Intracranial Bleeding

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4.1 Case Report

A 40-year-old man was admitted to the emergency room after a 2.5 m fall from a scaffold. He showed signs of impaired consciousness and a scalp wound localized in the left tempofrontal region. Personal pathological history was unknown. The neurological evaluation revealed a Glasgow Coma Scale (GCS) score of 10: eye response = 3 (eye opening to verbal command), motor response = 6 (obeys command), and verbal response = 1 (no verbal response). The patient, after tracheal intubation for airway protection, underwent head computed tomography (CT) scan, which revealed bifrontal and left temporal subcortical-cortical bleeding with subarachnoid hemorrhage (Fig. 4.1). Subsequent chest x-ray, abdominal ultrasound, and chest-abdominal CT revealed no further traumatic injuries.

Vital Signs

- Heart rate: 80 bpm
- Blood pressure: 108/80 mmHg
- Oxygen saturation: 98 %

Physical Examination

- *General*: traumatic scalp wound 4 cm

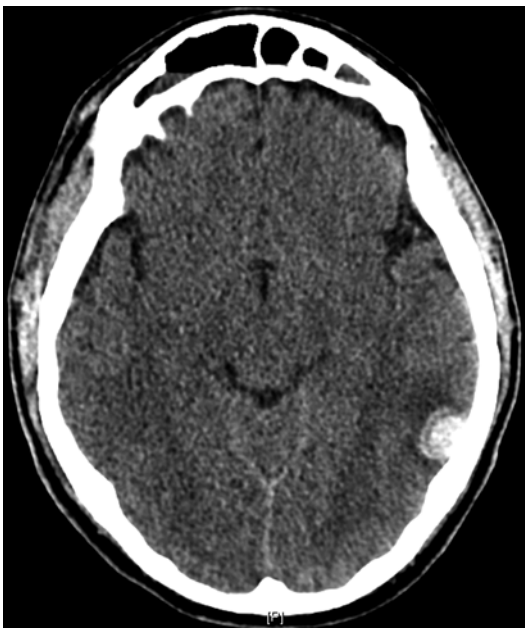

Fig. 4.1 Brain axial CT showing left temporal focal bleeding

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- *Neurological*: mental status was impaired. Pupil exam showed pupil asymmetry <1 mm and no dilatation or constriction; pupils were reactive to light and accommodation bilaterally. No alterations in deep tendon reflexes.
- *Cardiovascular*: regular rate and rhythm, S1 and S2 normal, no murmurs, rubs, or gallops, and no hepatojugular reflux.
- *Lungs*: no signs of respiratory effort on inspection. No wheezes, no crackles or stridor, and no bronchial or vesicular breath sounds on auscultation over the lung fields bilaterally.
- *Abdomen*: no lesions or signs of trauma. No pain or rebound on light and deep palpation, no organomegaly, and normal bowel sounds in all four quadrants.

Routine ECG was performed after the hospitalization, showing sinus rhythm, normal atrioventricular conduction, incomplete right bundle branch (RBBB), and no alterations in repolarization.

Laboratory Exams on Admission and Other Tests

Exams (including high-sensitivity cardiac troponin) were unremarkable except for a mild leukocytosis of 14,500/mm^3.

After 2 days in the hospital, the patient had a control CT of the brain that showed an increased size of the left temporal subarachnoid hemorrhage, with minimum brain compression and appearance of posterior subcortical-cortical bleeding. The patient was clinically stable, but ECG changed dramatically and showed sinus tachycardia; normal atrioventricular conduction; ST elevation in DII, DIII, aVF, V4, V5, and V6; and ST depression in aVR, aVL, V1, V2, and V3 (Fig. 4.2).

Fast echocardiography was performed, revealing no cardiac contractile abnormalities. Blood sample showed a rise in troponin level: 0.16 ng/ml.

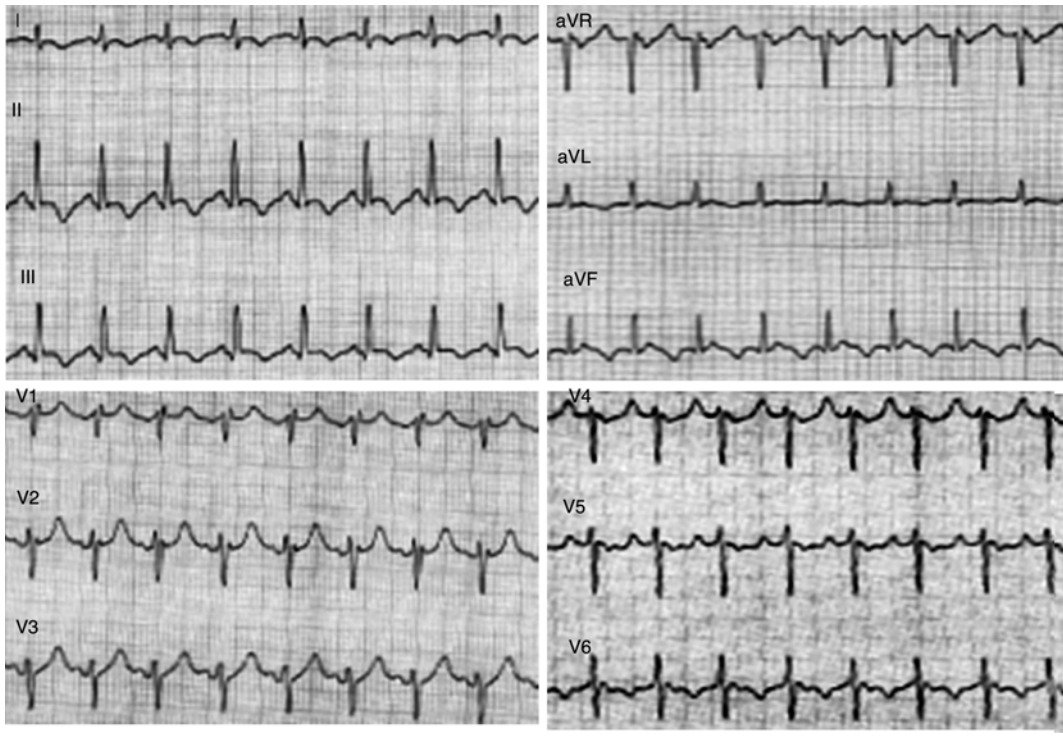

Fig. 4.2 Rest ECG showing incomplete right bundle block and normal repolarization

What Are the Possible Causes for ST Elevation?

- Acute ST-segment elevation myocardial infarction (STEMI)
- Coronary spasm
- Pericarditis
- Left ventricular aneurysm
- Brugada syndrome
- Left bundle branch block
- Left ventricular hypertrophy
- Early repolarization
- Drugs (e.g., cocaine, digoxin, quinidine, tricyclics, and many others)
- Electrolyte abnormalities (e.g., hyperkalemia)
- Neurogenic factors (e.g., stroke, hemorrhage, trauma, tumor, etc.)
- Metabolic factors (e.g., hypothermia, hypoglycemia, hyperventilation)

What Are the Possible Causes of Increased Troponin Level?

- Acute coronary syndrome
- High blood pressure in lung arteries (pulmonary hypertension)
- Blockage of a lung artery by a blood clot, fat, or tumor cells (pulmonary embolus)
- Congestive heart failure
- Myocarditis
- Prolonged exercise
- Trauma that injures the heart
- Weakening of the heart muscle (cardiomyopathy)
- Long-term kidney disease

In a setting of acute coronary syndrome and ST elevation, the patient has been promptly treated with metoprolol and acetylsalicylic acid intravenous and a percutaneous coronary angiography was urgently performed which showed just a non-critical stenosis (<30 %) of the right coronary artery. In the following 48 h, ST elevation gradually resolved, and waves were inverted in DII, DIII, aVF, V4, V5, and V6 (Fig. 4.3). A repeated head CT scan revealed a partial reduction of the encephalic bleeding. The patient was extubated and transferred to a spoke intensive care unit.

4.2 ECG Correlation and Intracranial Bleeding

ECG Abnormalities

In patients with acute cerebrovascular events, especially intracranial hemorrhage (ICH) and ischemic stroke, electrocardiographic repolarization abnormalities are frequently observed regardless of the presence or absence of previous cardiac diseases. Repolarization abnormalities warrant attention as they increase the vulnerable period during which an extrasystole is more likely to result in ventricular tachycardia or fibrillation. Repolarization abnormalities thus may explain the higher risk of arrhythmias and sudden death following acute neurological disorders [1–4].

The exact incidence of ECG abnormalities is still unclear but ranges from 50 to 98 % and includes QT prolongation, T wave inversion, prominent U wave, ST segment elevation or depression, peaked P waves, and transient pathological Q waves [1]. A typical, but not pathognomonic, pattern is characterized by "giant T waves," abnormally wide T waves of increased amplitude (mostly negative than positive); elevation or depression of the ST segment is less frequent [4].

However, as in our case, these ST changes are particularly important since they may simulate acute myocardial ischemia and therefore mislead physicians in the initial diagnosis. In fact, unconscious patients with ICH and ST segment elevation may be misdiagnosed with acute myocardial infarction and could be incorrectly treated with antiplatelet and anticoagulative drugs with devastating consequences. Also, in the setting of cerebrovascular events, changes in ST segment have

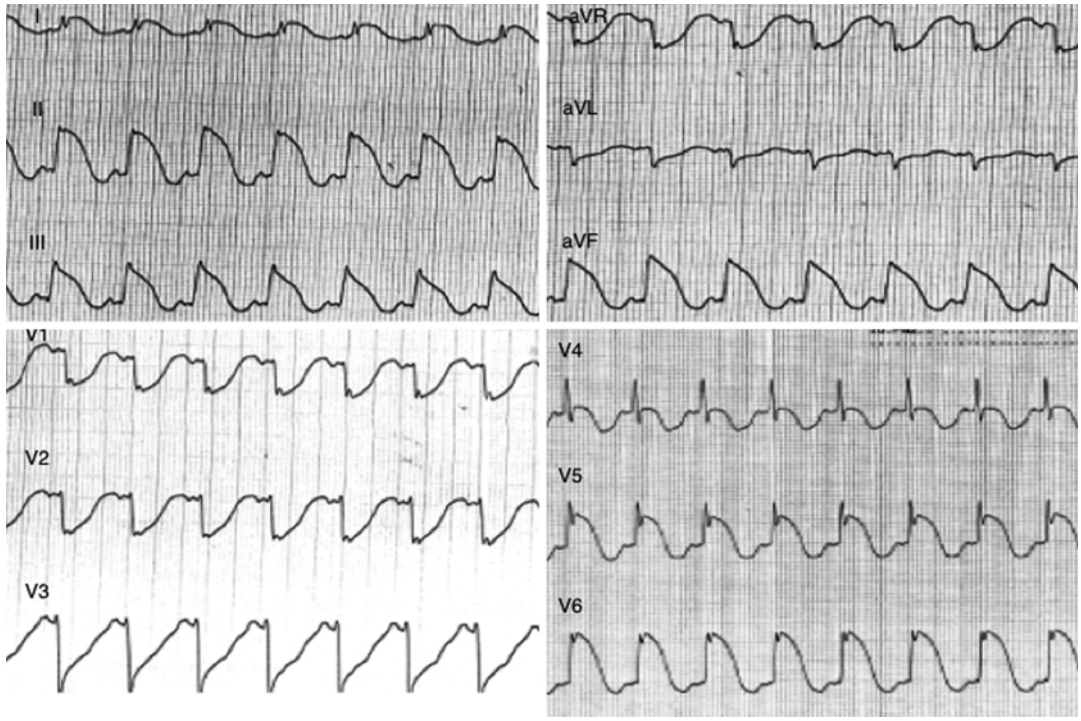

Fig. 4.3 Control ECG showing ST elevation in DII, DIII, aVF, V4, V5, and V6 and ST depression in aVR, aVL, V1, V2, and V3

generally the same polarity of T wave, and rarely, ST elevation is diffuse simulating acute pericarditis.

The ECG abnormalities usually reverse after the neurological clinical recovery.

Arrhythmias

In a prospective study, serious arrhythmia in the first 72 h after admission with acute stroke was detected in 25 % of 501 patients [5]. The risk for cardiac arrhythmia was highest during the first 24 h of care and declined with time during the first 3 days. In previous series, cardiac arrhythmias were detected in 6–25 % of patients after stroke [6, 7].

Atrial fibrillation is the most common cardiac arrhythmia accounting for nearly 60 % of all events of arrhythmia [5]. However, transient atrial fibrillation is more common among patients with ischemic stroke than with ICH; in this latter setting, atrial fibrillation occurs more frequently among patients who have developed brainstem or peri-insular hematomas [8].

As a consequence of repolarization abnormalities, especially with prolonged QT interval, malignant ventricular arrhythmias, including ventricular tachycardia, torsades de pointes, and ventricular fibrillation, may also be observed [9]. So, it is very important to check for QTc prolongation and to select drugs that do not prolong QT interval.

Mechanisms Leading to ECG Abnormalities

The mechanism through which cerebrovascular events lead to these ECG abnormalities is still unknown; however, autoptic findings reported that over 50 % of patients had cardiac muscle contraction band necrosis. The most plausible hypothesis suggests that ECG changes result as a consequent alteration of the autonomic nervous system control on cardiac electrophysiology that

results in catecholamine release at the terminal nerve at the cardiac myocyte [2, 3]. Increase of adrenergic stimulus increases myocardial oxygen demand, reduces coronary perfusion time, and also has a direct cardiotoxic effect with generalized spasm of the coronary arteries. Some evidence suggests also that sympathetic stroke-related activity causes calcium overload in ventricular myocytes and consequently arrhythmias. Furthermore, the type and location of the cerebrovascular event may correlate with the type of arrhythmia as each cerebral hemisphere seems to have a different influence on cardiac functions. Indeed, injury of the right insula may cause bradycardia and vasodepressor effects, while the left insular region may pose a higher risk for tachycardia and hypertension [10, 11].

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