Available online at www.medicinescience.org

CASE REPORT

Medicine Science International Medical Journal

Medicine Science 2018;7(3):694-6

Peripartum noncompaction cardiomyopaty:new diagnosed after pregnancy

Ramazan Asoglu[1], Serdar Turkmen[1], Erdal Akturk[1], Abdulmecid Afsin[2]

[1]Adiyaman Health Education and Research Hospital, Department of Cardiology, Adiyaman, Turkey
[2]Kahta Community Hospital, Department of Cardiology, Adiyaman, Turkey

Received 31 January 2017; Accepted 20 February 2018
Available online 15.05.2018 with doi: 10.5455/medscience.2017.07.8803

Abstract
Left-ventricular non-compaction (LVNC) cardiomyopathy is an arrest in the normal process of myocardial compaction and few cases have been reported just after pregnancy. In this case report, we present a patient who had heart failure symptoms at first month of after 38 weeks pregnancy. Echocardiography revealed prominent trabeculation and deep intertrabecular recesses at the left ventricular with an impaired ejection fraction. There is no specific treatment for LVNC just after pregnancy besides the usual management of dilated cardiomyopathy.

Keywords: Left ventricular non-compaction (LVNC), pregnancy, heart failure, prognosis

Introduction

Left ventricular non-compaction (LVNC) cardiomyopathy is a rare congenital cardiomyopathy and prevalence is 0.05% in adults [1]. Familial occurrence of LVNC has been reported with an incidence as high as 44% [2]. Echocardiography and Magnetic resonance imaging (MRI) usually use for diagnosis [3]. The presentation may follow heart failure, thrombus embolism, and ventricular arrhythmias [4] and is associated with poor prognosis [5]. LVNC with pregnancy is rare to observe [6]. We present the case of a patient who referred to our clinic with heart failure after pregnancy and prominent trabeculation in the lateral part of the left ventricular cavity observed with echocardiographic and MRI examination.

Case report

A 25-year-old pregnant woman was referred to our clinic with effort dyspnea at first-month after 38 weeks' gestation of her third pregnancy for further evaluation. She underwent elective caesarian section at 38 weeks with delivery of a healthy infant. She felt progressive dyspnea during last month after delivery although she had mild symptoms during pregnancy.

Her medical history was unremarkable and there was no family history. On physical examination, blood pressure was 130/80 mmHg, heart rate was 90 beats/minutes, and she had crepitation rales in both basal of the lung. She had not got jugular venous distention and cardiac murmur. Her chest radiography showed mild cardiomegaly.

Echocardiography revealed a dilated left ventricle (end systolic diameter 50 mm) with an ejection fraction of 30%, global hypokinetic, moderate mitral and tricuspid regurgitation, and pulmonary artery systolic pressure was estimated at 45 mmHg from tricuspid regurgitation. Echocardiography demonstrated prominent trabeculations predominantly over the left ventricle lateral wall, with color Doppler examination showing penetration of blood flow into the sinusoidal recess formed by these trabeculations (Figure.1). The ratio of the non-compacted to compacted layers was >2. An electrocardiogram showed sinus rhythm with non-specific T wave changes.

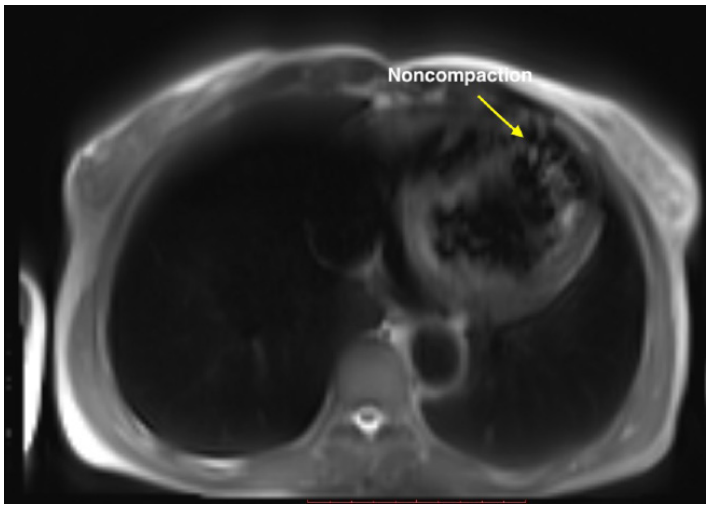

Figure 1. Echocardiography Showing Increased Left Ventricular Trabeculation

***Coresponding Author:** Ramazan Asoglu, Adiyaman Health Education and Research Hospital, Department of Cardiology, Adiyaman, Turkey
E-mail: koroner63@yahoo.com

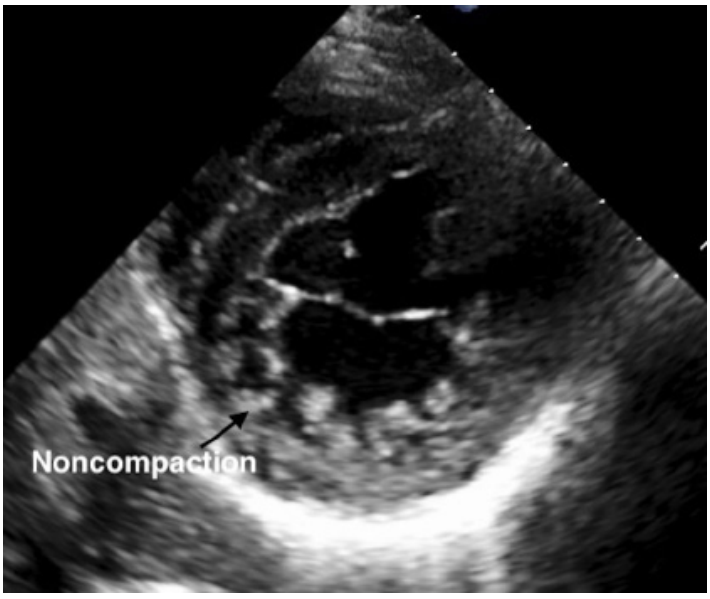

Figure 2. Magnetic Resonance Imaging Showing Increased Trabeculation

The patient was treated with angiotensin converting enzyme inhibitors, beta-blockers, spironolactone, and diuretics. Lactation is an additional stressor to exacerbate heart failure post-delivery and suppress with bromocriptine. A pediatric cardiologist examined the baby and echocardiography findings were normal. Anticoagulant and anti-arrhythmic therapy was not given. The patient's ejection fraction had not improved at her two-month follow-up examination and complained tachycardia. Rhythm holter was performed and non-sustained ventricular tachycardia observed three times. Single Lead Implantable Cardioverter-Defibrillator (ICD) was implanted to prevent sudden cardiac death. She was stable in New York Heart Association class I and genetic analyses declined by the patient.

Discussion

Left Ventricular Non-Compaction Cardiomyopathy is caused by an arrest in the normal process of endomyocardial morphogenesis and characterized with multiple prominent trabeculations and deep inter-trabecular recesses communicating with the ventricular cavity [2,3]. Patient with LVNC may be present with heart failure, thrombus embolism, and ventricular arrhythmias [4]. In this report we present a patient with LVNC in postpartum period. The patient had attributed her symptoms only mild during pregnancy and increased gradually until first month after delivery. LVNC is diagnosed by two-dimensional and color Doppler echocardiography [5]. Echocardiographic features described include multiple predominant ventricular trabeculations, multiple deep intratrabecular recesses communicating with the ventricular cavity visualized with color Doppler, ventricular dilatation, and reduced systolic and diastolic ventricular function [6]. Jenni and colleges suggested the detection of two myocardial layers, non-compact and compact, in short axis views of the left ventricle in end-diastole and LVNC is diagnosed according to the ratio of these layers [4,7]. Other congenital heart defects are accompanied with NCLV and our patient also had a patent foramen ovale.

During echocardiography examination, color doppler images showed patent foramen ovale and contrast echocardiography performed to define the diagnosis. Without associated congenital heart defects is designated as isolated NCLV.

In this case, the diagnosis of LVNC was made by echocardiography and MRI. Echocardiography showed prominent trabeculations in the lateral wall and apex of left ventricular cavity and Doppler echocardiography showed blood flow between the trabeculations. Trabeculations localization was consistent with previous work by Boyd et all [7]. Echocardiography and MRI showed similar trabeculations in this patient (Figure. 1) (Figure. 2).

Peripartum Cardiomyopathy is a form of idiopathic primary myocardial disease related with pregnancy. Although several etiological mechanisms have been suggested, none of them is definite. Some of them are Myocarditis, Cardiotropic viral infections [8] Apoptosis and Inflammation [9], and an abnormal hemodynamic response [10]. The European Society of Cardiology defined peripartum cardiomyopathy is a form of dilated cardiomyopathy with signs of heart failure in the last month of pregnancy or within six months of delivery [11]. Increased blood volume and cardiac output may precipitate heart failure symptoms such as dyspnea, dizziness, pedal edema, and orthopnea during pregnancy [12]. Kilic and colleges suggested if the symptoms develop suddenly or become serious further evaluation is needed [5]. In our case, it was not possible to determine this clinical presentation associated with LVNC or peripartum cardiomyopathy.

LVNC has been associated with high incidence of complications such as cardiac failure (%53), ventricular tachycardia (41%), thromboembolism (24%), and sudden death (16%) [13]. Cardiovascular complications such as decompensated heart failure, embolic event, ventricular arrhythmia and NYHA class III or greater at presentation were the strongest predictors of adverse outcomes [14]. This patient did not have these adverse predictive features. Given therapy for LVNC contains of heart failure, arrhythmia, and prevention of thromboembolism [15]. Heart failure related with LVNC can be effectively treated with angiotensin converting enzyme inhibitors, beta-blockers, and diuretics and arrhythmias are treated with various anti arrhythmic drugs [15].

This patient had an EF of 30% and she was in NYHA class I. She accepted the heart failure treatment and comply the medication at the last clinical follow up.

Conclusion

Left Ventricular Non-Compaction is rarely diagnosed during and after pregnancy and rare causes should be kept in mind when evaluating the heart failure symptoms. Awareness of familial occurrence should be considered for early diagnosis of LVNC.

Competing interests
The authors declare that they have no competing interest.

Financial Disclosure
The financial support for this study was provided by the investigators themselves.

References

1. Ritter M, Oechslin E, Sütsch G, Attenhofer C, Schneider J, Jenni R. Isolated noncompaction of the myocardium in adults. Mayo Clin Proc. 1997;72:26–31.

2. Pignatelli RH, McMahon CJ, Dreyer WJ, et al. Clinical characterization of left ventricular noncompaction in children: a relatively common form of

cardiomyopathy. Circulation. 2003;108:2672–8.

3. Richardson P, McKenna W, Bristow M, et al. Report of the 1995 world health organization/international society and federation of cardiology task force on the definition and classification of cardiomyopathies. Circulation. 1996;93:841-2.

4. Kohli SK, Pantazis AA, Shah JS, et al. Diagnosis of left-ventricular non-compaction in patients with left-ventricular systolic dysfunction: time for a reappraisal of diagnostic criteria? Eur Heart J. 2008;29:89-95.

5. Kilic ID, Tanriverdi H, Evrengul H. Left ventricular non-compaction in pregnancy. Cardiovasc J Afr. 2013;24:e1-2.

6. Alizad A, Seward JB. Echocardiographic features of genetic diseases: part 1. Cardiomyopathy. J Am Soc Echocardiogr. 2000;13:73-86.

7. Boyd MT, Seward JB, Tajik AJ, et al. Frequency and location of prominent left ventricular trabeculations at autopsy in 474 normal human hearts: implications for evaluation of mural thrombi by two-dimensional echocardiography. J Am Coll Cardiol. 1987;9:323-6.

8. Bültmann BD, Klingel K, Näbauer M, et al. High prevalence of viral genomes and inflammation in peripartum cardiomyopathy. Am J Obstet Gynecol. 2005;193:363-5.

9. Narula J, Haider N, Virmani R, et al. Apoptosis in myocytes in end-stage heart failure. N Engl J Med. 1996;335:1182-9.

10. Mabie WC, DiSessa TG, Crocker LG, et al. A longitudinal study of cardiac output in normal human pregnancy. Am J Obstet Gynecol. 1994;170(3):849-56.

11. Sliwa K, Hilfiker-Kleiner D, Petrie MC, et al. Heart failure association of the european society of cardiology working group on peripartum cardiomyopathy. current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the heart failure association of the european society of cardiology working group on peripartum cardiomyopathy. Eur J Heart Fail. 2010;12:767–78.

12. Ramaraj R, Sorrell VL. Peripartum cardiomyopathy: Causes, diagnosis, and treatment. Cleve Clin J Med. 2009;76:289–96.

13. Oechslin EN, Attenhofer Jost CH, Rojas JR, Kaufmann PA, Jenni R. Long-term follow-up of 34 adults with isolated left ventricular noncompaction: a distinct cardiomyopathy with poor prognosis. J Am Coll Cardiol. 2000;36:493–500.

14. Greutmann M, Mah ML, Silversides CK, Klaassen S, Attenhofer Jost C, Jenni R, Oechslin E. Predictors of adverse outcome in adolescents and adults with isolated left ventricular noncompaction. Am J Cardiol. 2012;109:276-81.

15. Stöllberger C, Finsterer J. Left ventricular hypertrabeculation / noncompaction. J Am Soc Echocardiogr. 2004;17:91-100.