Takotsubo Syndrome After Permanent Pacemaker Implantation

To the Editor:

I enjoyed reading the interesting case report by Dias et al, published in the November 2013 issue of the Journal,1 of a 72-year-old woman who was being treated with prednisone and methotrexate for rheumatoid arthritis (RA), and suffered Takotsubo syndrome (TTS), after an uncomplicated permanent pacemaker (PPM) implantation. Similar cases of patients with TTS in the aftermath of PPM implantation have been previously published, as the authors stated in their review of the literature. In addition to the consideration that the PPM implantation was the culprit, one can take the view that her complete heart block (CHB) was due to TTS as noted previously,2 and that she had been developing milder attacks of TTS in the 2 weeks prior to her original clinical presentation, with a resultant residual reduction of her left ventricular ejection fraction to 40%-45%. The authors also appropriately emphasized that CHB per se, related or unrelated to her RA, could have caused the TTS, as a result of stress from symptoms emanating from CHB. If CHB does not persist at follow-up, it may be supportive of the notion that TTS was its cause, although the comorbidity of rheumatoid arthritis complicates matters. Irrespective of the mechanism of TTS in this patient with TTS following PPM implantation, it appears that this procedure (along with many other diagnostic and therapeutic procedures to which our patients are subjected) is a reason to make us rethink our current modus operandi and consider, unless contraindicated, universal prophylactic administration of sedatives and beta-adrenergic blockers in patients being prepared for PPM implantation. Also, since there is a possibility that milder forms of TTS exist but remain undiagnosed, a high index of suspicion among physicians implanting PPMs is in order, so that they explore the etiology of even mild symptoms via electrocardiograms (ECG) and echocardiograms. Finally, this reader wonders whether the authors have recorded an unpaced (“magnet”) ECG during the PPM interrogation, corresponding to Figure 2 of their paper; such an ECG (if available) probably revealed an attenuated QRS voltage in the limb and lateral precordial leads, a new diagnostic ECG sign of TTS that has just been reported.3

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References

Author’s Reply:

We appreciate your comments regarding our report. We agree it is possible this patient could have been experiencing milder attacks of TTS in the 2 weeks prior to her presentation at the emergency department and as a result of it, she may have developed a CHB; however, we could not identify any specific event (psychological/physiological stressor or new drug) that could have triggered TSS. We wonder if her prior symptoms were mostly due to her CHB and the PPM implantation was the real culprit.

Her follow-up ECGs have shown normal sinus rhythm with electronic ventricular pacemaker. Follow-up echocardiogram obtained approximately 45 days after discharge showed an ejection fraction of 50%-55% and apical and septal wall-motion abnormalities believed to reflect pacemaker activation and significantly improved.

Unfortunately, we did not record an unpaced ECG during PPM interrogation, which we agree would have been relevant given the recently published paper mentioned in your letter.

Regarding the possible “universal prophylactic administration of sedatives and beta-adrenergic blockers in patients being prepared for PPM implantation,” we are unsure of its relevance. Among patients diagnosed with TSS, PPM does not seem to be a common trigger.1,2 In our own recently published experience,3 only 1/78 patients developed TSS in the setting of PPM, so we wonder about the benefits of a prophylactic beta-blocker administration, but we definitely agree there should be a high index of suspicion and low threshold for diagnosing TSS. More studies are probably needed to determine its risks and benefits.

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References