Biopsy-Verified Lymphocytic Myocarditis in a Previously Healthy 82-year-old Male

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Vaccine-induced lymphytic myocarditis (VILM) is a rare adverse effect following vaccination against covid-19, with most cases occurring in younger males following the second vaccine dose. We present the case of an 82-year-old male developing biopsy-verified lymphocytic myocarditis following a combined administration of a seventh dose of covid-19 mRNA vaccine, and a 23-valent pneumococcal polysaccharide vaccine. The aim is to emphasize the importance of early cardiovascular magnetic resonance (CMR) imaging in patients with chest pains and nonspecific ECG-changes, and to encourage vigilance for VILM as a differential, even in older patients and in patients with previously tolerated vaccination.

A vital and physically active 82-year-old male admitted with chest pains and elevated troponins was investigated using echocardiography, angiography, CMR and endomycoardial biopsy. ECG showed no ST-elevations and a newly onset right-bundle branch block. There was general hypokinesia on echocardiography with a reduced EF, and coronary angiography showed no stenosis. CMR revealed extensive LGE and elevated T1- and T2, and endocardial biopsy revealed lymphocytic infiltration of the myocardium. The findings were consistent with lymphocytic myocarditis and attributed to vaccination five days prior to presentation. The patient was treated with corticosteroids and was discharged on optimal medical therapy. A 6-month follow-up CMR revealed resolution of edema and a normalised EF.

VILM is to be considered a differential diagnosis even in older patients and in patients with previously tolerated vaccine doses. To the best of our knowledge, this is the first biopsy- and T2 mapping- confirmed reported case later than the third dose of covid-19 vaccine. This case is remarkable for the extensive involved myocardium and for the atypical setting, as similar cases are mainly reported in younger patients and rarely following a second vaccine dose. The case emphasizes the importance of CMR in the diagnosis of myocarditis, as per the updated 2025 ESC Guidelines.

Astrovirus associated acute myocarditis: a rare extra-intestinal manifestation

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This case is reported to enhance understanding of the potential cardiac tropism of astrovirus and its role in extra-intestinal disease. While astrovirus infection has been associated with extra-intestinal manifestations such as encephalitis and meningitis, only one case of cardiac involvement has been previously reported in literature.

A 21-year-old man with a past medical history of irritable bowel syndrome presented with three days of non-bloody, watery diarrhea accompanied by fever. On the third day of illness, he developed acute retrosternal, non-radiating chest pain. Electrocardiography revealed new diffuse ST-segment elevations, more pronounced in leads II, III, and aVF. Serum electrolytes were within normal limits, while cardiac biomarkers and C-reactive protein were elevated. Transthoracic echocardiography demonstrated normal right ventricular function and mild left ventricular dilatation with lateral and posterior wall hypokinesis, resulting in moderately reduced systolic function (left ventricular ejection fraction 40–45%). A small pericardial effusion was also noted. Cardiac magnetic resonance imaging was consistent with acute myocarditis. Laboratory evaluation showed leukocytosis. An extensive infectious workup, including a gastrointestinal pathogen panel, was positive for astrovirus. The patient was treated conservatively with an angiotensin receptor blocker, and no recurrent ventricular arrhythmias were observed during hospitalization. No additional complications occurred, and the patient was discharged in good clinical condition.

Wide QRS complex tachycardia associated with AVNRT

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Introduction: AVNRT is a common cause of narrow QRS complex tachycardia. The present case is a rare example of wide QRS complex tachycardia caused by this mechanism.

Case presentation: A 76-year-old man with a history of paroxysmal atrial fibrillation was admitted to the emergency department with recurrent palpitations, feeling of weakness, without loss of consciousness or angina. During observation, telemetry recorded wide QRS complex tachycardias, which were classified as ventricular. The patient was transferred to the cardiac intensive care unit. Intravenous Cordarone was added to the treatment with rapid cessation of the arrhythmia. During further observation, paroxysmal tachycardias of various morphologies were observed, both with wide QRS complexes (RBBB or LBBB morphology) and with narrow QRS complexes. After analyzing the ECG recordings, premature P-waves were observed before the tachycardias, which raised the suspicion of a supraventricular arrhythmia. The patient was transferred to a university hospital for electrophysiological testing. AVNRT was confirmed as the cause of the arrhythmia and ablation of the slow pathway was performed.

Conclusions: In the present case, AVNRT caused an ECG pattern that was atypical for this arrhythmia. The presence of premature P waves, with prolonged PQ at the begining of the tachycardia, is typical of AVNRT and suggests this arrhythmia as the cause of the problem. This phenomenon was observed in the presented patient, which helped to make the correct diagnosis, despite the presence of wide QRS complex tachycardia.

A thorough analysis of ECG recordings is necessary in each case to recognize less typical arrhythmia patterns and plan the most effective treatment.

From atrioventricular block to dilated cardiomyopathy: A case of LMNA gene–related disease

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Introduction: Early onset (< 50 years of age) of atrioventricular block (AVB) can be a sign of underlying cardiomyopathy (CM), sarcoidosis or myocarditis. We report a case of lamin A/C gene related CM with 13 years between first presentation and the diagnosis of the genetic background.

Case presentation: A male patient in his early 40s presented with presyncope. Intermittent AVB II/2 was seen as the cause and a DDD pacemaker implanted. Diagnostics including echocardiography and cardiac magnetic resonance imaging did not show abnormalities. A family history was said to be unremarkable.

During follow-up the patient developed paroxysmal atrial fibrillation (AF) and flutter. Despite two ablations AF became permanent. Ten years after initial presentation the patient developed symptomatic left ventricular systolic dysfunction that was treated with medical therapy and CRT-D. Family history was again investigated on the background of AVB, AF and dilated cardiomyopathy, revealing that the patient's half-brother and two of his daughters had been diagnosed with dilated cardiomyopathy. Recently performed genetic testing in these relatives had shown a pathogenic variant in the lamin A/C gene (LMNA). This information led to the identification of the same mutation in our patient and one of his children.

Conclusions: This case illustrates the typical course of LMNA cardiomyopathies with ECG abnormalities occurring several years before the onset of ventricular dysfunction and symptomatic heart failure. Even more significantly it illustrates the importance of a high suspicion of underlying heart disease in early onset AVB and AF, as well as the value of taking a thorough family history. LMNA cardiomyopathy is associated with a risk of life-threatening ventricular arrhythmias. In retrospect and even when considering the progress of cardiogenetics during the 13 years of follow-up, several clues were overlooked. Paying attention to these details facilitates early diagnosis and the possibility of preventing sudden cardiac death.