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Sudden cardiac death: a case of fulminant myocarditis

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Topic(s):
Myocarditis

Citation:
Introduction: A 31-year-old man collapsed while driving his car, without any warning signs. His co-driver immediately started basic life support. The first documented rhythm was ventricular fibrillation (VF). Repeated external defibrillation was unsuccessful and resuscitation was continued while patient was transported to the hospital.

Management: Veno-arterial extracorporeal life support was initiated after a resuscitation time of 90 minutes. Quick-look transthoracic echocardiography (TTE) displayed an akinetic heart without major signs of hypertrophy. Coronary angiography demonstrated normal coronary arteries. TTE 12 hours later showed extensive wall thickening and a depressed systolic function (EF<10%, fig. 1). Despite optimal medical treatment and circulatory support, brain death was diagnosed 48 hours after admission and the patient died after withdrawal of life support.

There was no relevant medical or family history. Autoimmune and toxicological screening came back negative. At necropsy, the heart weighed 520 grams. The left ventricular wall was thickened. The myocardium had grey-yellow streaks due to hemorrhagic and necrotic tissue. Microscopical examination revealed fulminant lymphocytic infiltration and myocytolysis (fig. 2). Postmortem viral PCR on the myocardial tissue was negative.

Differential diagnosis: Sudden cardiac death (SCD) is usually caused by an acute coronary syndrome (ACS). However, the patient had no risk factors and coronary angiogram ruled out ACS. Structural heart disease can also cause SCD, but TTE on admission showed normal ventricular dimensions. Nevertheless, pronounced myocardial hypertrophy was documented on TTE 12 hours after admission. Given the autopsy results, this is likely due to severe myocardial edema caused by fulminant myocarditis, which likely caused the VF. Underlying genetic cardiac conditions are possible, but genetic examination is still ongoing.

Conclusion: Myocarditis is a not uncommon cause of SCD. Patients mostly have symptoms such as chest pain or heart failure. Rarely, SCD may be the first presenting symptom. Echocardiographic signs suggestive of myocarditis can be absent on admission, but can evolve quickly. TTE is necessary in the workup for myocarditis and should be repeated if there is a high index of suspicion. Current guidelines advocate endomyocardial biopsy when myocarditis is complicated by life-threatening arrhythmias. In this case, the lack of structural myocardial abnormalities on admission was deemed sufficient to withhold myocarditis as a possible cause of SCD. PCRs for cardiotropic viruses were negative, but tissue was obtained after patient passed away. This case should alert clinicians to the possibility of myocarditis as cause of SCD and repeat TTE if no definite diagnosis has been made. There should be a low threshold to perform an endomyocardial biopsy and start cardiogenetic work-up in the acute setting if coronary atherosclerosis is ruled out in patients with VF.
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