Electrocardiographic findings in correlation to magnetic resonance imaging patterns in African patients with isolated ventricular noncompaction

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ABSTRACT

Objective: Isolated ventricular noncompaction is a rare primary genetic cardiomyopathy characterized by persistent embryonic myocardial morphology without any other cardiac anomalies. Arrhythmias are frequently present, including both tachyarrhythmia and conduction disturbance. Our study aimed to describe the electrocardiographic findings and to correlate them with the clinical presentation and cardiac magnetic resonance imaging findings.

Methods: We retrospectively reviewed 24 patients diagnosed with isolated ventricular noncompaction (IVNC) by cardiac magnetic resonance imaging. Correlations were investigated between arrhythmias and the site of ventricular noncompaction, number of noncompacted segments, presence of fibrosis, and left ventricular dysfunction.

Results: The mean age was 42.7±13.1 years. Patients were first presented with heart failure in 41.7% and arrhythmia in 45.8%. Electrocardiogram was abnormal in 91.6% of patients; the most common anomaly was left bundle branch block (LBBB) (41.7%), followed by supraventricular arrhythmias (29.1%), repolarization abnormalities (29.1%), and ventricular tachycardia (20.8%). A normal left ventricular systolic function was frequently observed in patients who first presented with rhythm disorders than heart failure (p=0.008). There was also a delayed diagnosis of IVNC when presented with arrhythmia versus heart failure (p=0.02). We found no correlation between arrhythmias and the noncompaction site or fibrosis, except for LBBB, which was associated to left ventricle lateral wall involvement (p=0.028). No correlation between systolic dysfunction and the number of noncompacted segments, fibrosis, or arrhythmia was demonstrated.

Conclusion: While electrocardiographic abnormalities are frequent in isolated ventricular noncompaction, no specific patterns were identified. More large studies are needed for stratification of arrhythmic risk of this highly arrhythmogenic substrate.

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Keywords: cardiac magnetic resonance imaging, electrocardiogram, isolated ventricular noncompaction

Introduction

Isolated ventricular noncompaction (IVNC) is a rare primary genetic cardiomyopathy characterized by persistent embryonic myocardial morphology without any other cardiac anomalies (1). Prominent left ventricular trabeculation and deep intertrabecular recesses communicating with the ventricular cavity and severely altering myocardial structure are the characteristics of this cardiomyopathy (2).

The clinical presentation of the disease is highly variable, ranging from coincidental discovery in asymptomatic patients to severe heart failure and even sudden death (2-5). Arrhythmias are frequently present, including both tachyarrhythmia and conduction disturbances, and progressive ischemia and interstitial fibrosis may be the underlying patho-anatomic correlate (6-8). Cardiovascular magnetic resonance (CMR) has been suggested as the technique of choice for ventricular noncompaction diagnosis, providing information on myocardial morphology and documenting the presence and extent of myocardial fibrosis through delayed-enhancement sequences (9-12).

Previous studies (6, 7) described and investigated correlations between arrhythmias and echocardiographic findings in patients with isolated ventricular noncompaction, whereas in our study, we described the electrocardiographic (ECG) findings

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in 24 African patients diagnosed with isolated ventricular non-compaction by CMR and correlated these patterns with the clinical presentation and mainly with CMR imaging findings.

Methods

Study population

We retrospectively reviewed all patients referred to our department with a suspected diagnosis of IVNC, confirmed by CMR, between 2009 and 2012. Twenty-four patients were enrolled, and all of the population study underwent 12-lead electrocardiogram, 24-hour Holter ECG monitoring, echocardiography, and CMR imaging. According to clinical judgment, invasive coronary angiography was performed in selected patients to exclude coronary artery disease.

ECG analysis

The 12-lead resting electrocardiogram and 24-hour Holter ECG monitoring were analyzed by the same reader. The following variables were assessed and measured: heart rate; rhythm; PR interval duration; P-wave amplitude in lead V1 and in inferior leads; QRS complex duration; presence of bundle branch block; maximum QRS amplitude; left ventricle hypertrophy, determined by Sokolow-Lyon voltage criteria and Cornell voltage-duration criteria; ST segment/T wave abnormalities, defined as asymmetrical inversion of the T wave ≥0.1 mV deep in two or more leads except aVR, V1, and V2; and ST-segment depression ≥0.1 mV at 0.08 s from the j point. Convex ST-T segment elevation ≥0.2 mV; QT interval corrected for cardiac frequency.

Cardiac magnetic resonance imaging

Acquisition protocol

Magnetic resonance imaging (MRI) was performed on a 1.5-Tesla magnetic resonance scanner (Siemens Medical Systems, Symphony Maestro Class Tim, Erlangen, Germany) and phased array surface coil. The protocol included several magnetic resonance imaging sequences. Images were obtained in two-chamber, four-chamber, and short-axis (from the atrioventricular ring to the apex) planes. Ten minutes after intravenous injection of gadolinium, delayed-enhancement magnetic resonance imaging was performed using an inversion recovery-prepared gated fast gradient-echo pulse sequence. The late gadolinium enhancement (LGE) images were acquired in end-systole in the same view as used for cine images. Cine images were analyzed using Argus post-processing software (Siemens Medical Systems). All patients had CMR imaging.

IVNC, left ventricle ejection fraction, and LGE

The diagnosis of IVNC was performed on short- and long-axis slices in the presence of the following criteria [this is in keeping with the CMR diagnostic criteria for IVNC proposed by Peterson (13)]: the presence of a two-layer structure, with a thin epicardial compacted layer and a thick noncompacted endocar-

dial one; the presence of marked trabeculation and deep inter-trabecular recesses within the noncompacted layer; and a noncompacted-to-compacted myocardial ratio of >2.3 as measured in end-diastole. Segmental analysis was assessed using the 17-segment cardiac model, as defined by the American Heart Association/American College of Cardiology statement for standardized myocardial segmentation (14). Left ventricular ejection fraction (LVEF) was measured using standard volumetric techniques with dedicated software. We considered LVEF <50% as left ventricular systolic dysfunction. Patterns of LGE were visually classified as sub-endocardial, sub-epicardial, mid-myocardial, or transmural.

Statistical analysis

Continuous variables are expressed as means±standard deviations (or median with inter-quartile range (IQR) when the distribution was non-normal), and categorical variables are expressed as percentages. We used the Kolmogorov-Smirnov test for checking the normality. To compare between groups, the continuous variables were analyzed using Mann-Whitney U test, and the categorical variables were analyzed using the chi-square test. A p<0.05 was considered statistically significant. The statistical analysis was performed using Statistical Package for the Social Sciences (SPSS), version 18.0 (SPSS Inc., Chicago, IL, USA).

Results

A total of 24 patients fulfilled the diagnostic criteria of IVNC and were entered in the study. The age at the time of diagnosis ranged from 14 to 68 years; the mean age was 42.7±13.1 years, and 16 (66.7%) patients were male. They had a low cardiovascular risk profile: 12.5% had hypertension, 16.7% was diabetic, and 4.2% had obesity. Most patients (95.5%) were symptomatic. The first presentation symptom was dyspnea in 45.8%, followed by palpitation (33.3%), syncope (8.3%), and chest pain (4.2%). The average time delay from the first symptom to diagnosis was 23.1 months. Patients with complaints of dyspnea had a low ejection fraction compared with other presentations (p=0.003).

A normal ECG was found only in 2 patients (8.3%). The most common anomaly was intraventricular conduction delay, observed in 12 patients (50%). One patient had syncopal complete sinoatrial block and was implanted with a permanent pacemaker. Two patients with complete left bundle branch block (LBBB) and low ejection fraction had resynchronization therapy.

Supraventricular arrhythmias (SVAs) occurred in 7 patients (29.1%), and ventricular tachycardia (VT) occurred in 5 patients (20.8%). Repolarization abnormalities were observed in 7 patients (29.1%); they were present, even in the absence of bundle branch block or ventricular hypertrophy.

Only one patient had Wolff-Parkinson-White (WPW) syndrome. The diagnosis was revealed by supraventricular tachycardia; he subsequently underwent ablation of the accessory pathway without success. Table 1 summarizes the characteristics of the population study.

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Eleven patients (45.8%) underwent *coronarography*; we diagnosed no significant coronary artery disease.

The apical level of the lateral wall was the more commonly affected by noncompaction (100% of patients), followed by the mid-ventricular level of the same wall (95.8% of patients). Infero-septal wall involvement in the basal level was not found in any patient. Right ventricular noncompaction was noted in 7 (29.1%) patients. The mean number of left ventricle noncompacted segments per

Table 1. Demographic, clinical, electrocardiographic, and cardiac magnetic resonance imaging characteristics of isolated left ventricular noncompaction patients

Status at first consultation	n (%)
Demographic characteristics	
Male	16 (66.7%)
Mean age at diagnosis	42.7±13.1
Mode of presentation	
Heart failure	10 (41.7%)
Arrhythmia	11 (45.8%)
Thromboembolism	1 (4.2%)
Electrocardiogram	
Abnormal electrocardiogram	22 (91.6%)
First-degree atrioventricular block	3 (12.5%)
Complete sinoatrial block	1 (4.2%)
Atrial fibrillation	4 (16.7%)
Atrial tachycardia	3 (12.5%)
Left bundle branch block	10 (41.7%)
Right bundle branch block	2 (8.3%)
Ventricular tachycardias	2 (8.3%)
Non-sustained ventricular tachycardia	3 (12.5%)
Repolarization abnormalities	7 (29.1%)
Wolff-Parkinson-White syndrome	1 (4.1%)
Left ventricle ejection fraction >50%	9 (37.5%)

patient was 9.29±3.78, and the mean non-compacted/compacted myocardium ratio was 2.91±0.67, with a maximum of 4.5.

The mean ejection fraction was 37.79%±13.85, ranging from 10% to 60%. Fifteen patients (62.5%) had an impaired ejection fraction (<50%). No correlation between systolic dysfunction and the number of noncompacted segments or arrhythmias was demonstrated. The occurrence of supraventricular arrhythmias was also independent of age and atrial size.

We found no correlation between arrhythmia and the non-compaction site. However, LBBB was frequently associated with left ventricle lateral wall noncompaction, especially with basal and med-ventricular wall involvement (p=0.028).

Myocardial fibrosis on LGE-CMR imaging was present in 13 patients (54.16%); the median number of left ventricle segments with LGE per patient was 5.5 (3-9.7). We found no correlation between the presence of myocardial fibrosis and systolic dys-function. Fibrosis did not correlate with the emergence of ventricular tachycardia (Table 2).

A comparison between two groups (arrhythmia vs. heart failure) regarding delay to diagnosis of IVNC and electrocardiographic and cardiac magnetic resonance imaging findings is presented in Table 3.

Discussion

Our study is one of the first series assessing ECG characteristics in patients with IVNC and correlating them with clinical and magnetic imaging features. Actually, CMR is the technique of choice for the diagnosis of IVNC. Because of its superior spatial resolution, this imaging technique can provide an accurate evaluation of myocardial wall involvement and detect myocardial fibrosis (9-12).

The main findings from this study are:

1. ECG abnormalities are frequent in isolated ventricular noncompaction.

2. The most frequent anomaly is LBBB, which was associated with lateral wall involvement.

Table 2. Subgroup analysis according to cardiac magnetic resonance imaging patterns

	Left bundle branch block Median (IQR) n (%)			Ventricular tachycardias Median (IQR) n (%)			Supraventricular arrhythmias Median (IQR) n (%)			Repolarization abnormalities Median (IQR) n (%)		
	Present n=10	**Absent n=14**	**P**	**Present n=5**	**Absent n=19**	**P**	**Present n=7**	**Absent n=17**	**P**	**Present n=7**	**Absent n=17**	**P**
Number of noncompacted segments	11 (7.7-12.2)	7.7 (5.5-10.2)	0.08	8 (7.5-10.5)	10 (7-12)	0.8	8 (6-10)	10 (7-12)	0.1	7 (4-11)	10 (7.5-12)	0.09
Fibrosis	5 (50)	5 (35.7)	0.4	2 (40)	8 (42.1)	0.9	2 (28.6)	8 (47.1)	0.6	4 (57.1)	6 (35.3)	0.39
Left ventricular ejection fraction (%)	29 (24-35)	47 (35-50)	0.05	50 (26-45)	35 (25-48)	0.25	40 (20-60)	38 (27-49)	0.8	40 (23-52)	38 (26-49)	0.6
IQR - inter-quartile range												

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Table 3. Comparison between two groups (arrhythmia vs. heart failure) concerning clinical, electrocardiographic, and cardiac magnetic resonance imaging findings

	Heart failure (n=10)	Arrhythmia (n=11)	*P*
Age, years (mean±SD)	43.6±16.4	43.6±9.3	0.6
Delay to diagnosis of IVNC, months, median (IQR)	1.5 (0-12)	12 (8-36)	0.02
LBBB, n (%)	8 (80%)	10 (90%)	0.56
Ejection fraction (mean±SD)	27.4±8.5	45.9±13.4	0.008
Presence of fibrosis, n (%)	4 (40%)	3 (27.3%)	0.8

IQR - inter-quartile range; IVNC - isolated ventricular noncompaction; LBBB - left bundle branch block; SD - standard deviation

3. No correlation was found between ventricular tachyarrhythmia and the presence of fibrosis or ventricular dysfunction.

4. No correlation was found between the number of noncompacted segments and ventricular dysfunction.

Patients with isolated ventricular noncompaction are relatively young without other comorbidities. In our study, most patients were symptomatic at presentation (91.6%); a similar finding was reported by Habib et al. (4) (83%). The most common reason for referral was arrhythmia and heart failure; the reported incidence is, respectively, 48.1% (7) and 33%-62% (3, 6). However, we noted a variable diagnosis delay, leading to difficulty in the diagnosis of the IVNC, which is frequently underdiagnosed or misdiagnosed. Arrhythmias are frequently observed. In the literature, 75.4%-94% of IVNC patients have an abnormal electrocardiogram (3, 6, 7).

LBBB was the most common electrocardiographic finding in this study (41.7%), as in other reports (35%-44%) (3, 8). This can result from progressive endocardial fibrosis and is often observed in adult patients, because fibrotic changes develop gradually (15). In our study, it seemed to be related to lateral left ventricular wall involvement, independent of ejection fraction or presence of LGE. Due to its physical proximity within the cardiac wall and early embryonic function, the Purkinje fiber network is directly impacted by altered wall maturation observed in noncompaction and trabecular diseases, and this can explain the association (16). Repolarization abnormalities are frequently reported. In Steffel et al. (6), they were the most common ECG abnormality (72%). They occurred secondary to LBBB and ventricular hypertrophy but were also isolated. In our series, 29.1% had an isolated repolarization abnormality; we unlikely observed no correlation between segmental involvement and localization of repolarization abnormalities.

Life-threatening ventricular tachyarrhythmia, including both sustained and nonsustained ventricular tachycardia (VT), occurred in 20.8% of our patients. The reported incidences are variable, ranging from 0% to 41%. Cardiac sudden death in association with IVNC was noted in 7%-35% (3, 15). It has been suggested that ventricular tachyarrhythmias were likely due to the noncompacted myocardium serving as the arrhythmic substrate. Myocardial ischemia and presence of scarred tissue may

play an important role. Ventricular tachycardias represent a therapeutic challenge, as no guidelines regarding their management in IVNC exist. Implantation of a cardiac defibrillator can be performed for primary and secondary prevention in these patients (17). Steffel et al. (18) report that sustained monomorphic VT was rarely induced, even with isoproterenol infusion, and no specific electrocardiographic or echocardiographic finding was predictive of VT inducibility. Zakhary et al. (19) found that left ventricular noncompaction was associated with a higher incidence of ventricular arrhythmias, even in patients with preserved ejection fraction. Similarly, our patients with VT had a normal ejection fraction without fibrosis. Their coronary angiographies were also normal.

Mutations in the human cardiac sodium channel alpha-subunit gene (SCN5A), a well-known gene involved in multiple cardiac arrhythmias, were highly associated with arrhythmias in patients with left ventricular noncompaction than in those without them (50% vs. 7%: p=0.0003). The most frequent arrhythmias were VT and premature ventricular beats (20). This report suggests that the mechanism underlying VT in IVNC could be a gene mutation and may explain the lack of correlation between VT and ventricular dysfunction or fibrosis. Accordingly, we need more studies to clarify risk factors for VT in IVNC.

Patients with IVNC may develop supraventricular arrhythmias (4%-29%) (6, 8). Supraventricular tachycardia revealed IVNC in 12.5% patients in the present study. In a large series (21), they identified 9 patients with atrial fibrillation among 238 patients affected by noncompaction. No case of supraventricular tachycardia was noted. The authors concluded that the atria are not involved in the noncompaction process when the majority of patients has dilated cardiomyopathy. In our study, supraventricular arrhythmias are not a consequence of atrial dilatation or systolic dysfunction but may be due to cardiac involvement in the context of ventricular noncompaction.

Only one patient in our series presented with WPW syndrome. It is more frequently reported in children (12%-15%) than in adults (0%-2.7%) (3, 6-8, 15, 22, 23). Failed regression of developmental embryologic atrioventricular anatomical and electrical continuity during embryonic development in the noncompacted myocardium can explain this association. In our patient, the accessory pathway was type B. This finding is consistent with other reports, because defects in the annulus fibrosis lead to the formation of accessory pathways on the right side of the heart around the tricuspid valve (24).

Previous studies showed that noncompacted segments are mainly located at the apex of the left ventricle, the mid-lateral wall, and the mid-anterior wall and are rarely observed in the basal and septal segments (3, 4, 25, 26). Our results are in agreement with these publications. However, there was discordance between reports when correlating the number of involved segments in IVNC and left ventricular ejection fraction (27-31). The discrepancies between reports can be explained by differences in the characteristics of the recruited patient populations and in the imaging modalities applied for the definition of disease. In

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this study, we found no correlation between the number of non-compacted segments and ventricular dysfunction. Normal left ventricular systolic function was frequently observed in patients who first presented with rhythm disorders. This can probably explain the delay in the diagnosis of IVNC when presenting with arrhythmia versus heart failure.

Study limitations

This study was limited by the retrospective nature of the analysis. The relatively small study population can explain the lack of significance of some results. There is a selection bias like patient with LBBB maybe sending espically to Echocardiographic investigation in this population, as patients were mainly symptomatic and referred to a tertiary care center. Furthermore, we could not perform an electrophysiological study for all patients.

Conclusion

IVNC is a highly arrhythmogenic substrate in our MRI investigation that needs large studies with long-term follow-up for stratification of arrhythmic risk in these populations and comprehension of this mysterious disease.

Conflict of interest: None declared.

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References

1. Maron BJ, Towbin JA, Thiene G, Antzelevitch C, Corrado D, Arnett D, et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association scientific statement from the council on clinical cardiology, heart failure and transplantation committee; quality of care and outcomes research and functional genomics and translational biology interdisciplinary working groups and council on epidemiology and prevention. Circulation 2006; 113: 1807-16. **[CrossRef]**
2. Weiford BC, Subbarao VD, Mulherm KM. Noncompaction of the ventricular myocardium. Circulation 2004; 109: 2965-71. **[CrossRef]**
3. Oechslin EN, Attenhofer Jost CH, Rojas JR, Kaufmann PA, Jenni R. Long-term follow-up of 34 adults with isolated left ventricular non compaction: a distinct cardiomyopathy with poor prognosis. J Am Coll Cardiol 2000; 36: 493-500. **[CrossRef]**
4. Habib G, Charron P, Eicher JC, Giorgi R, Donal E, Laperche T, et al. Isolated left ventricular non-compaction in adults: clinical and echocardiographic features in 105 patients; Results from a French Registry. Eur J Heart Fail 2011; 13: 177-85. **[CrossRef]**
5. Ali SK. Unique features of non-compaction of the ventricular myocardium in Arab and African patients. Cardiovasc J Afr 2008; 19: 241-5.
6. Steffel J, Kobza R, Oechslin E, Jenni R, Duru F. Electrocardiographic characteristics at initial diagnosis in patients with isolated left ventricular noncompaction. Am J Cardiol 2009; 104: 984-9. **[CrossRef]**
7. Shoji M, Yamashita T, Uejima T, Asada K, Semba H, Otsuka T, et al. Electrocardiography characteristics of isolated non-compaction of ventricular myocardium in Japanese adult patients. Circ J 2010; 74: 1431-5. **[CrossRef]**
8. 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. **[CrossRef]**
9. Thuny F, Jacquier A, Jop B, Giorgi R, Gaubertb JY, Bartoli JM, et al. Assessment of left ventricular non compaction in adults: side-by-side comparison of cardiac magnetic resonance imaging with echocardiography. Arch Cardiovasc Dis 2010; 103: 150-9. **[CrossRef]**
10. Alhabshan F, Smallhorn JF, Golding F, Musewe N, Freedom RM, Yoo SJ. Extent of myocardial non compaction: comparison between MRI and echocardiographic evaluation. Pediatr Radiol 2005; 35: 1147-51. **[CrossRef]**
11. Duncan RF, Brown MA, Worthlet SG. Increasing identification of isolated left ventricular non-compaction with cardiovascular magnetic resonance: a mini case series highlighting variable clinical presentation. Heart Lung Circ 2008; 17: 9-13. **[CrossRef]**
12. Dursun M, Agayev A, Nişli K, Ertuğrul T, Onur I, Oflaz H, et al. MR imaging features of ventricular noncompaction: emphasis on distribution and pattern of fibrosis. Eur J Radiol 2010; 74: 147-51. **[CrossRef]**
13. Petersen SE, Selvanayagam JB, Weismann F, Robson MD, Francis JM, Anderson RH, et al. Left ventricular non-compaction: insights from cardiovascular magnetic resonance imaging. J Am Coll Cardiol 2005; 46: 101-5. **[CrossRef]**
14. Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Circulation 2002; 105: 539-42. **[CrossRef]**
15. Ichida F, Hamamichi Y, Miyawaki T, Ono Y, Kamiya T, Akagi T, et al. Clinical features of isolated noncompaction of the ventricular myocardium: long-term clinical course, hemodynamic properties, and genetic background. J Am Coll Cardiol 1999; 34: 233-40. **[CrossRef]**
16. Samsa LA, Yang B, Liu J. Embryonic cardiac chamber maturation: Trabeculation, conduction, and cardiomyocyte proliferation. Am J Med Genet C Semin Med Genet 2013; 163: 157-68. **[CrossRef]**
17. Kobza R, Jenni R, Erne P, Oechslin E, Duru F. Implantable cardioverter-defibrillators in patients with left ventricular noncompaction. Pacing Clin Electrophysiol 2008; 31: 461-7. **[CrossRef]**
18. Steffel J, Kobza R, Namdar M, Wolber T, Brunckhorst C, Luscher TF, et al. Electrophysiological findings in patients with isolated left ventricular non-compaction. Europace 2009; 11: 1193-200. **[CrossRef]**
19. Zakhary D, Louka B, King C, Hafiz AM, Kahan J, Fazzari M, et al. Left ventricular non-compaction assessed by cardiac magnetic resonance imaging is associated with increased ventricular arrhythmias. J Am Coll Cardiol 2014; 63: 282-5. **[CrossRef]**
20. Shan L, Makita N, Xing Y, Watanabe S, Futatani T, Ye F, et al. SCN5A variants in Japanese patients with left ventricular noncompaction and arrhythmia. Mol Genet Metab 2008; 93: 468-74. **[CrossRef]**
21. Fazio G, Corrado G, Pizzuto C, Zachara E, Rapezzi C, Sulafa AK, et al. Supraventricular arrhythmias in noncompaction of left ventricle: Is this a frequent complication? Int J Cardiol 2008; 127: 255-6. **[CrossRef]**
22. Chin TK, Perloff JK, Williams RG, Jue K, Mohrmann R. Isolated noncompaction of left ventricular myocardium: A study of eight cases. Circulation 1990; 82: 507-13. **[CrossRef]**

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23. Pignatelli RH, McMahon CJ, Dreyer WJ, Denfield SW, Price J, Belmont JW, et al. Clinical characterization of left ventricular non compaction in children: a relatively common form of cardiomyopathy. Circulation 2003; 108: 2672-8. [CrossRef]

24. Nihei K, Shinomiya N, Kabayama H, Ikeda C, Hosono T, Aoki T, et al. Wolff-Parkinson-White syndrome in isolated noncompaction of the ventricular myocardium. Circ J 2004; 68: 82-4. [CrossRef]

25. Stöllberger C, Finsterer J. Septal hypertrabeculation/noncompaction: cardiac and neurologic implications. Int J Cardiol 2009; 132: 173-5. [CrossRef]

26. Jenni R, Oechslin E, Schneider J, Attenhofer Jost C, Kaufmann PA. Echocardiographic and pathoanatomical characteristics of isolated left ventricular non-compaction: a step towards classification as a distinct cardiomyopathy. Heart 2001; 86: 666-71. [CrossRef]

27. Dellegrottaglie S, Pedrotti P, Roghi A, Pedretti S, Chiariello M, Perrone-Filardi P. Regional and global ventricular systolic function in isolated ventricular non-compaction pathophysiological insights from magnetic resonance imaging. Int J Cardiol 2012; 158: 394-9. [CrossRef]

28. Fazio G, Corrado G, Novo G, Zachara E, Rapezzi C, Sulafa AK, et al. Ventricular dysfunction and number of non compacted segments in non compaction: non-independent predictors. Int J Cardiol 2010; 141: 250-3. [CrossRef]

29. Aras D, Tüfekçioğlu O, Ergün K, Özeke O, Yıldız A, Topaloğlu S, et al. Clinical features of isolated ventricular noncompaction in adults long-term clinical course, echocardiographic properties and predictors of left ventricular failure. J Card Fail 2006; 12: 726-33. [CrossRef]

30. Yousef ZR, Foley PW, Khadjooi K, Chalil S, Sandman H, Mohammed NU, et al. Left ventricular non-compaction: clinical features and cardiovascular magnetic resonance imaging. BMC Cardiovasc Disord 2009; 9: 37. [CrossRef]

31. Lofiego C, Biagini E, Ferlito M, Pasquale F, Rocchi G, Perugini E, et al. Paradoxical contributions of non- compacted and compacted segments to global left ventricular dysfunction in isolated left ventricular noncompaction. Am J Cardiol 2006; 97: 738-41. [CrossRef]