IMAGE OF INTEREST

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Beat-to-beat alternans

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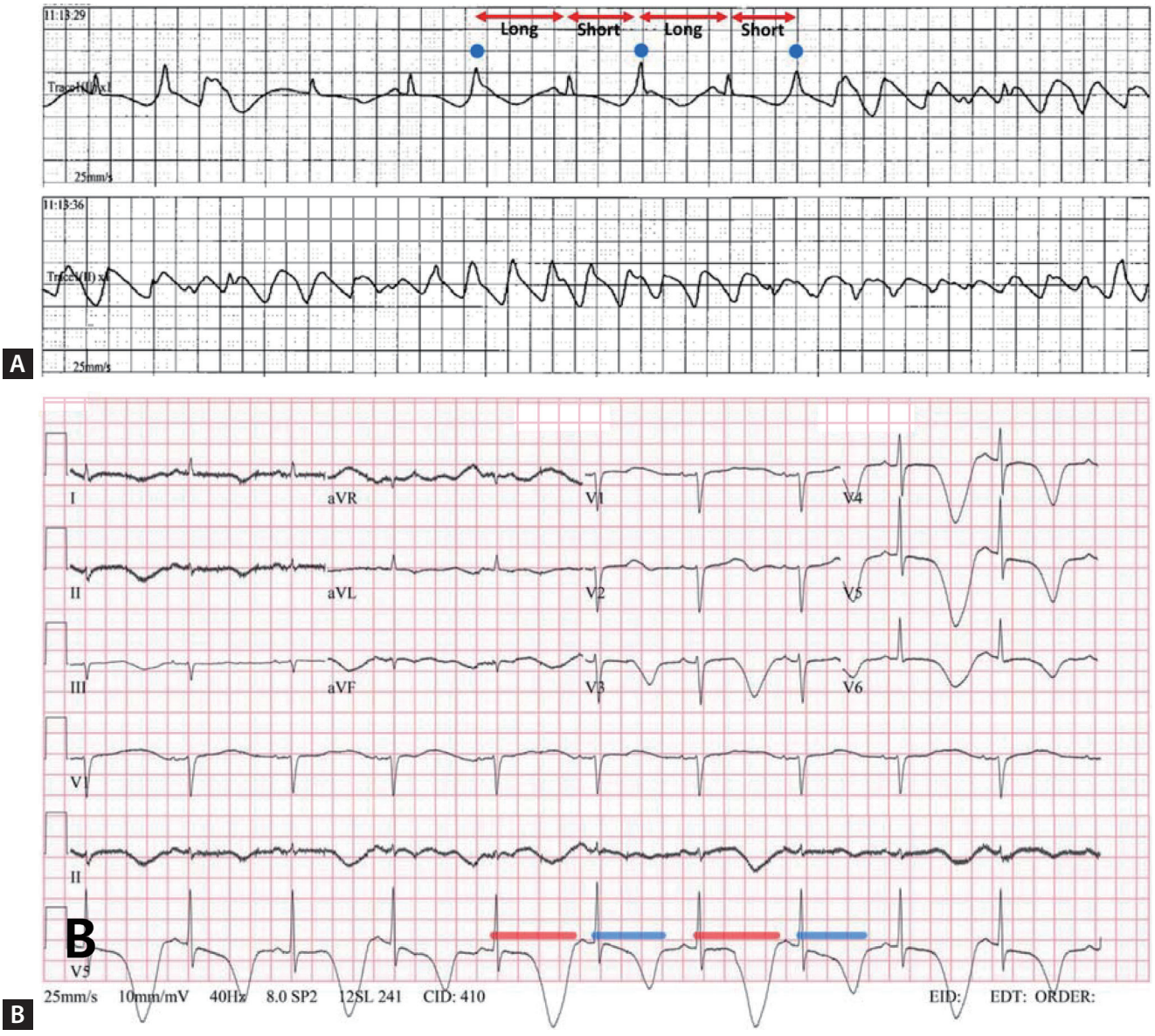

Figure 1. (A) The electrocardiogram strip shows a prolonged QT interval and frequent premature ventricular contractions (blue dots), which set up long-short sequences that initiated episodes of torsades. (B) The follow-up electrocardiogram revealed giant negative T-waves with a markedly prolonged QT interval. Note the beat-to-beat macroscopic T-wave alternans with an alternating change in the QT interval (red and blue bars).

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A 78-year-old male treated for pneumonia was referred to the cardiology department after post-cardiopulmonary resuscitation due to torsades de pointes (TdP) (Fig. 1A). The laboratory findings revealed an increased N-terminal pro-brain natriuretic peptide level of 569 pg/mL (normal range < 125 pg/mL) and an increased troponin-I level of 0.47 ng/mL (normal range < 0.04 ng/mL). With the cessation of ciprofloxacin, after an electrolyte correction and lidocaine infusion, the TdP episodes were no longer observed. Four days later, a follow-up electrocardiogram (ECG) (Fig. 1B) revealed a beat-to-beat macroscopic T-wave alternans (TWA) with an alternating change in the QT interval. Echocardiography revealed apical ballooning compatible with takotsubo cardiomyopathy (TCM). Considering his precarious condition, coronary angiography was not performed. Despite intensive combination antibiotic therapy, his pneumonia progressed, and he died.

TWA is a beat-to-beat alternation in the amplitude and/or shape of the T-waves on the surface ECG. These fluctuations of the T-wave are primary and unrelated to alternations in the other components of the ECG (i.e., QRS alternans). TWA has been described in patients with pathological conditions such as myocardial ischemia, long-QT syndrome, electrolyte imbalances, and TCM [1]. Also, TWA is closely associated with the development of ventricular arrhythmias [2].

This patient displayed interesting ECG findings. He initially presented with TdP, which was likely a result of the ciprofloxacin and abnormal electrolytes. After a correction of the above, he developed marked TWA, which was likely related to TCM. A likely mechanism of the TCM is catecholamine-mediated myocardial stunning. Catecholamines induce myocardial injury via a calcium overload [3], which may be related to alternans of cytosolic calcium [4]. Alternans of cytosolic calcium also cause fluctuations in the action potential duration; hence, TWA can be caused by a calcium accumulation [5,6] and is linked to mechanical alternans [7].

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Chan-Hee Lee: resources, data curation, writing - review & editing, supervision; Melvin M. Scheinman: conceptualization, writing - review & editing, supervision

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