CASE PRESENTATION

Left ventricular noncompaction in a highly arrhythmogenic, apparently structurally normal heart

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Summary: **Introduction** – Premature ventricular contractions (PVCs) are relatively common clinical findings. One of the major determinants of prognosis in these patients is the presence of underlying structural heart disease. We report a notable case of highly arrhythmogenic left ventricular noncompaction (LVNC), undetected with the standard techniques used in the workup of patients with frequent PVCs. **Case report** – The ECG performed in a 53 year-old, asymptomatic male, without any personal cardiac history, revealed frequent, polymorphic PVCs, confirmed by ambulatory ECG monitoring. Transthoracic echocardiography failed to show any significant structural abnormalities, and coronary angiography excluded significant coronary artery disease. Complete resolution of PVCs at rest was obtained with Propafenone, but treadmill ECG testing revealed tachycardia-related reoccurrence of PVCs, suggesting the involvement of sympathetic activation in the etiology of ventricular arrhythmias. Cardiac magnetic resonance imaging revealed multiple base-to-apex trabeculae, fulfilling the criteria for LVNC over four segments. **Conclusions** – Electrocardiographic criteria, such as the occurrence of the arrhythmias during exercise and/or the polymorphic appearance of the arrhythmias, may prove useful in selecting candidates for additional imaging workup. It remains to be established if these subtle structural abnormalities, undetected using the standard techniques, carry the same prognosis as those revealed by echocardiography.
Keywords: ventricular tachycardia, premature ventricular beats, myocardial noncompaction, magnetic resonance imaging

Rezumat: **Introducere** – Extrasistolele ventriculare (ESV) sunt frecvent întâlnite în practica clinică curentă. Prezența bolii cardiace structurale subiacente este unul dintre factorii majori de prognostic la acești pacienți. Lucrarea de față prezintă un caz particular de non-compactare ventriculară stângă (NCVS) intens aritmogenă nedetectată prin tehnicile standard utilizate în evaluarea pacienților cu ESV. **Prezentare de caz** – Electrocardiograma realizată la un pacient de sex masculin în vârstă de 53 de ani, fără antecedente cardiace, a evidențiat ESV frecvente, polimorfe, confirmate prin monitorizarea ECG ambulatorie. Ecocardiografia transtoracică nu a evidențiat anomalii structurale semnificative, iar angiografia coronariană a exclus prezența bolii coronariene semnificative. Sub tratament cu Propafenonă s-a obținut rezoluția completă a ESV în repaus, dar testul ECG de efort a evidențiat reapariția ESV odată cu creșterea frecvenței cardiace, sugerând implicarea activării simpatice în etiologia aritmiilor ventriculare. Imagistica prin rezonanță magnetică cardiacă a evidențiat prezența a multiple trabecule, îndeplinind criteriile de NCVS pe patru segmente. **Concluzii** – Criterii ECG precum apariția aritmiilor la efort și/sau caracterul polimorf al ESV se pot dovedi utile în selectarea candidaților pentru investigații imagistice suplimentare. Rămâne de stabilit dacă aceste anomalii structurale "subtile", nedetectate folosind tehnicile standard, asociază același prognostic ca și cele evidențiate ecocardiografic.
Cuvinte cheie: tahicardie ventriculară, extrasistole ventriculare, non-compactare miocardică, rezonanță magnetică nucleară

INTRODUCTION

Premature ventricular contractions (PVCs) and non-sustained ventricular tachycardia (NSVT) are relatively common findings in clinical practice[1]. The presence of these arrhythmias is often revealed during routine ECG, ambulatory ECG monitoring, or exercise stress ECG testing performed for other reasons in asymptomatic patients. The major challenge is to establish whether these ventricular arrhythmias are benign or indicative of an increased risk of sudden cardiac death (SCD). One of the major determinants of prognosis in these patients is the presence or absence of underlying structural heart disease[2]. Standard ECG, exercise ECG testing, transthoracic echocardiography, and coronary angiography are usually used, collectively or in various combinations, to rule out structural heart disease[3].

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However, even in the presence of fully normal standard diagnostic techniques, novel imaging techniques have often identified structural cardiac abnormalities, particularly in patients with right ventricular outflow tract tachycardias[4]. To date, it remains unclear how extensively should these patients be evaluated. Moreover, no consensus exists so far on the definition of structurally normal hearts.

We report a case of high ventricular arrhythmogenicity in the absence of any apparent cardiac structural abnormalities, as assessed using standard diagnostic techniques.

CASE REPORT

A 53-year-old asymptomatic Caucasian male, without any personal cardiac history or ambulatory treatment, presented to a cardiologist for routine physical examination. At that time, all blood tests were within normal ranges and physical examination didn't find any abnormalities. Particularly, there were no signs of anemia, hypoxia, or electrolyte imbalance. The ECG revealed frequent, polymorphic PVCs, including episodes of ventricular bigeminy and couplets. PVCs displayed at least three different morphologies, suggestive of left ventricular outflow tract, left bundle branch fascicles, and inferior septal origins (**Figure 1A**, arrows). Ambulatory ECG monitoring confirmed the presence and the polymorphic feature of PVCs, revealing over 20,000 PVCs / 24-h, as well as the presence of multiple NSVT episodes with left bundle branch block morphology (**Figure 1B**), probably originating in the inferior septal area. Remarkably, QRS morphology during tachycardia was not homogeneous, suggesting multiple exit-points, probably around a restricted area. Transthoracic echocardiogram (**Figure 2**) failed to show any significant structural abnormalities, but the patient had poor acoustic windows. Similarly, coronary angiogram (**Figure 3**) excluded significant coronary artery disease.

The high ventricular arrhythmic burden prompted us to start the patient on Amiodarone. The antiarrhythmic treatment was rapidly efficient, ambulatory ECG monitoring confirming complete resolution of PVCs and NSVT episodes.

Two years later, the patient developed Amiodarone-induced thyrotoxicosis of unclear mechanism. Amiodarone was halted and the patient was started on Prednisone and antithyroid medication, allowing full normalization of the thyroid function within a few months. Shortly after cessation of Amiodarone, ventricular arrhythmias' recurrence was noted. The patient was started on Propafenone 450 mg/day and was scheduled 48-h later for an ECG stress testing. By that time, complete resolution of PVCs was noted at rest. Treadmill stress ECG testing using the standard Bruce protocol revealed the reappearance of PVCs and NSVT at a heart rate of 114 bpm, with rapid resolution when the heart rate decreased bellow 100 bpm (**Figure 4**), suggesting the involvement of sympathetic activation in the etiology of ventricular arrhythmias. This finding prompted us to supplement the patient's treatment with a beta-blocker.

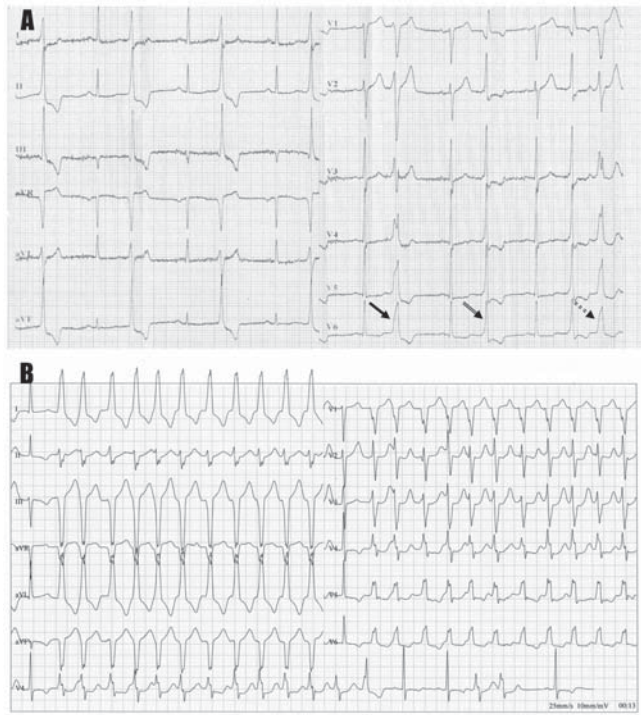

Figure 1. (A) Electrocardiographic recording depicting three different QRS morphologies of premature ventricular contractions: narrow, tall QRS complex in leads II, III, and aVF, and relatively broad R waves in leads V1-V2 indicating a left ventricular outflow tract origin (simple arrow); narrow QRS complex in the precordial leads, with right bundle branch block-like morphology, suggesting a fascicular origin (double arrow); and highly fragmented QRS complex, with left bundle branch block-like morphology (double, round dot arrow). This later morphology resembles that observed during the non-sustained ventricular tachycardia episode recorded using ambulatory ECG monitoring **(B)**, which displays a delayed transition in lead V4, with positive QRS complexes in lead I and equibiphasic QRS complexes in leads II and aVL, indicating an inferior septal origin. Remarkably, QRS morphology during tachycardia is not homogeneous, suggesting multiple exit-points, probably around a restricted area.

The patient was scheduled for cardiac magnetic resonance imaging (MRI), which revealed left ventricular volumes at the upper normal range and increased left ventricular mass index, as well as moderate global hypokinesia, with mild impairment of the left ventricular ejection fraction (LVEF; 58%). Left ventricular anterior and lateral segments displayed multiple ba-

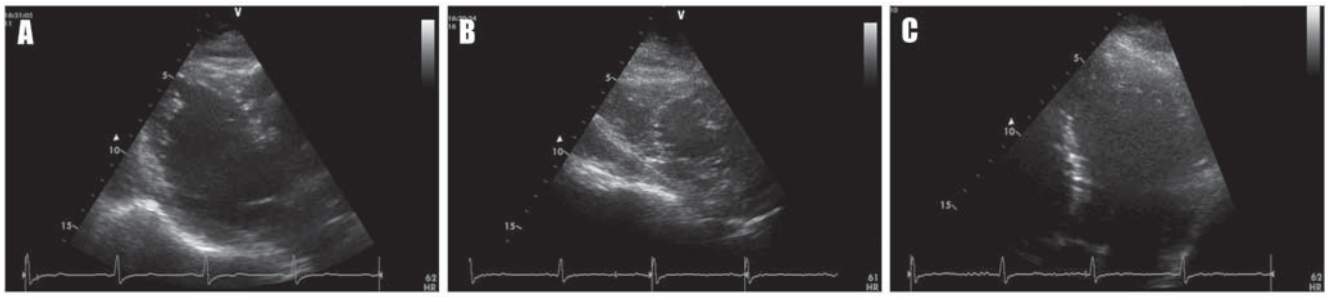

Figure 2. Echocardiographic B-mode images of the left ventricle in **(A)** long-axis view, **(B)** short-axis view, and **(C)** apical view, showing no significant structural abnormalities.

se-to-apex trabeculae, fulfilling the Petersen criteria[5] for left ventricular noncompaction (LVNC) over four segments (**Figure 5A-D**). The right ventricle presented normal volumes and normal systolic function. The free wall of the right ventricle displayed crenelated appearance, but no significant kinetic abnormalities or aneurismal changes were noted. On delayed enhancement imaging, there was late gadolinium enhancement involving the basal region of the infero-lateral segment of the left ventricle, affecting the endocardial layer of the myocardium, limited to an area of noncompaction (**Figure 5E** and **F**, arrows).

Six months later, clinical and echocardiographic follow-up of the patient revealed no progression of the myocardial disease. Resting and stress ECG under combined beta-blocker and Propafenone treatment showed complete resolution of ventricular arrhythmias.

DISCUSSION

Non-sustained ventricular tachycardia and PVCs have been recorded in a wide range of conditions, from apparently healthy individuals to patients with significant heart disease[6]. Although in the vast majority of patients PVCs entail a favorable benign prognosis[7], in patients with structural heart disease the presence of PVCs has been associated with a higher risk of SCD[8,9], particularly in patients with prior myocardial infarction[9].

Accordingly, current management of patients with frequent PVCs is designed to identify an underlying structural substrate of these arrhythmias. Although resting ECG, exercise ECG testing, transthoracic echocardiography, and coronary angiography are commonly used to exclude myocardial disease[7], in some cases, particularly in patients with poor acoustic windows, other techniques may be necessary for definitive ex-

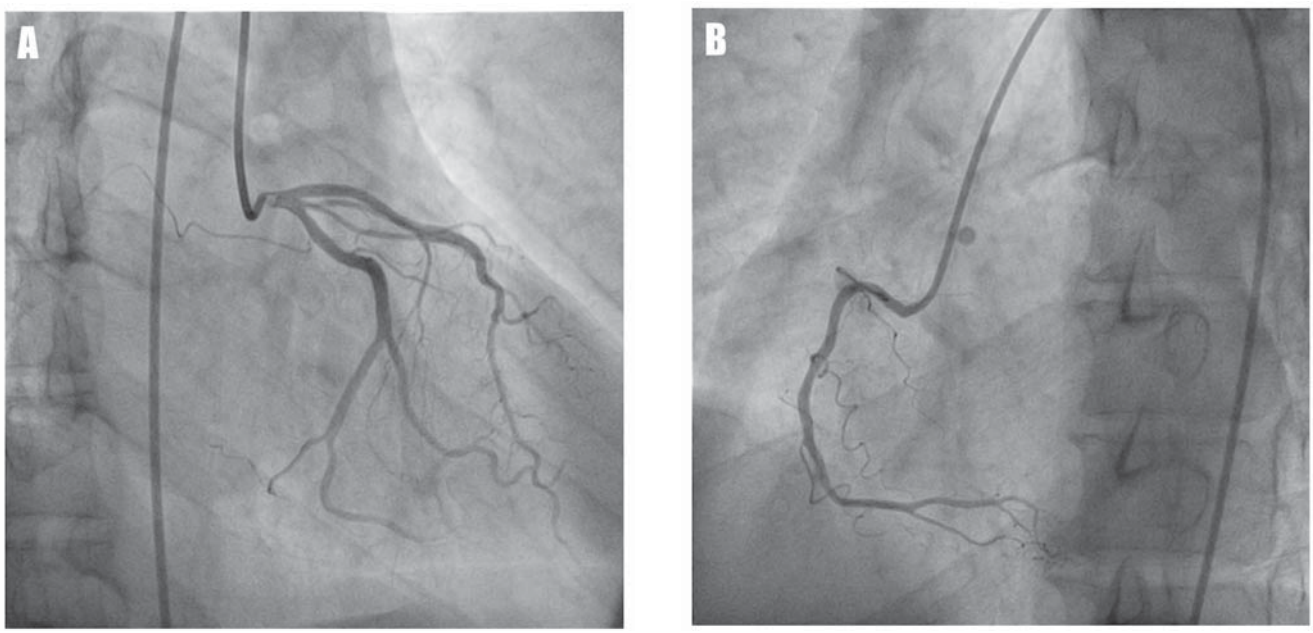

Figure 3. Coronary angiograms of the left coronary artery in right-anterior-oblique caudal view **(A)** and of the right coronary artery in left-anterior-oblique view **(B)** showing the absence of significant coronary artery disease.

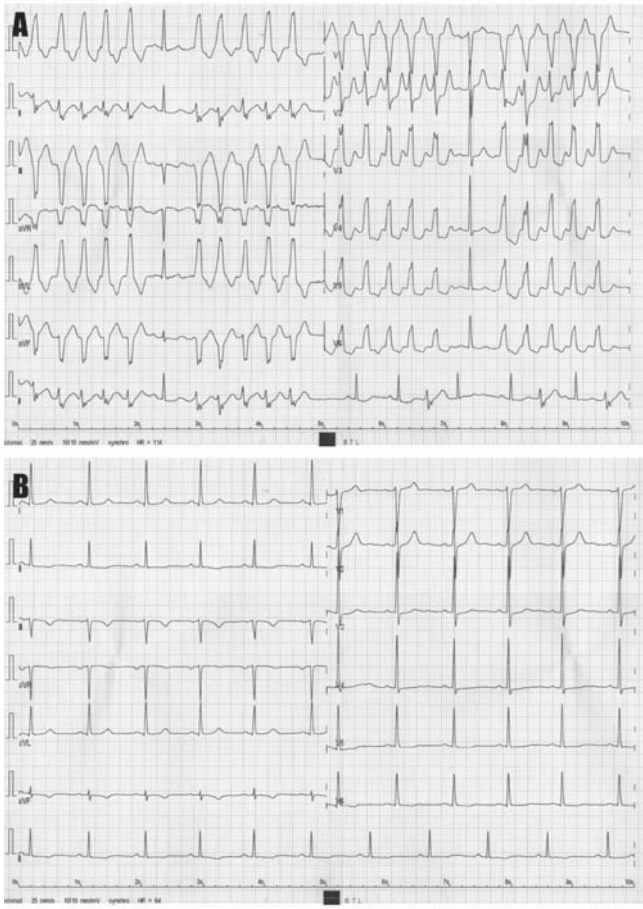

Figure 4. (A) Electrocardiographic tracing recorded during treadmill ECG testing showing the occurrence of non-sustained ventricular tachycardia episodes at a heart rate of 114 bpm. Note that the morphology of ectopic QRS complexes is similar to that of the QRS complexes recorded during the spontaneous non-sustained ventricular tachycardia episode depicted in Fig. 1B. **(B)** Post-effort, at lower heart rates, complete resolution of ventricular ectopic beats is noticed.

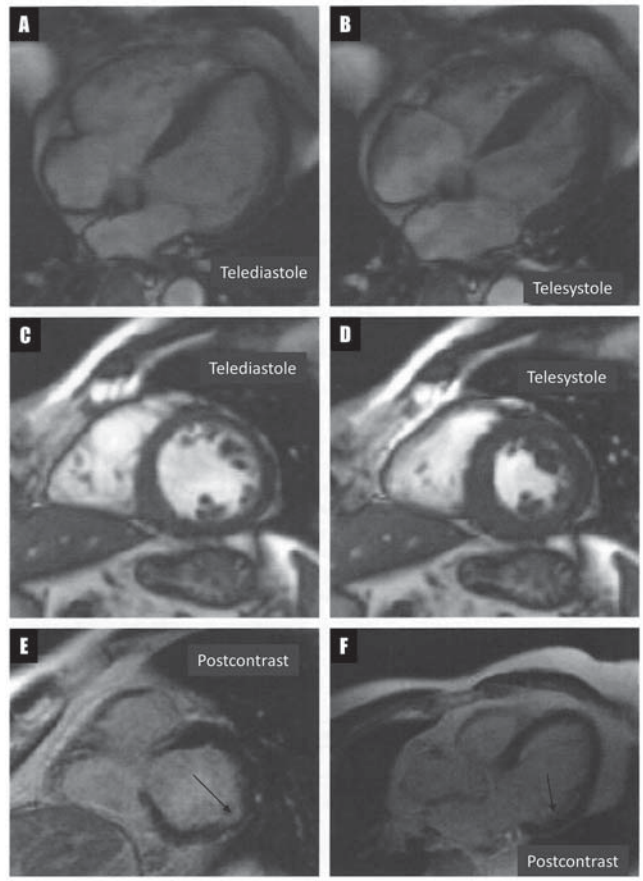

Figure 5. Long-axis (**A** and **B**) and short-axis (**C** and **D**) cardiac magnetic resonance images showing multiple base-to-apex trabeculae involving the left ventricular anterior and lateral segments, and crenelated appearance of the free wall of the right ventricle. Short-axis **(E)** and long-axis **(F)** delayed enhancement cardiac magnetic resonance images showing late gadolinium enhancement involving the basal region of the infero-lateral segment of the left ventricle, affecting the endocardial layer of the myocardium, limited to an area of noncompaction (arrows).

clusion of an underlying disease[2]. However, to date, it remains unclear how extensively should these patients be evaluated. Additional ECG criteria, such as ventricular arrhythmias' relationship with exercise and/or the polymorphic appearance of the arrhythmias, have been proposed to facilitate this decision. Thus, it has been demonstrated that PVCs that mostly occur at rest and suppress with exercise are usually benign, whilst those detected during exercise, and especially at recovery, may be indicative of increased cardiovascular mortality within the next decades[10]. In the same vein, polymorphic PVCs usually arise in the presence of structural heart disease and often entail an increased risk of sustained ventricular arrhythmias and SCD[2].

These exact features, the increase in ventricular ectopy at exercise and the polymorphic feature of ventricular arrhythmias, prompted us to investigate further for the presence of an underlying myocardial disease, using one of the modern, more sensitive imaging techniques. This allowed the diagnosis of LVNC in the absence of any apparent structural abnormalities with echocardiography.

To date, the morphological substrate and the prognostic significance of ventricular arrhythmias in patients with LVNC are far from clear[11]. Perfusion defects in areas of noncompaction may provide a substrate for reentrant arrhythmias, explaining the often polymorphic appearance of the arrhythmias, as well as the occurrence of the arrhythmias at exercise in these patients[12-15]. However, in the present case, the recorded QRS morphologies of PVCs and NSVTs can hardly be related to any area of noncompaction, as indicated by cardiac MRI, suggesting that other factors may also be involved in the high ventricular arrhythmogenicity observed in this population. Indeed, in the study of Shan

et al. the frequency of SCN5A variants, encoding for the alpha-subunit of the voltage-gated sodium channel, was significantly higher in patients with LVNC that presented ventricular arrhythmias than in those who did not, suggesting that genetic factors may also represent a risk factor for arrhythmias in this population[16].

In the present case, combined beta-blocker and Propafenone treatment allowed complete resolution of ventricular arrhythmias. However, suppression of arrhythmic events with antiarrhythmic drugs could not be associated with improved survival in various clinical scenarios[17,18], while in studies such as CAST and CAST II, class Ic antiarrhythmics were actually associated with a significant increase in mortality in post-myocardial infarction patients, despite a significant reduction in arrhythmia burden[19,20]. To date, no specific data are available in LVNC patients. Accordingly, the decision of initiating antiarrhythmic treatment in this patient was driven by the high arrhythmic burden and the consequent risk of tachycardiomyopathy, and not by the rationale that this might reduce the risk of SCD. In patients with structural heart disease, Amiodarone is usually the preferred antiarrhythmic drug. The occurrence of Amiodarone-induced thyrotoxicosis prompted us to stop the treatment and to change the patient to Propafenone. However, Propafenone treatment carries a significant proarrhythmic risk in patients with heart failure, imposing regular follow-up of the patients' left ventricular systolic function[21,22], as well as the need to regularly monitor the duration of the QRS complex.

Galizio et al. recently proposed a number of criteria useful for identifying patients at increased risk of SCD[23]. Based on these criteria, the authors propose cardiac defibrillator implantation for the primary prevention of SCD in patients with LVNC and LVEF <30%, or at least two of the following criteria: family history of SCD, syncope or NSVT. Out of the 80 patients included in the study 28.75% beneficiated of cardiac defibrillator implantation for the primary prevention of SCD. Only one of these patients beneficiated of an appropriate shock during the 28 ± 22 months of follow-up; this patient was implanted based on a low LEVF. These results underline the role of decreased LVEF as main predictor of mortality in these patients. Electrophysiological studies and assessment of malignant ventricular arrhythmias induction may seem attractive for identifying patients at increased risk for SCD. However, arrhythmia induction in 24 LVNC patients could not predict the occurrence of SCD during the 61.4 ± 50 months of follow-up[24]. Accordingly, although the ACC/AHA/HRS 2008 *Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities* recommended cardiac defibrillators implantation in all LVNC patients[25], in the light of the current data, it does not seem reasonable to formulate recommendations applicable for the entire population of LVNC patients. Until additional studies are available and definitive criteria formulated, it seems reasonable to approach these patients based on parameters with established prognostic roles. Accordingly, the strongest predictor of SCD and the most widely used parameter in deciding cardiac defibrillator implantation for the primary prevention of SCD in patients with structural heart disease remains a low LVEF[26].

Although cardiac MRI allowed the identification of ventricular noncompaction as substrate for arrhythmias in this patient, at present, there is little evidence to recommend the routine use of such imaging techniques in the workup of PVCs[27]. Moreover, it remains to be established if subtle structural abnormalities, undetected using the standard techniques, carry the same prognosis as the 'gross' structural abnormalities revealed by echocardiography. Further studies will have to be conducted in order to make definitive recommendations on the need and frequency of using these novel imaging techniques for the diagnosis and follow-up of these patients.

CONCLUSION

This report illustrates a case of highly arrhythmogenic LVNC undetected with the standard techniques used in the workup of patients with frequent PVCs. Additional ECG criteria, such as the occurrence of the arrhythmias during exercise and/or the polymorphic appearance of the arrhythmias, proved useful in deciding to perform further imaging workup. It remains to be established if these subtle structural abnormalities, undetected using the standard techniques, carry the same prognosis as those revealed by echocardiography.

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