COR ET VASA 55 (2013) E236–E241

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Review Article

Isolated non-compaction cardiomyopathy: A review

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ARTICLE INFO

Article history:
Received 16 October 2012
Accepted 1 November 2012
Available online 10 November 2012

Keywords:
Left ventricular non-compaction cardiomyopathy
Genetics
Echocardiography
Cardiac magnetic resonance
CMRI tomography

ABSTRACT

Left ventricular non-compaction cardiomyopathy (LVNC) is a rare disease which belongs to unclassified congenital cardiomyopathies. According to the ESC classification, LVNC is characterized by a spongy appearance of myocardium due to increased trabeculation and deep intertrabecular recesses that communicate with the left ventricle. This phenotype is thought to be caused by arrest of normal endomyocardial morphogenesis. Clinical manifestations of LVNC include heart failure, thromboembolic events, arrhythmias and/or sudden cardiac death. Progression of LVNC is highly variable and prediction of prognosis is very difficult. The aim of this paper is to provide an update about the topic of isolated LVNC.

Contents

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http://dx.doi.org/10.1016/j.crvasa.2012.11.001

1. Introduction

Left ventricular non-compaction cardiomyopathy (LVNC) is a rare condition which, according to the European Society of Cardiology classification, belongs to unclassified cardiomyopathies with a genetic etiology [1]. Localized thickening of left ventricular myocardium formed by thick spongiform myocardium from the endocardial side and thin compact epicardial layer is a typical feature of LVNC. Spongiform layer is made up from densely trabeculated myocardium with typical deep intertrabecular recesses communicating with the left ventricular cavity [2,3]. The condition can be found as an isolated disorder of myocardium (isolated LVNC) or may be associated with congenital heart defects such as pulmonary atresia, left or right ventricular outflow tract obstruction, atrial or ventricular septal defect or Ebstein's anomaly [2–4]. The aim of this review is to provide the summary of current knowledge of the isolated form of LVNC.

2. History

Non-compact myocardium was first described in 1932 when Bellet et al. reported this anomaly in newborn with aortic atresia and coronary artery-left ventricular fistula [1]. In 1975, Czech authors Dušek et al. described first series of patients with non-compact myocardium, some of whom would be nowadays diagnosed with LVNC [5]. First clinical description of LVNC diagnosed by echocardiography was published by Engberding in 1984 [6]. The term isolated LVNC was proposed by Chin et al. in 1990, who reported the first series of clinically diagnosed cases [7]. Until then, this condition was designated as spongious myocardium, myocardial dysplasia, myocardial dysgenesis or persistence of myocardial sinusoids [4]. In 1996, LVNC was included among unclassified cardiomyopathies according to the WHO classification of cardiomyopathies [8].

3. Embryology

Most authors suppose that LVNC is determined by disturbance of embryogenesis [4] and its morphological features result from increased trabeculation or arrest of myocardial compaction. During the 4th week of human embryogenesis, clonal proliferation of trabecules occurs and spongiform myocardium develops [9]. In this period, myocardium is formed by a meshwork of interwoven trabecules and cardiomyocytes are nourished by diffusion from circulating blood. Within the 5th–8th week, the coronary bed develops and during this process intertrabecular spaces for capillaries form. Subsequently, progressive fusion of myocardial trabecules occurs and compact myocardium prevails over the sponge-like one. The formation of the compact layer happens relatively fast within 10th–12th week and proceeds from left ventricular basis to the apex, from epicardium towards endocardium [9]. In accordance with the process of embryogenesis, excessive trabecularization in LVNC is localized usually in the apical area and adjacent segments of lateral and inferior wall of left ventricle. Sarcomere proteins mutations—the most common cause of LVNC, may cause excessive trabeculation or insufficient compaction of myocardium and imbalance between these processes leads to LVNC [10]. Sarcomere proteins mutations cause hypertrophic and dilated cardiomyopathy as well, therefore it is not surprising that these conditions can be diagnosed by screening in families with LVNC. Association of myocardial myosin heavy chain mutations (MYH7) and myocardial actin mutations (ACTC1) with non-compact myocardium, atrial and ventricular septal defects has been confirmed. It seems that correct sarcomere function is important for the accurate septation of the heart. This explains why LVNC is associated with congenital heart defects in some cases [10].

4. Anatomical pathology, histology

Anatomical criteria for diagnosis of LVNC were defined by Burke et al. [11]. Pathologicoanatomical diagnosis requires two criteria to be fulfilled: (a) absence of normally formed papillary muscles in left ventricle, (b) ratio of total left ventricular wall thickness to compact myocardium greater than 2. As far as right ventricle is concerned, at least 75% of wall thickness must be formed by highly trabecularized myocardium. Fig. 1(panel C) shows macroscopic finding of the heart with LVNC, explanted by transplantation. Microscopically, there is often endocardial thickening, endocardial fibrosis or fibroelastosis, microinfarctions and foci of interstitial fibrosis in LVNC [12]. In some cases, subendocardial myocytes present perinuclear or extensive central vacuolization which shows sublethal ischemic injury to myocytes, caused by microvascular dysfunction in sponge-like myocardial layer [13].

5. Genetics

LVNC is a condition with prevailing familial incidence, sporadic form is less frequent. Careful cardiac screening showed familial incidence in up to 63% of cases [14]. Genetic analyses proved that the most common cause of LVNC is sarcomere protein mutation. According to some studies, these are detected in up to 29% of cases [15]. In general, the most common mutation was MYH7, myosin- binding protein C (MYBPC3), alpha tropomyosin (TMP1), myocardial actin (ACTC1), troponin T (TNNT2) and myocardial troponin I (TNNI3) [14–18]. Familial LVNC accompanied by occurrence of hypertrophic, dilated and rarely restrictive cardiomyopathy is typical for MYH7 mutation [17]. In ACTC1 mutation, apical forms of hypertrophic cardiomyopathy were found in families with LVNC [18]. Less common genetic disorders in patients with LVNC include mutations of Z-band proteins such as Cypher/ZASP protein [19], cytoskeleton proteins mutations, i.e. alpha-dystrobrevin (DTNA) [20] or proteins taking part in calcium transportation, i.e. calsequestrin (CASQ2) and phospholamban (PLN) [14], nuclear membrane proteins (lamin A/C) [21] or mitochondrial enzymes such as protein G 4.5 belonging to tafazins [22] and some further mutations [23]. These disorders are usually transmitted

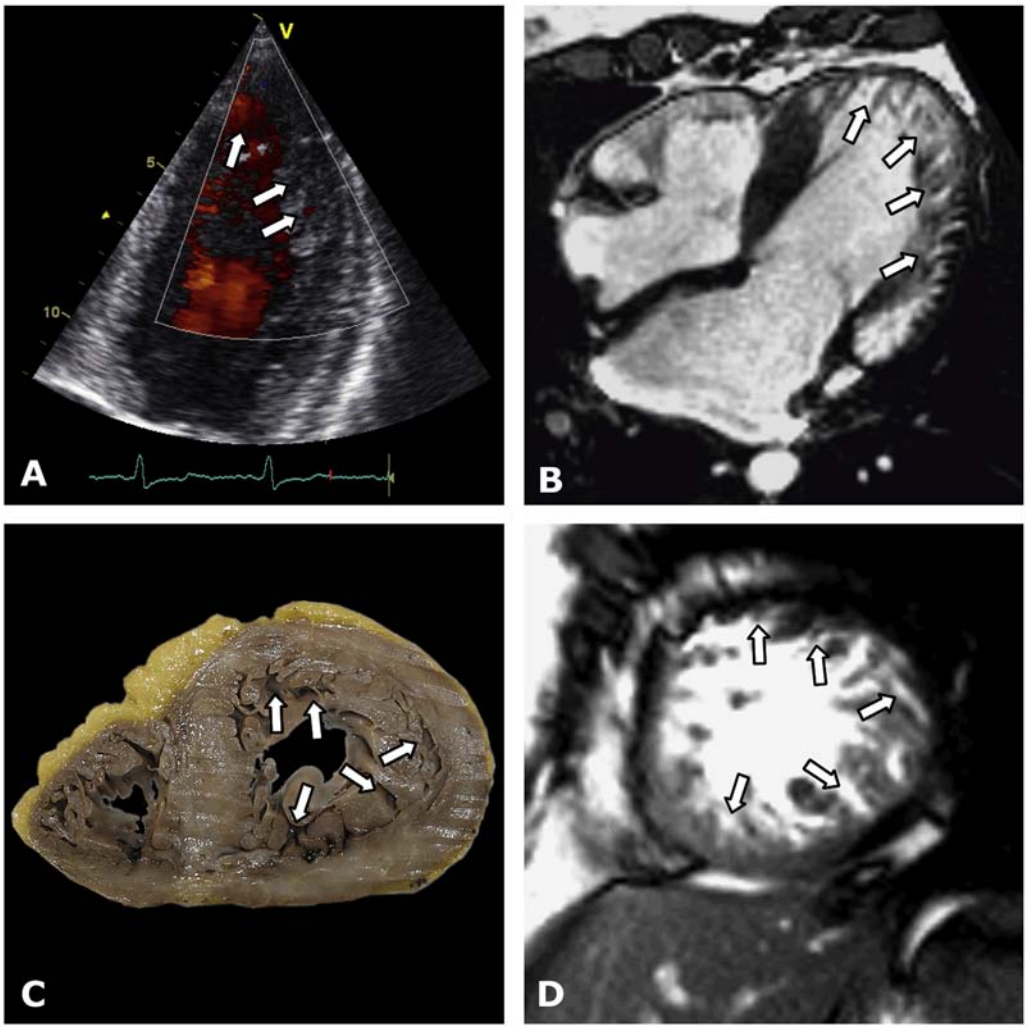

Fig. 1 – Examples of characteristic findings in patient with LVNC. (Panel A): Echocardiographic image of non-compact myocardium in apical and lateral part of left ventricular wall. Color Doppler flow mapping depicts the blood flow in spongiform myocardium. (Panel B): Magnetic resonance (T1-weighted image with TrueFISP sequence) illustrating non-compact myocardium in the four-chamber view. (Panel C): Macroscopic finding of the heart explanted by transplantation (apical third of ventricles, Sikl's technique). (Panel D): Magnetic resonance (T1-weighted image with TrueFISP sequence) documenting the non-compact myocardium in the apical third of left ventricle (short axis view). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

through autosomal dominant inheritance with incomplete penetrance. LVNC with G4.5 gene defect causing Barth syndrome in children is an exception, as it is gonosomally transmitted. Dilated cardiomyopathy or LVNC at early age in men, frequently accompanied by neutropenia, lactic acidosis and lipid metabolism abnormalities are typical for this mutation [22]. Currently it is recommended to screen at least three generations of families of patients with LVNC and to perform cardiac evaluation in close relatives [24].

6. Diagnostics

Echocardiography is a basic diagnostic tool for LVNC. Thickening of the left ventricular wall in the apical part below the papillary muscles is typical for LVNC. It is formed by thick sponge-like myocardial layer from the endocardial side with thin compact epicardial layer. Sponge-like layer is interwoven with deep recesses communicating with the main cavity of the left ventricle, this can be verified by color Doppler flow mapping (Fig. 1panel A). In symptomatic individuals, these findings are accompanied by left ventricular systolic or diastolic dysfunction or by secondary atrioventricular regurgitations. Historically, criteria stated by Chin et al. [7], Stollberger et al. [25] and Jenni et al. [26] were applied for diagnostics of LVNC. According to Chin, ratio of compact myocardial layer to total myocardial thickness lower than 0.5 suggests LVNC. Echocardiographic images should be assessed from parasternal and apical four-chamber view at the end of diastole [7]. Stollberger's criteria [25] are rather qualitative. They are based on presence of three and more trabecularizations entering the left ventricular cavity visualized at single echocardiographic view. Further criterion is the presence of deep intertrabecular recesses communicating with left ventricular cavity on Doppler imaging. Jenni's criteria

are the most commonly used. The assessment of spongiform and compact layer ratio is performed at the end of systole from parasternal short axis view. Ratio of non-compact to compact layer is considered to be diagnostic when greater than 2.0 in adults and 1.4 in children. For the diagnosis of isolated LVNC, presence of multiple trabecularizations in the apex and adjacent segments of lateral and inferior wall is required, as well as proof of blood circulation between the intertrabecular recesses and left ventricular cavity on Doppler flow imaging and absence of other abnormalities [26]. Contrast echocardiography may help in cases with poor standard echo visibility [27,28].

Presently, reliability of the stated diagnostic criteria is controversial. Kohli et al. applied the above-mentioned criteria to 199 patients with heart failure. Chin's criteria were fulfilled in 79%, Jenni's criteria in 64% and Stollberger's criteria in 53% of cases. All criteria were met in only 30% of cases. In the group of volunteers, at least one criterion for LNVC was fulfilled in 14% of black patients and 3% of rest of the patients [29]. It shows that current diagnostic criteria are overly sensitive and may lead to overestimation of LVNC prevalence. Quantification of non-compact myocardium by 3D echocardiography [30] or cardiac magnetic resonance (CMRI) could offer a solution (Fig. 1panels B and D). Future studies should include the patients with LVNC backed by positive family history and positive genetic testing, respectively. As mentioned, CMRI can be helpful due to 3 D-imaging and better spatial resolution. In the work of Petersen et al., group of 7 patients with LVNC was compared to control groups of healthy individuals, athletes, hypertensive patients, patients with hypertrophic cardiomyopathy and aortic stenosis. CMRI distinguished patients with LVNC with high sensitivity and specificity (86% and 99%) [31]. The diagnostic criterion for LVNC was the non-compact to compact myocardium thickness ratio in diastole greater than 2.3. Jacquier et al. used CMRI for quantitative evaluation of the spongiform myocardial mass in LVNC. Volume of trabecularized mass larger than 20% of total left ventricular mass distinguished patients with LVNC with 94% sensitivity and specificity [32]. These results signalize that quantitative evaluation of the non-compact mass could become a new standard for LVNC diagnostics. The thickness of trabecularized and compact layer of left ventricular wall were studied in large group of healthy individuals by CMRI and normal values have been established. From 4th decennium on, decrease in thickness of trabecularized layer and increase in that of compact mass has been described [33]. Other authors depicted relation between delayed accumulation of gadolinium and severity of heart failure in LVNC [34–36]. There is less information concerning the use of computed tomography (CT) in LVNC diagnostics. The asset of CT is a possibility of displaying the coronary arteries and possible associated inborn anomalies (i.e. pulmonary stenosis, patent ductus arteriosus)

7. Epidemiology and natural course of LVNC

Prevalence of LVNC in retrospective studies lied between 4.5 and 26 in 10,000 individuals who had been sent for echocardiographic evaluation [37–39]. In the group of patients with ejection fraction lower than 45%, LVNC was diagnosed in 3.7% [39]. In children, LVNC is the third most common cardiomyopathy, representing approximately 9% of all cardiomyopathies [40]. Clinical presentation is an important factor concerning the analysis of incidence and prognosis. The main clinical symptoms of LVNC are: signs of heart failure, arrhythmias, chest pain and systemic embolizations [2,3]. First groups of patients included only severely symptomatic patients in advanced stages with 58% rate of 5-year survival. Sudden cardiac death, severe heart failure, systemic embolizations or necessity of cardiac transplantation were frequent [37–39,41]. Asymptomatic patients in initial phases of LVNC were included into later studies, in accordance; 5-year prognosis was more favorable with 75% rate of survival [42–44]. In the work of Lofiego et al., advanced heart failure of NYHA III-IV class, sustained ventricular tachycardias and left atrium size were independent predictors of unfavorable prognosis [43]. In the study of Habib et al., NYHA class III-IV, high filling pressures of left ventricle, its ejection fraction and hospitalization due to heart failure were the predictive factors [44]. B-natriuretic peptide assessment can be applied for risk stratification of LVNC [45].

8. Clinical presentation and treatment

Clinical presentation of LVNC illustrated by Habib's study [44] shows that heart failure is the main symptom of LVNC. 43% of patients with LVNC presented with heart failure and left ventricular ejection fraction was lower than 30% in 46% of patients. Other symptoms leading to diagnosis of LVNC were arrhythmias (22%), familial screening (8%) and embolization (4%). During the average 2.3-year follow up, advanced heart failure developed in 31% of cases, in 8% cardiac transplantation was performed, in 7% malignant ventricular arrhythmias were documented and in 28% of cases, cardioverter-defibrillator was implanted (ICD). Atrial fibrillation was documented in 7% of cases and in 5% ischemic stroke occurred.

Pharmacologic treatment of heart failure in LVNC follows the general rules for chronic heart failure [46]. In group of patients with LVNC, favorable effect of cardiac resynchronization therapy has been reported and the rate of adequate ICD interventions after implantation for primary prevention was 33% during 40-month follow-up. Therefore, in patients with LVNC and severe left ventricular systolic dysfunction, early ICD implantation is recommended [47]. Defibrillation threshold testing should be performed on efficient anticoagulation therapy or should not be performed at all due to high risk of thromboembolic event. In severe heart failure, mechanical circulatory support device implantation or heart transplantation should be considered. Therapy of supraventricular and ventricular arrhythmias follows the same rules as arrhythmias by other structural affections of the heart. Anticoagulation therapy is recommended for patients with LVNC and history of systemic embolization, atrial fibrillation or ejection fraction lower than 40%. For the rest of the patients, acetylsalicylic acid is recommended as thromboembolic prevention [3,42]. Careful clinical screening in closest relatives of LVNC patients is important, as it can reveal individuals with the same diagnosis or different cardiomyopathy.

9. Conclusion

In years to come, efforts to improve diagnostics of LVNC thanks to more exact quantification of non-compact cardiomyopathy are expected. The progress in genetic analysis could contribute to better diagnostics and possibly to prognostic stratification and screening of LVNC in families. It is important to ensure systematic clinical screening in families of patients with LVNC and early treatment of newly diagnosed individuals.

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