

ST elevation in neurogenically stunned myocardium

A 48-year-old woman was admitted with diagnosis of tubercular meningitis with multiple cerebral granulomas. She had acute onset respiratory distress and hemodynamic collapse. Patient was mechanically ventilated and started on inotropes.

Examination showed heart rate of 120/min, blood pressure of 90/56 mm Hg, left ventricular (LV) S3 and bilateral basal rales. A 12-lead electrocardiogram showed new-onset global ST elevations (figure 1A,B). A bedside troponin assay was negative. To rule out an acute myocardial infarction, a coronary angiography was done, which revealed normal coronaries (figure 2A–C). LV angiogram (figure 2D and E, see online supplementary video 1) showed severe LV systolic dysfunction with apical ballooning and preserved contractility of basal segments. Repeat troponin test was positive after 6 h. She received an extracranial shunt for

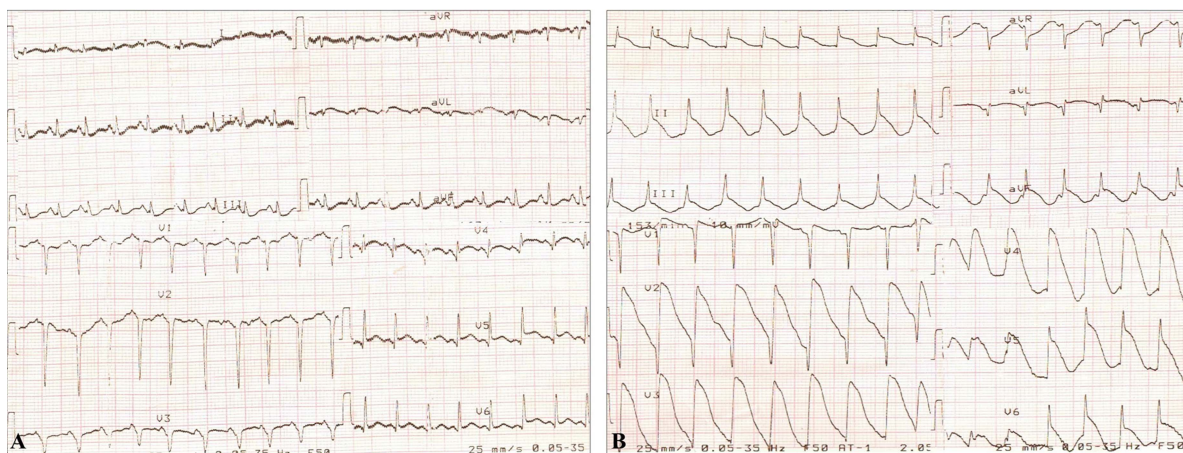


Figure 1 (A) Baseline electrocardiogram at admission, showing normal sinus rhythm and no significant ST-T changes. (B) Electrocardiogram at the time of acute event showing extensive ST elevations.

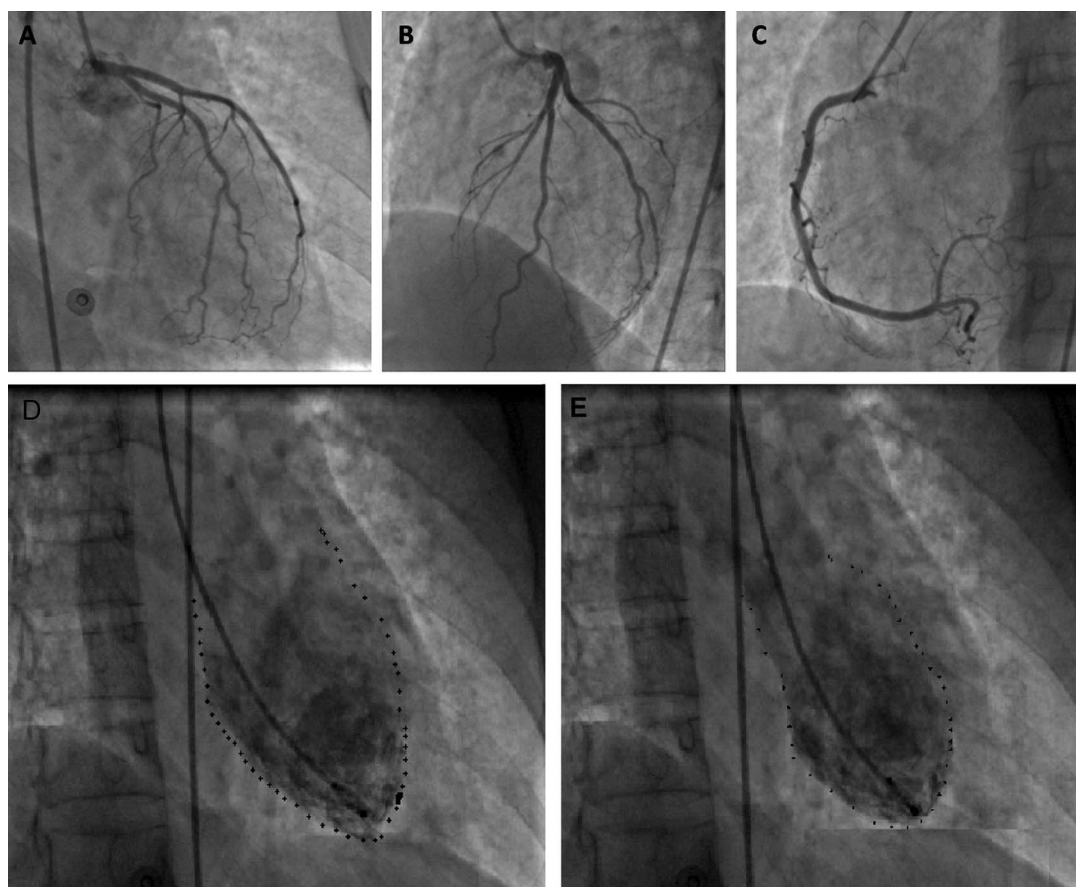


Figure 2 Coronary angiograms are showing normal coronary arteries. (A) Normal Left Anterior Descending (LAD) and Left Circumflex (LCX) coronary arteries in Right Anterior Oblique (RAO) caudal projection. (B) Normal LAD and LCX arteries in Left Anterior Oblique (LAO) cranial projection. (C) Normal Right Coronary Artery (RCA) in LAO cranial projection. (D) Left Ventricular (LV) angiogram in RAO projection in diastole, and (E) Systole, showing apical ballooning and preserved basal LV contractility.

progressive rise in intracranial tension. The patient succumbed to her illness 48 h later.

Neurogenically stunned myocardium (NSM) is a transient reversible LV dysfunction described to occur in the setting of sudden catecholamine surge during events of acute cerebral insult.¹ Sympathetic nerve-mediated myocardial release of catecholamine leading to myocardial damage is currently the most accepted hypothesis.² Both NSM and Takotsubo cardiomyopathy share a common pathophysiology and clinical course. With some differences in clinical presentation, both seem to represent the same clinical entity of stress cardiomyopathy.² Chest pain and ST elevations are more common in Takotsubo cardiomyopathy, unlike NSM, which more commonly present with pulmonary oedema.²

Such extensive ST elevations in our patient due to NSM are rarely seen.

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