Reader Comments


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Reader comments

INADVERTENT INJECTION OF NOREPINEPHRINE, TAKOTSUBO SYNDROME, AND LOW-VOLTAGE ELECTROCARDIOGRAM

Read with interest the report by Sherif et al, published in the April 2016 issue of Proceedings (1), about the 76-year-old woman who was diagnosed with takotsubo syndrome (TS) after inadvertent injection of 4 mg of norepinephrine. The topic of iatrogenic epinephrine administration and TS was recently reviewed (2), and 3 of the 22 cases included patients who received epinephrine inadvertently either for the specific drug or for a larger-than-ordered dose; in addition, another patient, not included in the review (2), who inadvertently received an infusion of 4.5 mg of norepinephrine suffered TS and subsequently received infusions of epinephrine and norepinephrine for hypotension (3). Thus, this is not the first report of TS secondary to iatrogenic norepinephrine injection, as the authors state. However, the reason for this letter is to inquire whether the low voltage of the QRS complexes, in both the limb and precordial leads, in the presented electrocardiogram (ECG) was transient, as could be ascertained by a comparison of the admission ECG with previous ECGs of the patient and ECGs recorded during her hospitalization or at ambulatory follow-up. Transient low-voltage QRS complexes in association with TS have been reported recently (4).

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The authors respond:

It was a pleasure to receive a reader letter that has questions related to our recently published case report (1). I would like to clarify some points that Dr. Madias made. Dr. Madias mentioned that our case was not the first case about iatrogenic norepinephrine-induced takotsubo cardiomyopathy (TC) and referenced a review article (2). The review article concerned articles related to epinephrine induction of TC, while in our case the offending agent was norepinephrine. We agree on the similarity between these two catecholamines, but the purpose of our case report was to highlight norepinephrine as a substance that can cause this condition. In the other case report (3) that Dr. Madias mentioned, it was not clear if the patient had the diagnosis of TC, as there was no mention of whether the patient had coronary angiography to rule out coronary artery disease. Furthermore, the patient in that case report first received norepinephrine and then was later placed on both epinephrine and norepinephrine, and it was not clear if that echocardiography was done after the first exposure to norepinephrine alone or after it was combined with epinephrine. Regarding QRS voltage, the ECG on admission showed a normal QRS voltage, which might support the idea of the effect of TC on ECG voltage. Dr. Madias referred to an interesting finding in his article (4), and certainly more observational studies are needed.

—Khaled Sherif, MD, Sharmila Sehli, MD, and Leigh A. Jenkins, MD
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AN INTERESTING ELECTROCARDIOGRAM

A better interpretation of the electrocardiogram on page 165 of April’s Proceedings (Occam’s razor) (1) is type 1 second-degree heart block (Wenckebach) with intermittent A-V dissociation caused by an escape junctional rhythm faster than half the atrial rate. Complexes 8, 10, 13, 15, 18, 20, 21, and 23 are all conducted beats. The others are all junctional escape beats. The proof is in complexes 20 and 21, where there is a progressive lengthening of the PR interval in two consecutive conducted beats. When the P wave falls when the A-V node is not refractory, it is conducted albeit by an escape junctional rhythm faster than half the atrial rate. Complexes 20 and 21 are both conducted beats. The others are all junctional escape beats. The proof is in complexes 20 and 21, where there is a progressive lengthening of the PR interval in two consecutive conducted beats. When the P wave falls when the A-V node is not refractory, it is conducted albeit at a prolonged A-V interval. The P-P intervals are slightly shorter when they contain a QRS complex—a known phenomenon.

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The author responds:

There is no disagreement over the interpretation. The key to understanding this rhythm is to identify the ectopic junctional pacemaker. Once that is done, the remainder is simple Wenckebach. In summary: Sinus rhythm (75 bpm) with AV block, right bundle branch block, competing junctional pacemaker (39 bpm). The easiest way to identify the junctional pacemaker is by its distinct cycle length.

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