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Некомпактный миокард и аневризма левого желудочка у 6-летнего ребёнка

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АННОТАЦИЯ

Некомпактный миокард — редкий тип кардиомиопатии, часто сопровождающийся аневризмой желудочка. В статье описан клинический случай 6-летней девочки, поступившей в нашу клинику с жалобами на плохое самочувствие при физической нагрузке. Эхокардиография выявила выраженную трабекулярность стенки левого желудочка и выбухание в области базально-боковой стенки, что соотвествует аневризме и некомпактности миокарда левого желудочка. На магнитно-резонансной томографии сердца наличие некомпактности миокарда было подтверждено соотношением некомпактного слоя к компактному 2,6:1. Кроме того, была выявлена систолическая дисфункция и аневризма левого желудочка с рубцеванием миокарда. Коронарная ангиография исключила поражение коронарных артерий, что позволило предположить природу рубцевания эндомиокарда как результат нарушения микроциркуляции в некомпактном слое миокарда.

Ключевые слова: кардиомиопатия; клинический случай; некомпактность миокарда левого желудочка; аневризма левого желудочка; рубцевание.

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Left ventricular noncompaction with ventricular aneurysm in a 6-year-old patient

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ABSTRACT

Ventricular noncompaction is a rare type of cardiomyopathy often associated with a ventricular aneurysm. We present a clinical case of a 6-year-old female who arrived at our clinic complaining of physical exertion. Echocardiography revealed prominent trabeculations in the left ventricular wall and a lateral-basal part bulging out, indicating noncompaction of the left ventricular myocardium with an aneurysm. With a noncompacted-to-compacted myocardium ratio of 2.6, magnetic resonance imaging revealed the presence of noncompacted myocardium. It also revealed impaired left ventricular systolic function and a left ventricular aneurysm with myocardial scarring. Coronary angiography ruled out coronary artery disease. Therefore, myocardial scarring was caused by noncompacted myocardium microcirculatory disorder.

Keywords: cardiomyopathy; case report; left ventricle noncompaction; left ventricular aneurysm; scarring.

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一名6岁儿童的左室心肌致密化不全和左心室动脉瘤

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简评

左室心肌致密化不全是一种罕见的心肌病。它通常伴有心室动脉瘤。本文描述了一个6岁女孩的临床病例。这名女孩因主诉在体育活动时身体不适而被送进本诊所。超声心动图检查显示了左心室壁有明显的小梁，侧壁基底部有隆起。这些症状与左心室动脉瘤和左室心肌致密化不全相符。心脏磁共振成像显示了，非致密层与致密层的比例为2.6:1，证实了存在左室心肌致密化不全。此外，还发现了收缩功能障碍和伴有心肌瘢痕形成的左心室动脉瘤。冠状动脉造影排除了冠状动脉病变。在这种情况下，我们可以认为心内膜瘢痕形成的性质是非致密心肌层微循环障碍的结果。

关键词：心肌病；临床病例；左室心肌致密化不全；左心室动脉瘤；瘢痕形成。

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ABBREVIATIONS

Ao: aorta
CMR: cardiac magnetic resonance imaging
EF: ejection fraction
HF: heart failure
LA: left atrium
LGE: late gadolinium enhancement
LV: left ventricle
LVNC: left ventricular noncompaction
NC: noncompact myocardium
NC-to-C ratio: noncompact-to-compact ratio
RA: right atrium
RV: right ventricle

INTRODUCTION

Left ventricular noncompaction (LVNC) is a rare type of cardiomyopathy caused by myocardial compaction failure during embryogenesis. It has multiple prominent trabeculations with deep intertrabecular recesses [1]. Its clinical manifestations vary from no symptoms to cardiac dysfunction, heart failure (HF), arrhythmias, and systemic thromboembolism [2]. However, there are only a few reports of LVNC being aggravated by LV aneurysm [3], particularly in children, which is uncommon. Clinically, most LV aneurysms are asymptomatic, although they may cause arrhythmias (18.4%), embolic events (5.4%), rupture (4%), congestive HF (21.5%), and angina pectoris in rare cases [4]. In recent years, two major ideas on the development of LVNC have emerged: the embryogenesis hypothesis and the molecular genetic mechanism. Initially, it was believed that the inadequate consolidation of cardiomyocytes during abnormal embryonic morphogenesis led to the formation of prominent myocardial trabeculae and spaces between them. However, recent improvements in molecular genetic research techniques have revealed a growing number of genes associated with LVNC. Most identified genes are sarcomere protein genes, ion channel genes, and mitochondrial genes, with sarcomere protein genes most often implicated in pathogenesis [5]. In adults, the prevalence of isolated LVNC varies from 0.01% to 0.3% [6]. LVNC is classified as primary genetic cardiomyopathy by the American Heart Association. Conversely, the European Society of Cardiology classifies it under "unclassified cardiomyopathies" [7].

CASE REPORT

A 6-year-old female was admitted to our clinic with complaints of physical exertion. At 26 weeks of gestation, she had exudative pericarditis due to anamnesis. Echocardiography at 15 months revealed pericardial fluid up to 600 mL, prompting pericardiocentesis when the patient was diagnosed with hemopericarditis. Before admission, the patient was diagnosed with HF with a slightly decreased ejection fraction of LV (53). Body temperature was 36.6 C at the time of admission. SpO_2 is 98%. The breath rate is 23 per minute. There is no organic noise in the heart tones, which are rhythmic and muffled. Blood pressure is 115/83 mmHg. The heart rate is 110 per minute.

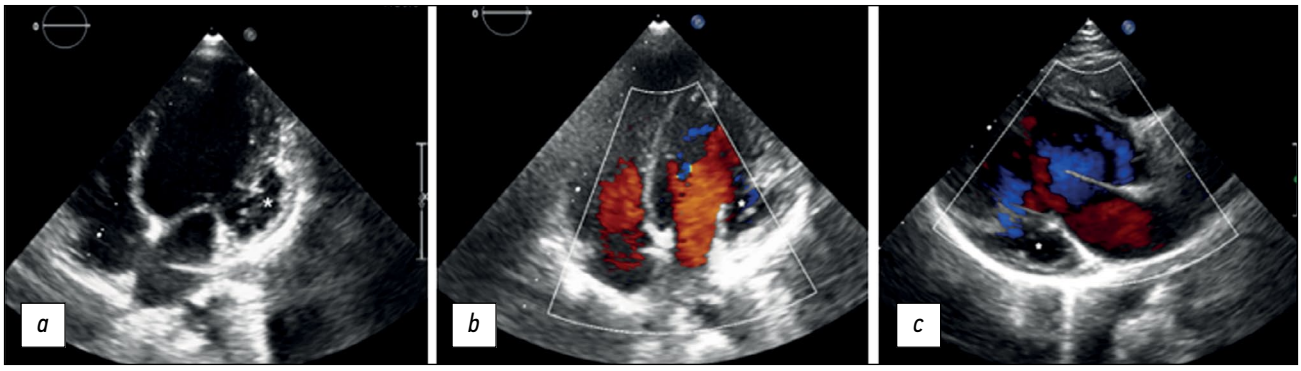

Fig. 1. (*a*) A 6-year-old female patient was diagnosed with left ventricular noncompaction with an aneurysm. An apical four-chamber view demonstrates outpouching of the left ventricular wall with noncompact myocardium. The ventricle is connected to the aneurysm (asterisk). Transthoracic echocardiography (cardiac transducer, two-dimensional scanning), greyscale. (*b*) A 6-year-old female patient with left ventricular noncompaction and an aneurysm. An apical four-chamber view shows a large anechoic lesion (aneurysm denoted by an asterisk) with prominent trabeculation of the apical and lateral walls of LV. Transthoracic echocardiography (cardiac transducer, two-dimensional scanning), color Doppler images. (*c*) A 6-year-old female patient was diagnosed with left ventricular noncompaction with an aneurysm. A three-chamber view shows an aneurysm (asterisk), inlet and outlet of LV. Transthoracic echocardiography (cardiac transducer, two-dimensional scanning), color Doppler images.

During hospitalization, echocardiography revealed LV dilatation, increased trabecularity of the apex and lateral wall of the LV, sac-like bulging of the lateral-basal part as aneurysm, and possibly deep trabeculae (Figure 1a–c). The echocardiography revealed a decline in LV systolic function and a 48% LV ejection fraction. There was minimal regurgitation on the tricuspid, mitral, and pulmonary valves.

The main rhythm, according to the echocardiography, was sinus rhythm. The 24-h ECG Holter monitoring showed sinus rhythm, a mean heart ratio of –109 bpm, a minimum heart ratio of –64 bpm, a maximum heart ratio of –173 bpm, no pauses longer than 2 s, and no tachy-bradyarrhythmia.

A cardiac magnetic resonance imaging (CMR) with contrast enhancement (Gadobutrol 2.0 mL) was performed to exclude congenital heart diseases. The systolic function of both ventricles was reduced (LV EF [41%] and RV EF [45%]). The total LV mass of the myocardium was 54.6 g, with noncompacted myocardium accounting for 14.4 g or 26.3% of the total. CMR revealed signs of myocardial scarring and aneurysmatic ballooning of the basal anterior and lateral-basal walls of the left ventricle, noncompact myocardium (with an NC-to-C ratio = 2.6:1), and left ventricular dilatation. Accumulation of contrast media (one, five, and six segments) in delayed scans was observed (Figure 2a–e).

According to EchoCG and CMR, the patient has a noncompact left ventricular myocardium and aneurysm. Given

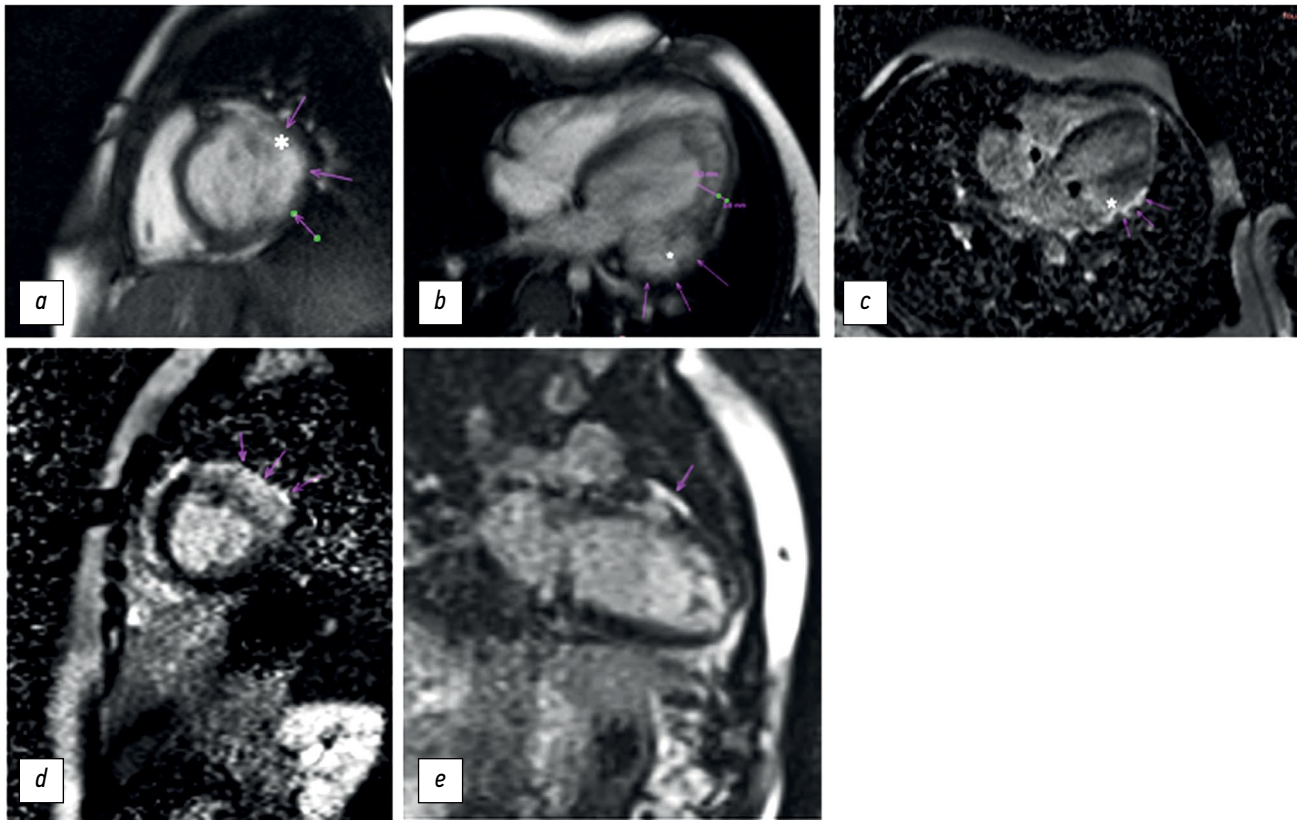

Fig. 2. (*a*) A 6-year-old female patient with left ventricular noncompaction and an aneurysm. Cardiac magnetic resonance imaging (MRI) showed a well-defined outpouching (asterisk) of the left ventricle's lateral wall, as well as local thinning of the myocardium (arrows). Cardiac magnetic resonance, 1.5-T MR System (MAGNETOM Avanto, Siemens Healthcare, Germany) with breath-holding, short-axis view, before the administration of contrast agent, Cine-two-chamber short-axis images, time to echo (TE) of 1.5 ms, repetition time (TR) of 42 ms. (*b*) A 6-year-old female patient with left ventricular noncompaction and an aneurysm. Cardiac magnetic resonance imaging in the four-chamber view demonstrates ventricular aneurysms (asterisk) in the lateral-basal segments of the left ventricle (LV) with noncompaction of the myocardium with a ratio 2.6:1. Cardiac magnetic resonance, 1.5-T MR System (MAGNETOM Avanto, Siemens Healthcare, Germany) with breath-holding, four-chamber long-axis view, before the administration of contrast agent, Cine-four-chamber long-axis images, TE of 1.5 ms, and repetition time (TR) of 42 ms. (*c*) A 6-year-old female patient with left ventricular noncompaction and an aneurysm. LGE, four-chamber view shows late gadolinium enhancement (arrows) on the basal anterolateral wall of the LV, where the aneurysm (asterisk) is located. CMR, 1.5-T MR System (MAGNETOM Avanto, Siemens Healthcare, Germany) with breath-holding, LGE, four-chamber long-axis view, TE of 1.5 ms, and repetition time (TR) of 700 ms. (*d*) A 6-year-old female patient with left ventricular noncompaction and an aneurysm. Cardiac magnetic resonance in the two-chamber short-axis view with LGE demonstrates contrast agent accumulation in the one, five and six segments. CMR, 1.5-T MR System (MAGNETOM Avanto, Siemens Healthcare, Germany) with breath-holding, LGE, two-chamber short-axis view, TE of 1.5 ms, and repetition time (TR) of 2,000 ms. (*e*) LGE, two-chamber long-axis view shows late gadolinium enhancement on the anterior basal wall of the LV. CMR, 1.5-T MR System (MAGNETOM Avanto, Siemens Healthcare, Germany) with breath-holding, LGE, two-chamber long-axis view, TE of 1.4 ms, and repetition time (TR) of 700 ms.

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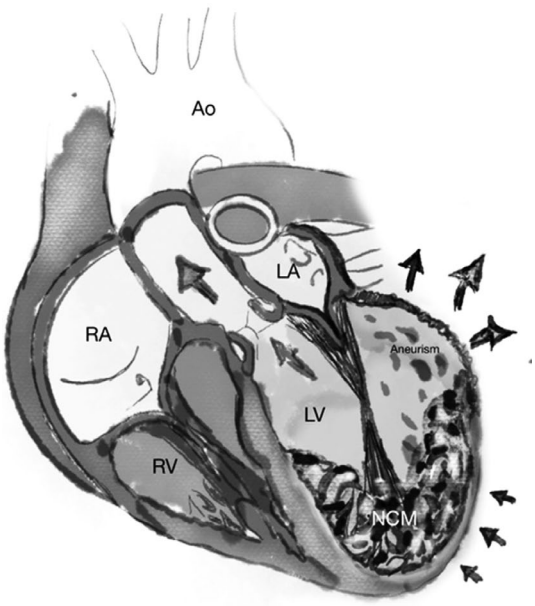

Fig. 3. Noncompaction of the left ventricle with the thin-walled aneurysm with a wide connection to the LV. Aneurysm bulges out from the anterior basal wall during systole. Arrows show the direction of the blood flow. Ao, aorta; LA, left atrium; LV, left ventricle; NCM, noncompact myocardium; RA, right atrium; RV, right ventricle.

the findings, it was decided to have a coronary angiography to assess the coronary vessels and exclude coronary artery anomalies. Coronary angiography showed no evidence of coronary artery abnormalities (Figure 4a and b).

Based on these findings, he was diagnosed with LVNC complicated by an aneurysm.

Given the examination results, weight gain, preserved tolerance to physical activity, and NT-proBNP level of 43.60 pg/mL, the patient was referred to a local hospital for continued monitoring with prescribed medication.

Treatment

Due to limited data on the treatment of this condition, it is recommended that clinical complications be managed in accordance with current guidelines for each specific complication. Anticoagulation therapy is recommended in LVNC patients with a history of thromboembolism, atrial fibrillation, and/or impaired systolic function (LV ejection fraction of <40%) due to an increased risk of thrombus formation resulting from deep intertrabecular recesses and slow blood flow [6]. Therefore, it is suggested that anticoagulation therapy may be necessary in patients with LVNC and accompanying LV aneurysms, even in the absence of systolic dysfunction or atrial fibrillation. However, there have been reports of patients undergoing more radical therapy, including surgical removal, to prevent stress and rupture of the aneurysm wall and the risk of thrombus formation. Furthermore, excising the fibrotic tissue of the LV aneurysm could help prevent arrhythmias, and reshaping the ventricle could improve the patient's HF symptoms. Surgery is considered a definitive treatment that can be used in conjunction with optimal medical therapy [8].

DISCUSSION

The diagnosis of LVNC is mainly based on imaging anatomical characteristics. Although there is no universally established definition of LVNC, the following echocardiographic criteria are widely accepted: (a) two-layered myocardium with multiple, prominent trabeculations in end-systole; (b) NC-to-C ratio of >2; (c) Doppler color flow within the recesses and communication between the intertrabecular space and LV; and (d) absence of coexisting cardiac abnormalities. The classic triad of complications comprises HF, ventricular arrhythmia, and systemic embolic events [9].

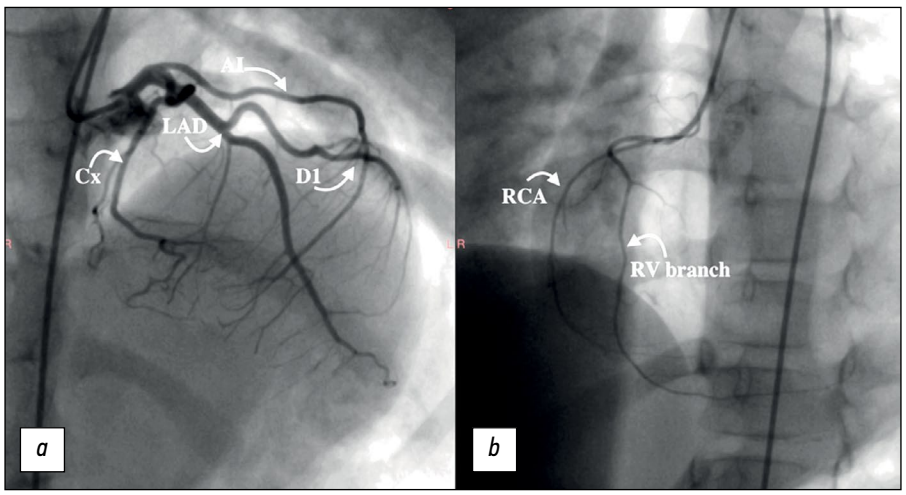

Fig. 4. (*a* and *b*) Coronary angiography of a 6-year-old female patient diagnosed with left ventricular noncompaction and an aneurysm. Coronary arteriography using two catheters revealed no evidence of changes in the coronary arteries. Cx was found in a left dominant pattern of coronary circulation. Coronary angiography using introducer 4Fr and two catheters with 5Fr size. AI, intermediate artery; Cx, circumflex artery; D1, diagonal artery; LAD, left anterior descending; RCA, right coronary artery; RV branch, right ventricular branch.

We encountered a rare case of LVNC with an LV aneurysm formation due to microcirculatory disorder. As demonstrated in our patient, the CMR criterion for diagnosing LVNC is a ratio of maximum thickness between the noncompaction and compaction layer of more than 2.3 in end-diastole, and the mass of trabeculated lv myocardium represents >25% of the total LV mass [10]. Myocardial delayed enhancement revealed scars along the aneurysm wall. Aneurysms have a large connection to the ventricular cavity (Figure 3), whereas diverticula are typically elongated and have a narrowed neck. Aneurysms are most commonly observed in the LV apex (28%) and the perivalvular area close to the mitral valve (49%). LV aneurysms are typically acquired following an acute myocardial infarction with systolic bulging and scar formation in the myocardium. Without knowledge of the patient's medical history and coronary angiogram, acquired aneurysms are difficult to distinguish from congenital left ventricular aneurysms [11]. However, poor microcirculation is believed to be the etiology of aneurysm and scar formation in LVNC [12]. The aneurysm was unrelated to coronary artery territory because our patient's coronary angiography was normal. In our case, an aneurysm appears to be acquired because her previous echocardiography analysis showed EF above 50%; subsequently, when symptoms of cardiac failure began to manifest, it began to diminish. Complications from ventricular aneurysms include intramural thrombus, cardiac output impairment, and aneurysm rupture.

TEACHING POINT

A highly uncommon variant of cardiomyopathy is the combination of ventricular noncompaction and ventricular aneurysm. Echocardiography, CMR, and coronary angiography are valuable techniques for identifying the components of LVNC. Surgery can be combined with medical treatment, regardless of the prevalence of symptoms.

ADDITIONAL INFORMATION

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