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Neurogenic electrocardiographic changes with intermittent bundle branch block after interventricular hemorrhage

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A 65-year-old man presented with a presyncope episode associated with shortness of breath, palpitations, and sweating. He had a left cerebrovascular accident 2 years previously, but was not hypertensive. His blood pressure on admission was 251/138 mmHg and intravenous nitrate was initiated. On the 5th day of hospitalization, he tried to stand up to walk to the toilet, fainted, and collapsed to the floor. Cardiopulmonary resuscitation was administered for 10 minutes prior to the return of spontaneous circulation. He was intubated because of a low Glasgow Coma Scale ($E^1V^TM^1$) score. Inotropic support with dobutamine was started for hypotension postresuscitation. Electrocardiography (ECG) postresuscitation showed anterior ST segment elevation (Fig. 1). Hence, he was transferred to our center with a cardiac catheterization facility.

Serial ECGs did not show any changes indicative of acute myocardial infarction (AMI). In fact, subsequent ECGs revealed ST segment elevation in leads V1–V3 changing to a right bundle branch block (RBBB) pattern with ST segment elevation in lead aVR and widespread deep anterior T wave inversion and vice versa (Fig. 2). Noncontrast computed tomography of the brain showed a

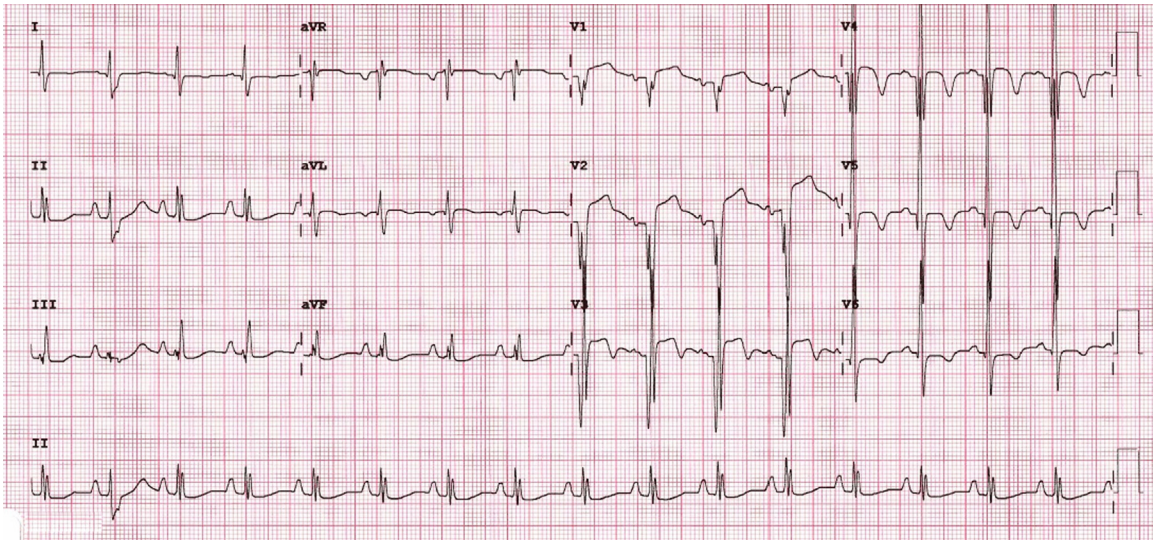

Fig. 1. Electrocardiogram showing anterior ST segment elevation and T wave inversion.

Conflicts of interest: none.

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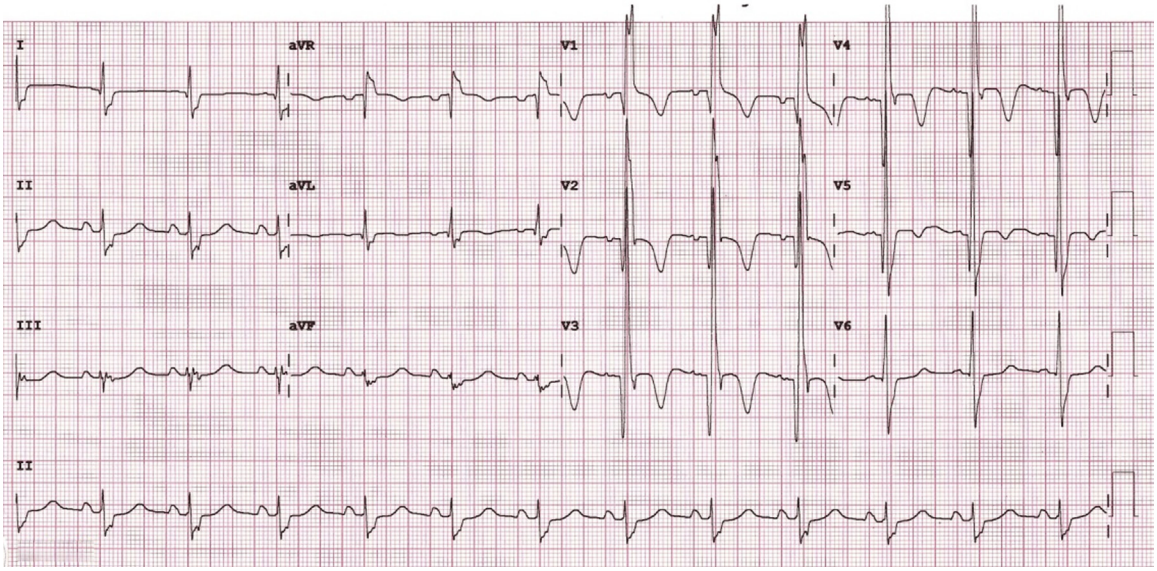

Fig. 2. Electrocardiogram showing ST segment elevation in lead aVR with a right bundle branch block pattern and widespread anterior deep symmetrical T wave inversion.

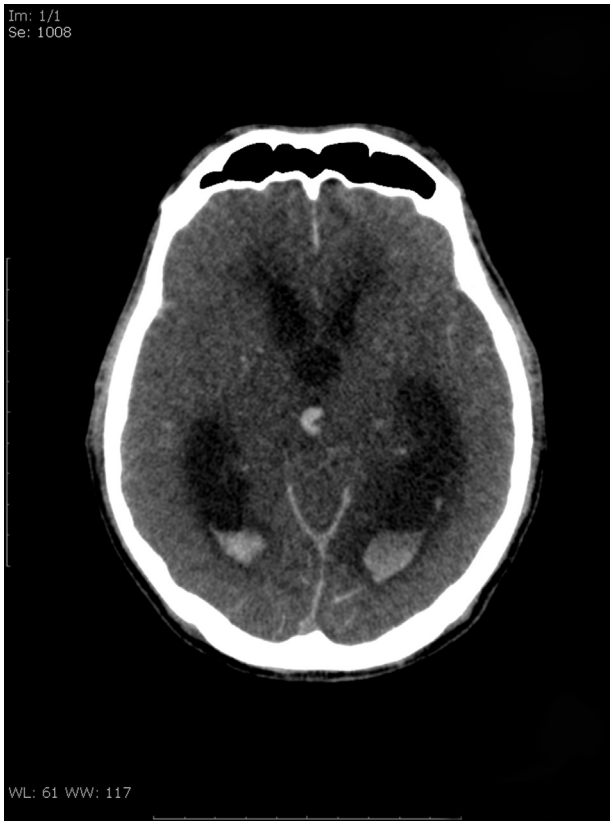

Fig. 3. Non-enhanced axial computed tomography of the brain at the level of the third ventricle reveals diffuse acute subarachnoid hemorrhage with intraventricular extension and communicating hydrocephalus.

generalized hypodense appearance of the cerebrum consistent with hypoxic brain damage. Bleeding was noted in all ventricles with extension into the upper spinal cord, along with obstructive hydrocephalus (Fig. 3). Cardiac enzymes were normal. No antithrombotic therapy was administered except at the referring hospital. The patient survived for another 3 days.

ECG findings of ST elevation after resuscitation for cardiac arrest may not always indicate AMI. Intracranial hemorrhage has been well described to mimic AMI [1]. As such, inappropriate administration of potent thrombolytic and antiplatelet agents would no doubt worsen the hemorrhage. Intracranial hemorrhage includes intracerebral hemorrhage, subarachnoid hemorrhage (SAH), and interventricular hemorrhage (IVH). Although IVH is rare, it has a very high mortality rate of 50–80% [2]. IVH is almost always associated with SAH and rarely happens as a single entity. The prognosis is extremely poor when there are increases in intracranial pressure, hydrocephalus, and eventual brain herniation.

ECG abnormalities have been described for intracranial hemorrhage, in particular intracerebral hemorrhage and SAH [3,4]. ST-segment elevation and T wave inversion may mimic the changes in AMI, as described in a case of traumatic SAH [1]. Other changes such as a prolonged QTc interval with large inverted T waves, pathological Q waves, ST-segment elevation or depression, and prominent U waves have been reported in SAH [3,4]. Our patient's cardiac enzymes were normal, precluding the likelihood of AMI due to acute coronary occlusion. The intermittent nature of RBBB and ST segment elevation in lead aVR has never been previously described in the setting of IVH or intracranial hemorrhage. It can be associated with an abnormal brain-neural milieu and excess catecholamines with possible deleterious effects on the myocardium and conduction system [5]. The findings of intermittent transient RBBB and ST segment elevation in lead aVR, as illustrated in this case, should be added to already-described neurogenic ECG changes in intracranial hemorrhage in the literature.

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