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Pragmatic electrocardiogram tracings in non-ischaemic dilated cardiomyopathy: diagnostic and prognostic role

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Dilated cardiomyopathy (DCM) is a primitive heart muscle disease characterized by a great heterogeneous aetiology and prognostic outcome. Dilated cardiomyopathy is an umbrella term encompassing different aetiologies that might require specific treatments. It principally affects young and male adults, with high-risk arrhythmic competitive risk. Unfortunately, the prevention of major ventricular arrhythmic events remains a clinical challenge. In the era of advanced multimodality imaging and widely available genetic testing, electrocardiogram (ECG) continues to represent a reliable diagnostic tool, for specific work up of every single patient. However, approaching DCM patients, only a cardiomyopathy-oriented reading makes the role of ECG central in the management of DCM, both for diagnosis, prognosis, and therapeutic management. In this paper, we present four ECGs of four different DCM patients, in order to guide a cardiomyopathy-oriented ECG reading, emphasizing its impact in an early, cost-effective, and personalized diagnostic and prognostic work up in this specific setting.

Introduction

Dilated cardiomyopathy (DCM) is a heart muscle disease characterized by left ventricular (LV) or biventricular dilation and systolic dysfunction in the absence of either pressure or volume overload or coronary artery disease sufficient to explain the dysfunction.[1] Despite relatively improved natural history of DCM in the last decades, its clinical management is still challenging. Dilated cardiomyopathy is an 'umbrella' term that describes the final common pathway of different pathogenic processes and gene–environment interactions.[2] In fact, in the same definition of DCM, we include a variety of diseases, ranging from tachy-induced cardiomyopathies[3] to genetically determined DCM[4] to chemotherapy-induced cardiomyopathy.[5] With a view to increasingly targeted and effective therapy, it appears crucial to specifically characterize and stratify patients as quickly and accurately as possible.

Nowadays very powerful diagnostic tools are available: it is possible to study myocardial tissue by non-invasive multimodal imaging, and it is also possible to characterize the patient and family members by genetic evaluation. Through cardiac magnetic resonance imaging (CMRI), we can assess the presence and distribution of myocardial fibrosis, as well as the presence of oedema or deposits of intra- or extracellular material.[6] By PET-CT, we can study the level of immune and inflammatory activation of myocardial tissue, and thus the activity status of inflammatory cardiomyopathies, particularly sarcoidosis.[6] These are essential diagnostic tools for proper aetiologic diagnosis, but they are not systematically available, are expensive, and can require long waiting times to be performed. For these reasons, the electrocardiogram is experiencing a real renaissance in the field of cardiomyopathies. The electrocardiogram is a technology as old as it is fascinating,

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intercepting multiple aspects inherent in primitive heart muscle disorders. Through a trivial expense and in a matter of minutes, it is able to provide us with a holistic view of the myocardial electrical state, which is derived from the synthesis of tissue characteristics (e.g. fibrosis), disorders of rhythm and electrical conduction. Thus, the strength of electrocardiography lies in the ability to diagnose and prognostically stratify DCMs in order to accurately address second and third diagnostic investigations and therapeutic strategies. Importantly, in the context of DCM, the ECG should not be read as a routine examination, but should be read carefully with a magnifying glass, analyzing the individual ECG signs by adopting a critical and deep ECG reading. Therefore, we can talk of 'renaissance of electrocardiography' in DCM, only if we highlight the main electrocardiographic signs that should be systematically read and interpreted in a critical ECG reading, namely: *P* waves and PR trait, the presence of Q waves, QRS voltage (high or low), QRS duration (including right and left bundle branch blocks), and repolarization changes (i.e. T wave inversions). In the present review, through four ECG tracings of four different DCM patients, we describe the importance of ECG critical reading in daily clinical practice, in diagnostic and therapeutic management of DCM patients.

Electrocardiogram: the prognostic role of low QRS voltages

The first ECG that we present, was performed in a 21-year-old male, with a diagnosis of biopsy -proven 'post-myocarditis' DCM. He had not any cardiovascular risk factors nor familial history for cardiomyopathies or sudden cardiac death. His cardiologic history began with cardiac arrest, showing a left ventricle ejection fraction (LVEF) of 34% with left ventricle end-diastolic volume (EDV) of 84 mL/m^2.

Analyzing in detail the ECG in the *Figure 1A*, we have: a sinus bradycardia (47 bpm), a *P* wave of normal voltage and duration, a PR tract of 169 ms, the electrical axis of the QRS at +70° in the frontal plane, a small isolated Q wave in aVL, a low QRS voltage on the peripheral leads (total 18 mm summation), a slight fragmentation of the QRS in the terminal portion of V2, a normal duration of the QRS (112 ms) and T wave inversion in the infero-lateral site.

The most striking ECG sign is the low QRS voltages, which prognostic role is crucial in the context of DCM. Low QRS voltages represent severe myocardial destructuring resulting from major myocyte fibrotic replacement, as we can see in *Figure 1B* and *C*.

The patient's follow-up was characterized by prognostically unfavourable progression, with significant worsening of left ventricular systolic function and multiple hospitalizations for heart failure. The patient also had two additional episodes of ventricular fibrillation, which were effectively treated by ICD shock. The patient died in heart transplant waiting list from sudden cardiac death due to pulseless electrical activity after multiple ICD shocks.

Low QRS voltages is not frequent in DCM, as it accounts for only 3–6%.[7] It reflects the loss of vital myocardium and diffuse LV fibrosis, especially in precordial leads.[8] It is important to observe the prognostic role of this ECG sign, as it is more associated with a severe cardiac phenotype, and it might be considered as an useful tool in the evaluation of arrhythmic stratification and as a marker of unlikely reverse remodelling. Of a consequence, low QRS voltages should be taken into account when evaluating advanced cardiac support or cardiac transplantation.[9] It is also very important to follow-up these low QRS voltages, contextualizing them in the clinical picture, since they may be an expression of myocardial oedema in addition to fibrosis in active myocarditis. Indeed, in the latter setting, following resolution of the oedema, the voltages increase again.

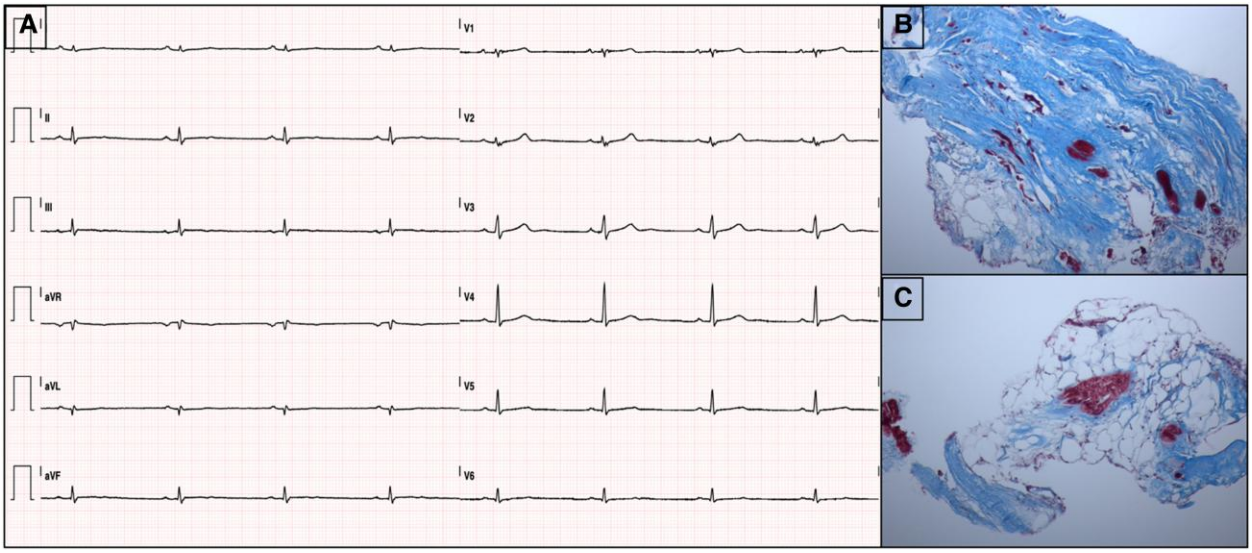

Figure 1 **Electrocardiogram of a post-myocarditis DCM.** In this, figure is presented in the panel A the electrocardiogram of a 21-year-old male, with a severe left ventricle systolic dysfunction, who is further worsening LVEF and ventricular volumes despite optimized medical therapy. There is also a worsening of the arrhythmic burden. Fundamental from the prognostic point of view in this ECG is the enhancement of the low voltages of the QRS in particular in the peripheral derivations, which suggests the loss of muscle tissue due to fibro-adipose replacement as shown in panels B and C. LVEF, left ventricle ejection fraction; ECG, electrocardiogram; DCM, dilated cardiomyopathy.

Noteworthy, prognostic role of QRS fragmentation in the contest of post-myocarditis DCM. Ferrero et al. showed that the persistence of QRS fragmentation at the follow-up is correlated to the persistence of LGE and incomplete contractile recovery, with a sensitivity of 21% and specificity of 100% in predicting LGE persistence.[10] Interestingly, in the same population, the subgroup of patients in which the QRS fragmentation disappeared at follow-up, seemed to be correlated with a better prognosis in term of mechanical function.[10]

Finally, this ECG can be an important 'red flag' to direct the clinician in further diagnostic investigation of patients with myocarditis. In fact, it is increasingly known that myocarditis can coexist with arrhythmogenic cardiomyopathies in the context of 'hot phases'. In this ECG, important coexistent red-flags (low QRS voltages, QRS fragmentation, and negative T waves) may indicate an arrhythmogenic cardiomyopathy (possibly with hot phases) and address to genetic testing in the context of 'post-myocarditis' DCM.

Electrocardiogram: the genetic diagnosis

The ECG presented in *Figure 2A* was performed in a 45-year-old man, with important family history of sudden cardiac death and DCM. He has neither cardiovascular risk factors nor comorbidities. His cardiologic work up starts with a preoperatively ECG finding of frequent premature ventricular beats (more than 24.000/24 h documented at 24 h Holter ECG and a nonsustained ventricular tachycardia of 16 beats at 160 bpm). Echocardiography showed a dilated left ventricle (telediastolic diameter of 66 mm) and a LVEF of 45% (*Figure 2B*). Cardiac magnetic resonance (CMR) imaging, in addition to confirming left ventricular dilatation and mild dysfunction, showed an extensive area of intramyocardial and mid-basal epicardial late gadolinium enhancement (LGE) (*Figure 2C*).

Analyzing in detail the ECG (*Figure 2A*), we find a sinus rhythm with 56 bpm, a normal atrio-ventricular conduction with a PR interval of 192 ms. The ECG pattern was typical of arrhythmogenic DCM (with a prevalent left ventricle involvement): the QRS complex has voltages at the lower limits of the norm. In the inferior leads there is a QRS fragmentation, especially in D3 and aVF. Of note, there is T wave inversion from V3 to V6; T waves are isodifasic in D1, aVL and in inferior leads.

The patient was placed on optimized medical therapy and then an implantable cardioverter defibrillator (ICD) was implanted in primary prevention. Crucial in this regard was genetic analysis, which revealed a likely pathogenic mutation in the Filamin C gene. The patient's follow-up, was characterized by a significant arrhythmic burden, with multiple ATP interventions on ventricular tachycardia (VT) and an episode of syncopal ventricular fibrillation resolved with ICD shock.

Gigli et al.[4] demonstrated that Filamin C truncating variants (FLNCtv) cause a form of arrhythmogenic cardiomyopathy that is phenotypically heterogeneous and characterized by a high risk of life-threatening arrhythmias, which does not seem to be associated with the severity of left ventricular systolic dysfunction. In this cohort of tertiary referral hospital centre, negative T waves and NSVTwere significantly more present in probands than non-probands. The prognostic importance of having the suspect of this genetic mutation correlated with the DCM, relies on the indication of ICD implantation regardless of non-severe systolic disfunction.[11] This ECG is typical of an 'arrhythmogenic cardiomyopathy,' considering the term 'arrhythmogenic' in a wider sense, referring to a pathology with

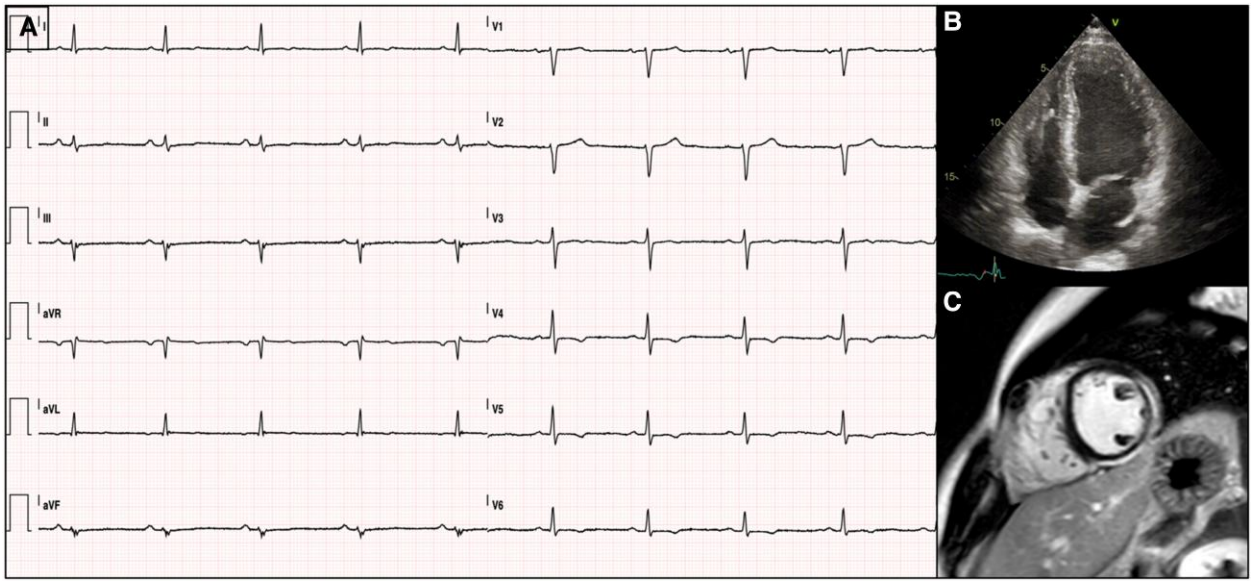

Figure 2 A genetic diagnosis suggested at the electrocardiogram. In this, figure is presented in the panel A the electrocardiogram of a 41-year-old man, with a DCM genetically determined by a likely pathogenic mutation in the Filamin C gene. The main aspect of the electrocardiogram is based on the infero-lateral repolarization abnormalities, with negative Twaves from V3 to V6, isodifasic Twaves in DI and aVL and also in inferior leads. The panel B shows the left ventricle dilatation, with mildly reduced left ventricle systolic function with a LVEF of 45%. The panel C shows the typical subepicardial ringlike distribution of LGE involving the inferior, posterior and lateral wall at the CMR. DCM, dilated cardiomyopathy; LVEF, left ventricle ejection fraction; LGE, late gadolinium enhancement; CMR, cardiac magnetic resonance.

possible left ventricle involvement and not just the classic right ventricle involvement typical of desmosomal genes.

Another important point to note, concerns QRS fragmentation, an electrocardiographic sign that can sometimes be difficult to detect, but in the analysis of Goldberger *et al.*[12] was shown to be one of the main predictors of sudden death [OR: 6.73, 95% confidence interval (CI): 3.85–11.76] even compared with QRS duration (OR: 1.51, 95% CI: 1.13–2.01).

Electrocardiogram: left bundle branch block as a marker of unlikely reverse remodelling

The ECG showed in *Figure 3A*, was performed in a 54-year-old man, with dyslipidaemia and no other cardiovascular risk factors, nor comorbidity and no familial history for sudden cardiac death nor DCM. At age 45 years old, he was hospitalized for acute heart failure, with the evidence of severe dilation, left ventricle systolic and diastolic disfunction (EDV 322 mL; LVEF 19%; E/E' medial ratio of 50) and severe mitral regurgitation with pulmonary arterial pressure estimated of 52 mmHg. The CMR revealed the presence of transmural LGE of the infero-posterior wall and subendocardial LGE of inferior and anterior wall. Non-significant epicardial coronary artery disease. The genetic analyses revealed a variant of uncertain significance (VUS) in the NEXN gene.

Analyzing the ECG in detail, we find sinus rhythm, bi-atrial enlargement, an atrio-ventricular first-degree block (PR interval 201 ms) and a complete LBBB with QRS duration of 201 ms.

During the hospitalization, it has been introduced the guideline directed therapy for heart failure, with difficulty on titration. In consideration to the severe and advanced left ventricle adverse remodelling and the extended of fibrosis in the CMR, he was implanted with cardiac resynchronization therapy defibrillator (CRT-D) in primary prevention, with subsequent reverse remodelling.

LBBB has been considered for a long time a specific marker of DCM, but today we know that it is found in roughly one third of DCM patients, sometimes preceding the structural phenotype, and carries an adverse prognostic value.[8] LBBB is also negatively associated with likelihood of left ventricle reverse remodelling, and the development of new LBBB during the follow-up is a strong independent predictor of all-cause mortality.[13] Of note, CRT should timely be considered after LBBB finding, considering that CRT was shown to reduce adverse outcome, specifically in DCM patients and in those who cannot tolerate the medical therapy. In absence of LGE in CMR and severe left ventricle remodelling and with a negative genetic, LBBB should be the underlying cause of DCM, considering it as a pathology caused by dyssynchrony. Identifying this form of DCM is crucial, as it should normalize left ventricle function and volume with CRT therapy.

Electrocardiogram: the therapeutic role in sarcoidosis

The ECG showed in *Figure 4A*, was performed in a 56-year-old man, with no cardiovascular risk factors, no comorbidities and no familial history for sudden cardiac death nor DCM. The ECG present in the *Figure 4A* was performed because he was symptomatic for asthenia and palpitations.

Analyzing the ECG in detail, we find sinus rhythm, a normal *P* wave and a first-degree atrio-ventricular block (PR interval 255 ms). An isolated ventricular extrasystole with a left bundle branch block like morphology and with a transition in V4-V5. The QRS duration is augmented (162 ms) with a right bundle branch block (RBBB) and a left anterior fascicular block. The repolarizations abnormalities are secondary to the intraventricular

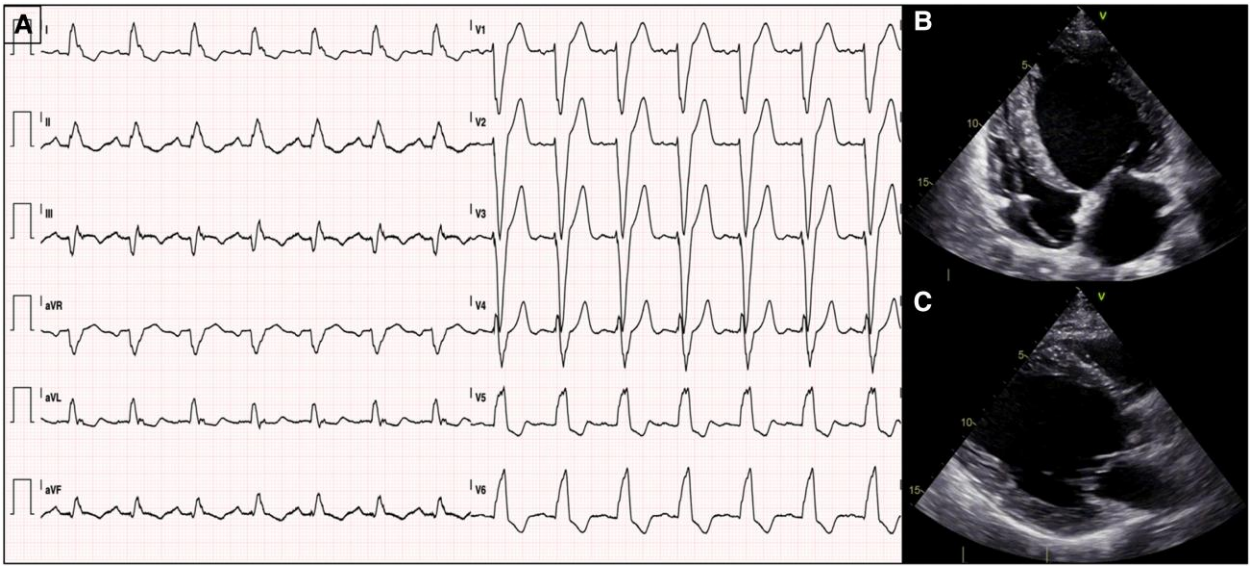

Figure 3 **Left bundle branch block as a marker of unlikely left ventricle reverse remodelling.** In this figure is presented in the panel A the electrocardiogram of a 54-year-old man, with an idiopathic DCM with negative genetic test. The main aspect of the electrocardiogram is based on LBBB, which is a marker of unlikely left ventricle reverse remodelling in the context of DCM, and it also represents a therapeutic target to treat with CRT. The panel B and C show the severe left ventricle dilation, respectively in apical four chamber and parasternal long axis. DCM, dilated cardiomyopathy; LBBB, left bundle branch block; CRT, cardiac resynchronization therapy.

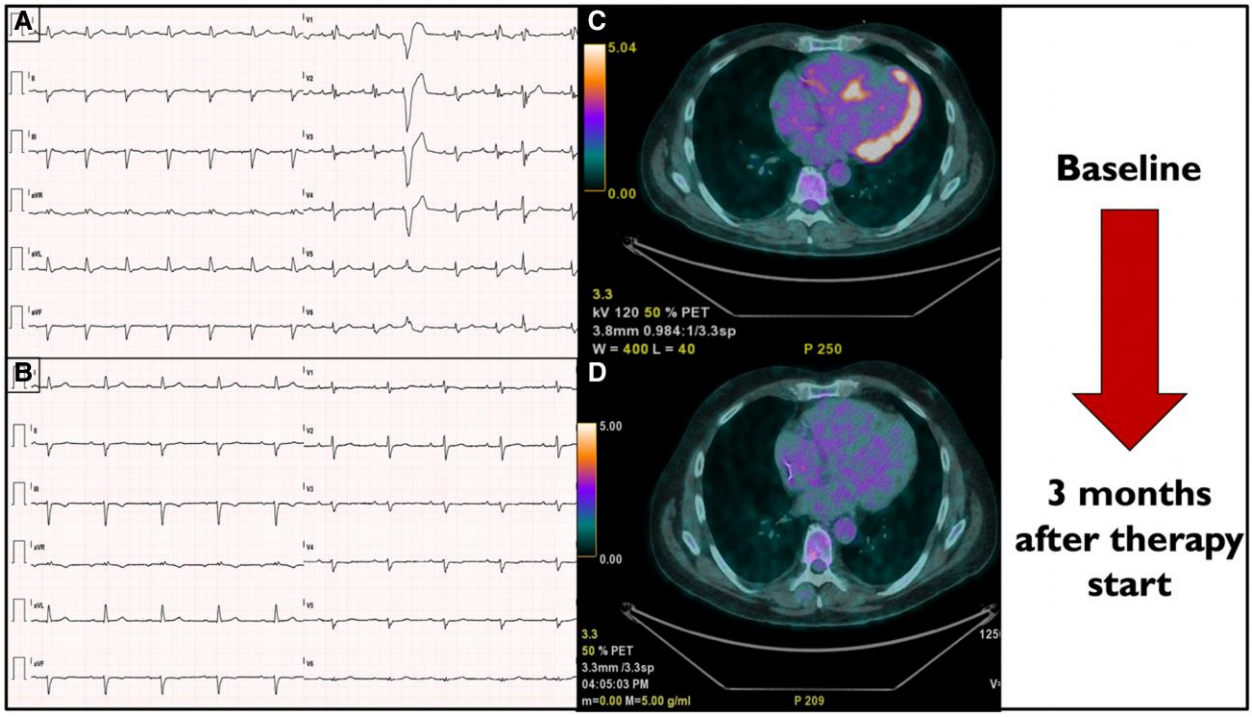

Figure 4 **Electrocardiogram and its therapeutic guidance in sarcoidosis therapy.** In this, figure is presented in the panel A the basal electrocardiogram of a 56-year-old man, affected by cardiac sarcoidosis, showing first-degree of atrio-ventricular block, right bundle branch block and QRS complex fragmentation. In the panel C is also presented the PET-TC scan at baseline, showing the important inflammatory activation in the myocardium. In the panel B is presented the ECG during the follow-up, after XX months, during immunosuppressive therapy, and in the panel D it is also presented the control PET-TC scan at XX months in immunosuppressive therapy. ECG, electrocardiogram; PET-TC, positron emission tomography and computed tomography.

depolarization abnormalities, in right precordial leads and inferior leads. We can detect also a slightly QRS fragmentation in the terminal portion of QRS in V3 and II and aVF.

This is clearly a pathological ECG, so the patient was referred for various cardiological investigations. Echocardiogram demonstrated a mildly dilated left ventricle (EDVi 71 mL/m^2) with moderate left ventricular systolic dysfunction (LVEF 42%) and a mildly dilated and dysfunctional right ventricle. CMR confirmed the biventricular dilatation and dysfunction and showed the presence of extensive, multifocal, non-ischaemic left ventricle LGE, which was also diffuse to the free wall of the right ventricle.

Because the laboratory test were suggestive of inflammatory cardiomyopathy, and specifically sarcoidosis, the patient also had a PET-CT scan, which confirmed the suspicion (*Figures 4B*). To have the diagnosis of certainty, endomyocardial biopsy was also performed, which demonstrated the presence of non-caseous granulomas within the myocardium (*Figure 5B* and *D*), pathognomonic of cardiac sarcoidosis.

The most interesting aspect, besides the diagnostic suspicion originating from the pathological ECG, lies in its role in monitoring the response to immunosuppressive therapy. In fact, as shown in the figure (*Figures 5C*), the ECG has significantly improved, with the absence of the previously present conduction disturbances, a finding further confirmed by the follow-up PET-CT scan, which demonstrates that the inflammation resolved under therapy. Of note, RBBB is a relative rare sign, present in 2–6% of cases,[8] in the context of DCM, and when present, it suggests some specific differential diagnosis among neuromuscular disorders due to pathogenic variants in the dystrophin gene, right ventricle arrhythmogenic cardiomyopathy and cardiac sarcoidosis.

Cardiac involvement in sarcoidosis is relatively rare, but when present, the outcome is unfavourable due to granulomatous infiltration of ventricular myocardium. It remains a diagnostic and therapeutic challenge, with a paucity of data to guide management. The natural history is not well known, and the presentation can vary from asymptomatic cardiac inflammation to high-grade atrio-ventricular (AV) block, ventricular arrhythmia, heart failure, and/or sudden cardiac death. Non-necrotizing granulomas are the histopathological hallmark of sarcoidosis, and therapy is principally based on immunosuppression and the management of cardiac involvement (conduction disorders, ventricular arrhythmia and heart failure).[14] The ECG is fundamental in the management of these patients, in order to identify the cardiac involvement and the severity of arrhythmias and conduction disorders. A common manifestation of cardiac sarcoidosis is the first and high degree of atrio-ventricular block. Also the QRS complex represents an important red flag to emphasize, in particular: QRS complex fragmentation as well as bundle branch block, are associated with cardiac involvement. Another ECG feature deserving a separate note, is the Epsilon wave, which may be present in cardiac sarcoidosis, placing a diagnostic challenge to differentiate form arrhythmogenic right ventricular cardiomyopathy (ARVC).[15] Finally, the critical evaluation of ECG modification during treatment is important to assess the benefit of immunosuppression on cardiac involvement,[16] and this is complementary to PET-TC.

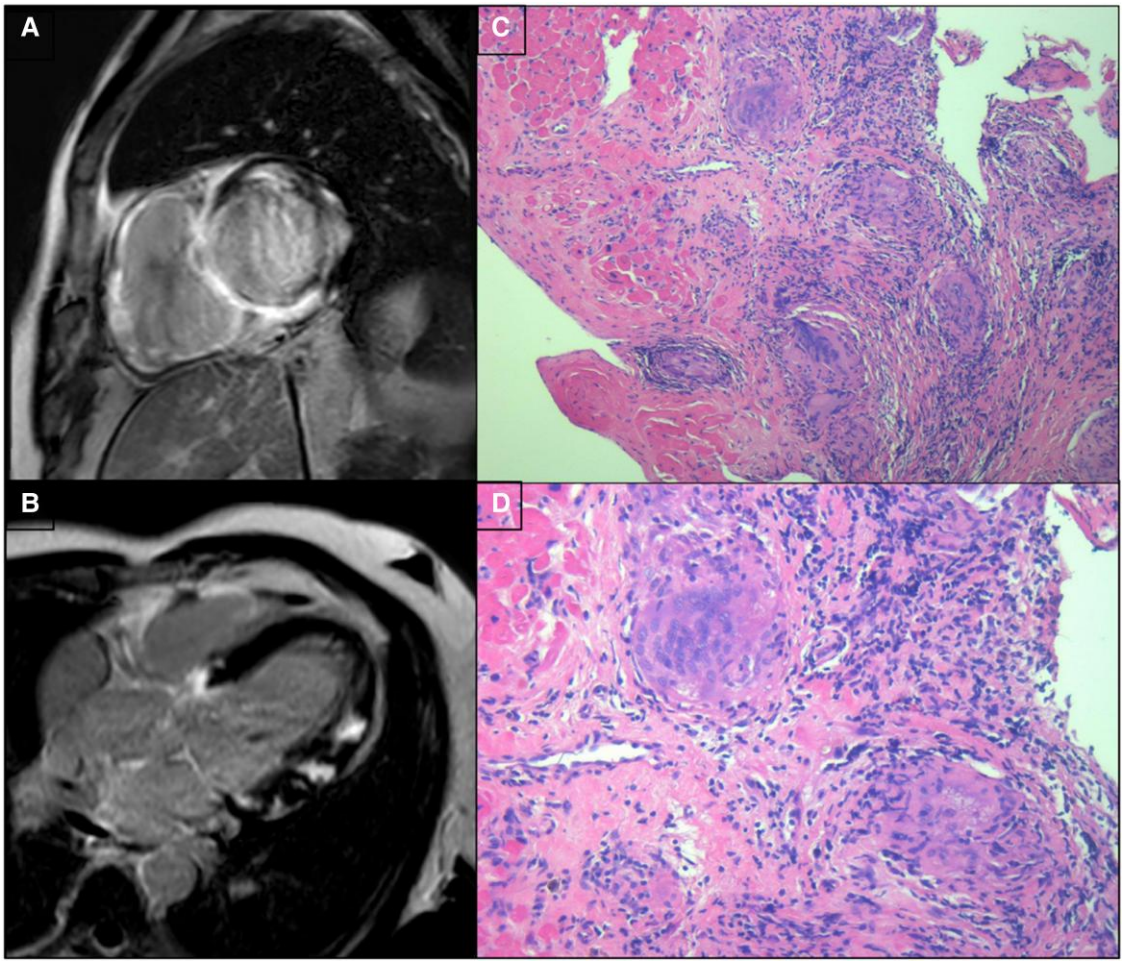

Figure 5 Cardiac magnetic resonance and histology of cardiac sarcoidosis. In this figure is presented in the panel A the cardiac magnetic resonance of a 56-year-old man, affected by cardiac sarcoidosis, in which is present septal and inferior non-ischaemic LGE in left ventricle and also diffuse LGE in right ventricle. In the panel B, there is a four chamber section showing the patchy LGE distribution in basal interventricular septum, mid-basal lateral wall and free right ventricle wall. In the panels C and D are presented the non-necrotizing granulomas. LGE, late gadolinium enhancement.

ECG as a milestone of precision medicine in DCM

Dilated cardiomyopathy is a primitive heart muscle disease, which affects prevalent male and young adults, and the unfavourable outcome is driven both by heart failure and arrhythmic events. The prognostication, and specifically the arrhythmic stratification, of this population remains a clinical challenge, and should be based mainly on an aetiological diagnosis.[16] In this perspective, the ECG is emerging as an important tool to use, but its reading should be based on a critical and deep ECG reading. The systematic interpretation of *P* waves, PR duration, Q waves presence, high or low QRS voltage, QRS fragmentation, T wave inversion, through a 'red flag' approach, represents the milestone in the diagnostic work up (*Table 1*), prognostication (mostly arrhythmic) and therapeutic strategies and monitoring.

In contrast to ischaemic cardiomyopathy, the pathophysiology of ventricular arrhythmias in DCM is less well understood. Arrhythmogenesis is likely multifactorial and may be related to structural changes such as fibrosis and left ventricular dilation as well as to primary and secondary electrophysiological changes; these may result in ventricular tachyarrhythmias due to re-entry, abnormal automaticity, and triggered activity.[12]

In previous studies, both diagnostic and prognostic ECG features have been considered in the management of DCM, both for heart failure and arrhythmic prognostication.[17,18] However, it is important to note that no one has never studied the phenotype in depth, including: family history, critical ECG reading, echocardiography, CMR and eventually endomyocardial biopsy. Paldino et al.[19] have recently observed that a genotype based stratification, on top of phenotype characterization, has an incremental role in the arrhythmic stratification of left-sided, non-hypertrophic cardiomyopathy patients. An important feature of ECG findings in the management of DCM is related to the possibility to suggest DCM genotype positive patients with an arrhythmogenic mutation.[20] Therefore, genetics and phenotype characterization should be complementary in the comprehensive and modern work up of the patient with DCM, toward true precision medicine.

Furthermore, the evaluation of heart failure related outcome can be evaluated through various ECG sign, as seen in the first and the third ECG tracing.

Table 1 Electrocardiogram features to critically and systematically evaluate in diagnostic work up of DCM patients

ECG sign	Suggested DCM diagnosis
Prolonged PR tract	Sarcoidosis Genetic DCM: LMNA; SCN5A; DES; EMD; Chagas disease
Q waves	Genetic DCM: DMD;
Left bundle branch block	Genetic DCM: LMNA;
Right bundle branch block	Sarcoidosis Arrhythmogenic right ventricle cardiomyopathy Genetic DCM: DMD;
Fragmented QRS complex	Myocarditis Arrhythmogenic right ventricle cardiomyopathy (including Epsilon waves)
Low QRS voltages	Myocarditis Arrhythmogenic right ventricle cardiomyopathy Genetic DCM: FLNC; DSP; PLN;
High QRS voltages	Hypokinetic hypertensive DCM
T waves inversion	Myocarditis (especially in the lateral leads) Arrhythmogenic right ventricle cardiomyopathy Genetic DCM: FLNC; DSP;

DES, desmin; DMD, dystrophin; DSP, desmoplakin; EMD, Emery-Dreifuss; FLNC, filamin C; LBBB, left bundle branch block; LMNA, lamin A/C; PLN, phospholamban; RBBB, right bundle branch block; SCN5A, cardiac sodium channel type 5 α-subunit

Future perspectives

In conclusion, despite in the era of artificial intelligence, genetics multimodal imaging, electrocardiography still represents a key diagnostic and therapeutic tool in the work up of DCM patients. It is therefore essential to evaluate individual ECGs by systematically enhancing all their individual aspects, considering the ECG not only as a first-level examination but as a true complementary 'tissue characterization' examination.

There is a developing need for a more modern classification of cardiomyopathies and DCMs in particular. In this context, critical reading of the ECG is a key tool for clinical management of the patient, both diagnostically and prognostically towards a real precision medicine applied in DCM.

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Data availability

No new data were generated or analysed in support of this research.

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