A 64 years old male patient presented in A & E department with low grade fever since 2 days and one episode of presyncope associated with transient imbalance. In view of suspected cerebrovascular event, MRI stroke protocol was done, which was normal. ECG on presentation s/o rsR’ pattern with ST elevation in V1-V3 segment with T inversion without any reciprocal changes (Figure 1). Serial TROP I were normal. He had persistent changes on serial ECG (Figure 2). 2D echo s/o good LV systolic function with structurally normal heart. Patient was taken for coronary angiography which revealed insignificant coronary artery disease. Repeat ECG on the next day s/o persistence of ST elevation with T wave inversions (Figure 3).

With these findings it was suspected that patient could be having Brugada syndrome. Patient then taken for low dose isoprenaline (1µgm/min) challenge rather than inducing arrhythmia with sodium channel blocker following which patient had normalization of ECG changes (Figure 4) which supported the ECG diagnosis of Brugada syndrome with a type 1 pattern. Serial ECG (Figure 5) showed reversion to type I Brugada pattern with cessation of isoprenaline.

Figure 1: ECG on admission.
Figure 2: Serial ECG.

Figure 3: Serial ECG.

Figure 4: Post isoprenaline.
Brugada syndrome is a cardiac disease which demonstrates an autosomal dominant inheritance with variable expression and is associated with a pseudo Right Bundle Branch Block (RBBB), ST-segment elevation and terminal T-wave inversion in the precordial leads V₁ to V₃. The cellular basis or mechanism thought to underlie the ST-segment elevation and the higher susceptibility for ventricular fibrillation in Brugada syndrome is an imbalance of transmembrane ionic currents in the right ventricular epicardium resulting in a transmural voltage gradient due to the loss of the phase 2 action potential dome [1]. Studies show that sympathetic agonists can normalise the ST-segment elevation and prevent VF in Brugada syndrome. Intravenous isoproterenol administration is especially known to be effective in suppressing ST elevation in leads V₁ to V₃, and in restoring the action potential dome in experimental models of this syndrome and in patients with Brugada syndrome, because it increases the ICa secondary to an elevation in the intracellular level of cyclic AMP [2]. Thus, it is useful to differentiate from some of the mimics without much risk of inducing arrhythmia.

References