Letter to the Editor

Persistent Pacemaker Dependence after Takotsubo Recovery Suggests that Complete Heart Block is the Trigger of Takotsubo

Dear Editor,

I enjoyed reading the article by Dubey et al[1] regarding a 53-year-old woman who presented with complete heart block (CHB) and a 73-year-old woman who presented with syncope and CHB, both in the setting of having suffered Takotsubo syndrome (TTS). Both patients received a permanent pacemaker (PPM) implantation due to conduction delays, persisting after left ventricular function recovery. Indeed, both patients at their 6-month follow-up revealed 90% and 98% pacemaker dependency rates, correspondingly.

I will be grateful to the authors for a response to the following remarks and inquiries:

1. I agree with the authors that bradyarrhythmias (ie, sinus bradycardia, pulseless electrical activity, first and second-degree atrioventricular blocks, CHB, and sinoatrial blocks), in contrast to tachyarrhythmias, in patients with TTS, are not well characterized and evaluated. CHB, in particular, presents vexing problems regarding its pathophysiology, clinical implications, and management.[1]
2. To invoke the analogy of "chicken and egg", one wonders whether CHB is a complication of TTS, like many tachyarrhythmias, or precedes TTS, acting as the trigger for TTS (via a compensatory catecholamine surge to the CHB) to emerge.
3. Perhaps in some TTS cases, the patients may be able to provide a history suggesting that chest pain and dyspnea preceded or followed the symptoms of faintness, dizziness, or presyncope, suggesting that CHB was a complication or the trigger of TTS, respectively. I wonder whether the authors could elicit such information from their patients.
4. The bulk of the TTS literature echoes the present study's findings, revealing that CHB persists after left ventricular function recovery.[1] PPM implantation frequently becomes necessary, and PPM dependency is confirmed at follow-up. However, the overall consensus is that the management of CHB in patients with TTC remains controversial, and the decision to proceed with PPM implantation continues to be case-dependent.[2-4]
5. Occasionally, one could consider that the trigger for TTS emergence is the administered catecholamines (often epinephrine) with or without atropine, for the management of bradycardia associated with CHB.[4]
6. Finally, another point favoring the notion that CHB is the trigger of, then the complication of, TTS is animal work showing that the Purkinje network tissue is more resistant to ischemia than the cardiomyocytes. In a porcine animal model, Purkinje cells survived after 90 minutes of ischemia and subsequent reperfusion to a significantly greater degree than cardiomyocytes.[5] Additionally, in a dog animal model, subendocardial Purkinje fibers survived after an extensive myocardial infarction.[6] These findings suggest that more intense or prolonged ischemia would be required for the development of atrioventricular conduction abnormalities; thus, the emergence of CHB as a complication of TTS appears unlikely. Furthermore, myocardial edema, micronecrosis, and subtle fibrosis, resulting from TTS, which could lead to CHB, would be expected to materialize later in the clinical course of TTS and are not early features of TTS.

Best regards,

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Persistent Pacemaker Dependence after Takotsubo Recovery Suggests that Complete Heart Block is the Trigger of Takotsubo: A Reply

Dear Editor,

Thank you for the submission. In one of the patients, chest pain and syncope occurred simultaneously; hence, it is difficult to ascertain whether CHB was the cause or effect of TTS. In the other patient, presyncope preceded other symptoms, and CHB seems to be the cause of TTS. The need for permanent pacing and the reasons for it are well articulated in the submission.

Best regards,

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