm without significant arrhythmia. During DSE blood pressure was significantly increased with paradoxical bradycardia. Positive DSE was reported due to regional wall abnormalities during recovery and achieving 88% of the target heart rate (Kamperidis *et al.,* 2017).

To the best of our knowledge, one reported case was similar to ours. In that case, a 44-year male patient had acute myocardial infarction that was treated with primary stent angioplasty of the left anterior descending artery. The patient's baseline echocardiogram showed anterior hypokinesis and akinesis wall with an ejection fraction of 50%, hisresting heart rate (HR) was 63 bpm, and blood pressure (BP) was 115/70 mmHg. The patient underwent DSE for the suspicion of in-stent restenosis. During DSE of normal blood pressure, echocardiography did not reveal any current new wall motion abnormalities, although Electrocardiogram (ECG) revealed the onset of sinus bradycardia. The patient performed coronary angiography that showed arteries were free of atherosclerosis and patent stent (Olszowska *et al.,* 2012). Our case showed a normal baseline of sinus rhythm and normal baseline transthoracic Echocardiogram of normal left ventricle function with an estimated ejection fraction of 60-65%, normal regional wall motion abnormalities, and LV wall thickness. The patient's blood pressure was stable, and no regional wall abnormalities progressed, Electrocardiogram changes, abnormal blood pressure, or chest pain. After the peak dose of Dobutamine 40 mg/mg/min, the heart rate dropped from 121bpm to 81 bpm of an abruptjunctionalrhythm with normal blood pressure. CT revealed a patent coronary with no stenosis. In line with previous studies, arterial hypotension has always accompanied paradoxical sudden sinus deceleration. The mechanism for paradoxical sinus deceleration during DSE is ambagious. Bezold-Jarisch reflex could be the underlining cause of sudden sinus deceleration (Olszowska *et al.,* 2012).

CONCLUSION

The paradoxical sinus node deceleration finding in both our case and (Olszowska, Musiałek, Drwiła & Podolec, 2022) case are unlikely to be the result of Bezold-Jarisch reflex or baroreflex response during the DSE which usually presents with hypotension. Physicians and healthcare providers should be aware of such a side effect, with normal or high blood pressure that could not always indicate underlining a coronaries artery disease.

Consent

Written informed consent was obtained from the patient for publication.

Ethical approval

The study was approved by King Abdullah International Medical Research Center (KAIMRC).

Competing interest

The authors have declared that no competing interests exist.

REFERENCES

1. Attenhofer, C. H., Pellikka, P. A., McCully, R. B., Roger, V. L., & Seward, J. B. (1997). Paradoxical sinus deceleration during dobutamine stress echocardiography: description and angiographic correlation. Journal of the American College of Cardiology, 29(5), 994–999. https://doi.org/10.1016/s0735-1097(97)00030-2
2. Elhendy, A., Bax, J. J., &Poldermans, D. (2002). Dobutamine stress myocardial perfusion imaging in coronary artery disease. Journal of nuclear medicine : official publication, Society of Nuclear Medicine, 43(12), 1634–1646.
3. Sicari, R., Nihoyannopoulos, P., Evangelista, A., Kasprzak, J., Lancellotti, P., Poldermans, D., Voigt, J. U., Zamorano, J. L., & European Association of Echocardiography (2009). Stress Echocardiography Expert Consensus Statement--Executive Summary: European Association of Echocardiography (EAE) (a

registered branch of the ESC). European heart journal, 30(3), 278–289. https://doi.org/10.1093/eurheartj/ehn492

4. Hossain, N., Hossain, N., Al-Sadawi, M., & Haq, S. (2020). Bezold-Jarisch reflex-mediated asystole during dobutamine stress testing: a case report. European heart journal. Case reports, 4(6), 1–6. https://doi.org/10.1093/ehjcr/ytaa348
5. Eroglu, S., Sade, L. E., Polat, E., Bozbas, H., & Muderrisoğlu, H. (2015). Association between coronary flow reserve and exercise capacity. Hellenic journal of cardiology : HJC = Hellenikekardiologikeepitheorese, 56(3), 201–207.
6. Kamperidis, V., Giannakoulas, G., Vlachou, M., Ntourtsiou, S., Sianos, G., &Karvounis, H. (2017). Paradoxical bradycardia and blood pressure elevation during dobutamine stress echocardiography reveals ischemia in a patient with syncope. Hellenic journal of cardiology : HJC = Hellenikekardiologikeepitheorese, 58(4), 291–293. https://doi.org/10.1016/j.hjc.2016.11.025
7. Hofmeister, E. H., Keenan, K., & Egger, C. M. (2005). Dobutamine-induced bradycardia in a dog. Veterinary anaesthesia and analgesia, 32(2), 107–111. https://doi.org/10.1111/j.1467-2995.2004.00151.x
8. Takeuchi, M., Hanada, H., & Numata, T. (1998). Is dobutamine-induced sinus node deceleration a marker of significant stenosis of the right coronary artery?. Chest, 113(2), 306–311. https://doi.org/10.1378/chest.113.2.306
9. Holland, E. M., & Stouffer, G. A. (2022). Bezold-Jarisch Reflex. Journal of the Society for Cardiovascular Angiography & Interventions, 1(2).
10. Olszowska, M., Musiałek, P., Drwiła, R., &Podolec, P. (2012). Progressive bradycardia with increasing doses of dobutamine leading to stress echo interruption. Cardiology journal, 19(1), 79–80. https://doi.org/10.5603/cj.2012.0012

****