High-Degree Atrioventricular Block And Takotsubo Cardiomyopathy: Coincidence Or Connection?

L Samuelov-Kinori, E Leshem-Rubinov, D Lahav, O Touvia, I Laron, D Justo

Citation

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Abstract

We present an elderly patient with high-degree atrioventricular block and Takotsubo cardiomyopathy, and we review the literature for several mechanisms that may explain this association.

INTRODUCTION

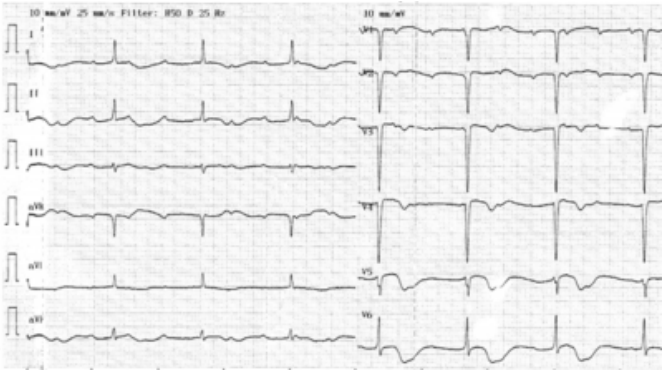
High-degree atrioventricular block (AVB) and Takotsubo cardiomyopathy (TC) have been reported together several times in the medical literature [1-9]. Still, the association between the two is not clear: most authors believe that TC is associated with myocardial ischemia which triggers an atrioventricular conduction disorder. We present a patient with both conditions in order to discuss several mechanisms that may explain this association.

CASE PRESENTATION

An 85-year-old woman collapsed at home and hit her head. She did not recall what had happened before the collapse. Her medical history was consistent with hypercholesterolemia, osteoporosis, and a transient second-degree AVB that was documented eight months earlier. She took no anti-arrhythmic agents. Upon arrival to the emergency room she was dyspneic and a chest roentgenogram showed pulmonary congestion. ECG showed a third-degree AVB with a ventricular escape rhythm of 45 beats/min and deep inverted T waves in the chest leads (figure 1).

Figure 1

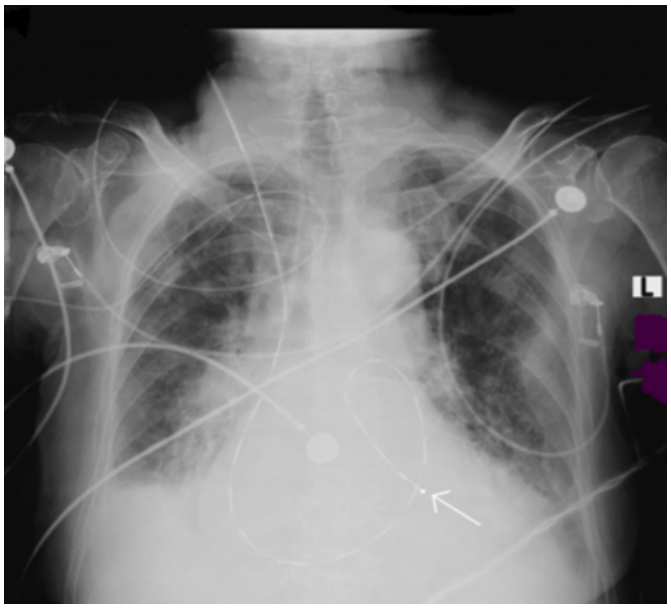
Figure 1:



A temporary cardiac pacemaker was placed immediately (figure 2).

Figure 2

Figure 2:



No skull fractures or intracranial bleeding were documented by head computer tomography. Few hours later, dyspnea became worse and Troponin I levels increased from 1.032 ng/ml to 2.738 ng/ml. Although myocardial infarction was suspected, coronary angiography showed no obstructive coronary artery disease the following day. Moreover, ventriculography showed an akinetic apex consistent with the typical contractile pattern of TC. Echocardiography showed a severely depressed left ventricular function with an ejection fraction of 35%. Since the third-degree AVB was still present two days later, a permanent VVI cardiac pacemaker was implanted. Two weeks later, a second echocardiography showed no apical wall motion abnormalities and a less depressed left ventricular function (ejection fraction of 45%). The patient was discharged in a good clinical condition.

DISCUSSION

There is no doubt the presented patient has had high-degree AVB and TC. But it is not clear which of the conditions has triggered the other. Two scenarios may explain this clinical presentation: In the first one, the patient collapsed because of a high-degree AVB, and TC appeared because of stress secondary to immobility and/or head trauma; In the second scenario, high-degree AVB and TC appeared together causing collapse and dyspnea, respectively. This issue is not theoretical what so ever; it is possible that a pacemaker insertion eight months earlier due to a transient second-degree AVB might have prevented collapse and a stress-associated TC.

In few case reports, TC appeared following a cardiac pacemaker insertion due to high-degree AVB, and some authors suggested that emotional distress following a cardiac pacemaker insertion might cause TC [7-9]. Dyspnea, pulmonary congestion, elevated Troponin, and large inverted T waves in the ECG's chest leads upon arrival to the emergency room, all suggest TC prior to pacemaker insertion in our case. Hence, this scenario is probably irrelevant to our patient.

In most case reports, high-degree AVB and TC appear together [1-6]. Hence, most authors suggest that diffuse spasms in small branches of the coronary arteries consistent with TC might cause myocardial ischemia and atrioventricular conduction disorder. However, myocardial ischemia in TC is transient, and in most of these cases, high-degree AVB has continued although contractile dysfunction has resolved, necessitating a permanent cardiac pacemaker implantation. It is possible that contrary to the contractile system, the recovery of the cardiac conduction system is prolonged in TC. Indeed, Nault et al. have demonstrated in an electrophysiological study no resolution of a high-degree AVB one year following TC, but eventually the block has resolved two years later [6]. Still, the earlier transient second-degree AVB in our patient implies for the presence of a conduction disorder in the first place, probably making the high-degree AVB and TC a misfortune coincidence rather than a disease and its cause.

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Author Information

Liat Samuelov-Kinori, MD
Department of Internal Medicine, Tel-Aviv Sourasky Medical Center

Eran Leshem-Rubinov, MD
Department of Internal Medicine, Tel-Aviv Sourasky Medical Center

Dror Lahav, MD
Department of Internal Medicine, Tel-Aviv Sourasky Medical Center

Oholi Touvia, MD
Department of Internal Medicine, Tel-Aviv Sourasky Medical Center

Ido Laron, MD
Department of Internal Medicine, Tel-Aviv Sourasky Medical Center

Dan Justo, MD
Cardiology, Tel-Aviv Sourasky Medical Center