Letter to the Editor — Editöre Mektup

A patient with ventricular fibrillation and inverted Takotsubo syndrome triggered by sinus surgery: plausible causes, and electrocardiographic features

Dear Editor,

I read with great interest the report by Demir et al. published in the July 2016 issue of the journal[1] about the 27-year-old woman who suffered ventricular fibrillation (VF) while recovering from propofol anesthesia after undergoing endoscopic nasal sinus surgery, and who, after successful resuscitation, was diagnosed with inverse Takotsubo syndrome (TTS), a phenotypic variant more commonly seen in young patients than classic Takotsubo cardiomyopathy.

Pathophysiology of TTS continues to elude us. What could have been plausible trigger(s) of TTS in this patient? 1) I will assume that the "osteal plaque formation in the right coronary artery,"[1] did not have any physiological significance. 2) It is possible that the procedure itself (endoscopy and/or surgical trauma) were at the root of TTS. 3) Also, anesthesia with propofol has been associated with TTS at least in 7 cases.[2] 4) Since the patient developed VF while recovering from anesthesia, it is conceivable that VF preceded and triggered TTS.[3,4] 5) There is no information provided whether drugs were used during resuscitation (e.g., epinephrine), and one wonders about their potential role in precipitating TTS.[5,6] 6) On a different matter, a comparison of the 2 electrocardiograms (ECG) (Figure 1, recorded on admission, and Figure 4, recorded 1 month later[1]) reveals transient attenuation of the amplitude of the QRS complexes (attQRS) in leads II, III, aVR, and aVF, V1, V2, and V6, in keeping with a recent publication[7] associating attQRS and TTS attributed to myocardial edema and detected with cardiac magnetic resonance imaging (CMRI). Consequently, it will be contributory if the authors provide information whether there was myocardial edema in the cardiac basal wall in the CMRI. 7) Indeed, Figure 1 ECG[1] showed ST-segment elevation (STSE) in leads I and aVL, which have been previously associated with TTS affecting the basal and midventricular myocardial territories,[8,9] reflecting basal pathology that would be expected to produce STSE in high lateral leads and reciprocal ST-segment depression in the inferior and mid precordial leads. 8) Finally, since there was some ongoing "posterobasal hypokinesia" persisting at "strain and strain rate imaging analysis obtained prior to discharge,"[1] it will be informative to reveal whether there was any evidence of scar or fibrosis in the late gadolinium enhancement analysis of the CMRI.

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