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J-wave formation in patients with acute intracranial hypertension

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Abstract Various electrocardiographic changes are found in patients with increased intracranial pressure. The most common findings are sinus bradycardia, QT prolongation, ST-segment changes, and T- or U-wave abnormalities. The presence of J wave is reported rarely. We describe 3 patients with increased intracranial pressure caused by different cerebral pathologies accompanied by the dynamic formation of J waves in time.

Keywords: ECG; J-wave; Intracranial hypertension

Introduction

J wave is described as a positive deflection at the end of the QRS complex, that is, around the J point with an amplitude of at least 1 mm and duration of horizontal elevation of at least 10 milliseconds.[1] Prominent J wave is a common electrocardiographic finding in patients with hypothermia.[2] This wave has also been described in patients with hypercalcemia, early repolarization syndrome, acute myocardial ischemia, Prinzmetal angina, hypertrophy of the left ventricle, serious ventricular arrhythmia, cocaine or haloperidol overdosing, and subarachnoid hemorrhage irrespective of the presence of hypothermia.[2,3]

We describe 3 patients admitted with acute intracranial hypertension caused by different cerebral pathologies. None had hypothermia or a history of cardiovascular disease. At the time of electrocardiographic (ECG) recording, all patients had radiographic signs of intracranial hypertension. The blood biochemistry, including peripheral blood morphology, serum electrolytes, and arterial blood gases and acid-base balance, did not reveal any significant abnormalities. All 3 patients died of massive brain damage.

Case 1

A 48-year-old man underwent elective neurosurgery of an aneurysm of the left middle cerebral artery. The ECG recorded before surgery revealed sinus bradycardia of 52 beats per minute (bpm), a flat T-wave in lead aVL and QT equal to 420 milliseconds (Fig. 1, panel A). The patient's clinical state deteriorated rapidly within a few hours after the surgery. Computed tomography (CT) scanning showed an extensive stroke of the left cerebral lobe with accompanying edema. In the ECG recorded at the same time, there was a nodal rhythm of 43 bpm; Q waves in leads III and aVF with a width of 30 milliseconds and a depth of 1.5 mm; increased amplitude of the T waves in leads I, aVL, and V_1 through V6; negative T waves in lead III; and QT of 500 milliseconds. In addition, in leads III and aVF, there were J waves with an amplitude between 1.5 and 3 mm, which were descending to the isoelectric line or with a horizontal elevation lasting for up to 30 milliseconds (Fig. 1, panel B). Subtle deflections at the end of R waves were present in lead II. The formation of positive J waves was accompanied by the disappearance of S waves in the same leads. A negative J wave with a depth of 2 mm and a duration of 120 milliseconds was present in aVL.

Case 2

A female patient, aged 52 years, underwent neurosurgical removal of a tumor located close to the foramen

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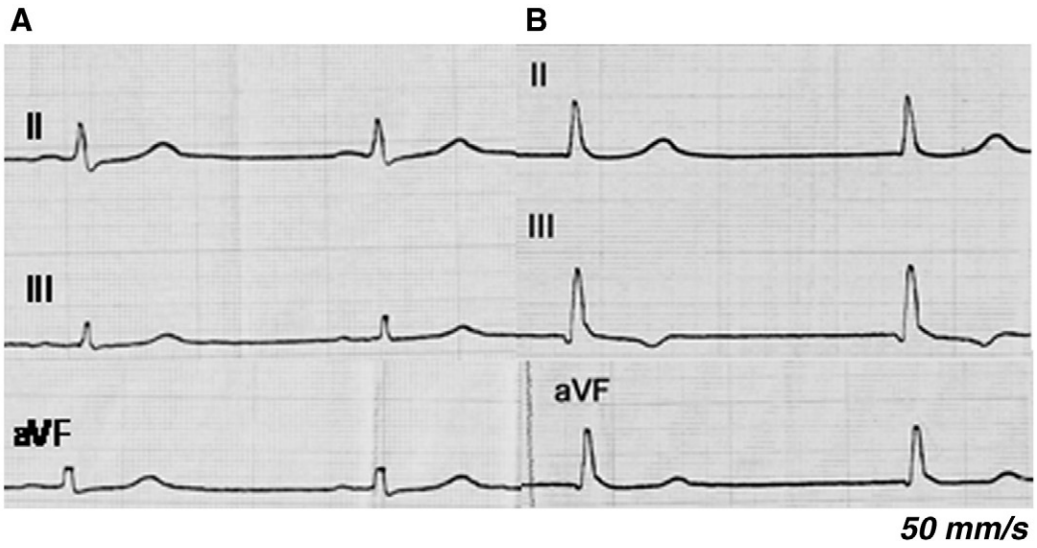

Fig. 1. Twelve-lead ECG recordings (paper speed, 50 mm/s) in the first patient who was admitted for elective neurosurgery of a ruptured aneurysm of the left middle cerebral artery. Panel A shows leads II, III, and aVF of the ECG before surgery, whereas panel B shows the same leads after the development of intracranial hypertension secondary to postsurgical stroke and edema. In panel B compared to panel A, note the disappearance of S waves in leads II, III, and aVF; presence of distinct J waves in leads III and aVF; and subtle deflections at the end of R waves in lead II.

magnum. In the ECG before the procedure (Fig. 2, panel A), there was a sinus rhythm of 66 bpm, a negative T wave in aVL, a biphasic T wave in V_2, and QTc of 400 milliseconds (corrected according to Bazett's[4] formula). A subarachnoid hemorrhage, which appeared during the surgery, was arrested by the vascular staunching. However, the hemorrhage recurred 12 hours later causing a further increase in intracranial pressure, worsening of the clinical status with acute respiratory failure. The ECG recorded at the time showed sinus tachycardia of 100 bpm, flattening of T waves in aVL and V_2, and prolongation of QTc to 450 milliseconds. J waves, with an amplitude up to 1 mm and horizontal elevation lasting for up to 20 milliseconds, were found in lead II; in other inferior leads, that is, III and aVF, there were only subtle deflections at the end of R waves (Fig. 2, panel B).

Case 3

A 33-year-old woman was hospitalized because of severe headache and persistent vomiting. The cerebral CT scan showed a subarachnoid hemorrhage, and angiography of the intracranial arteries revealed a ruptured aneurysm of the right internal carotid artery. A sinus rhythm of 63 bpm, QTc of 430 milliseconds, ST-segment depression of 0.1 mV in leads V_2 through V_5, and discrete deflections at the end of R waves in

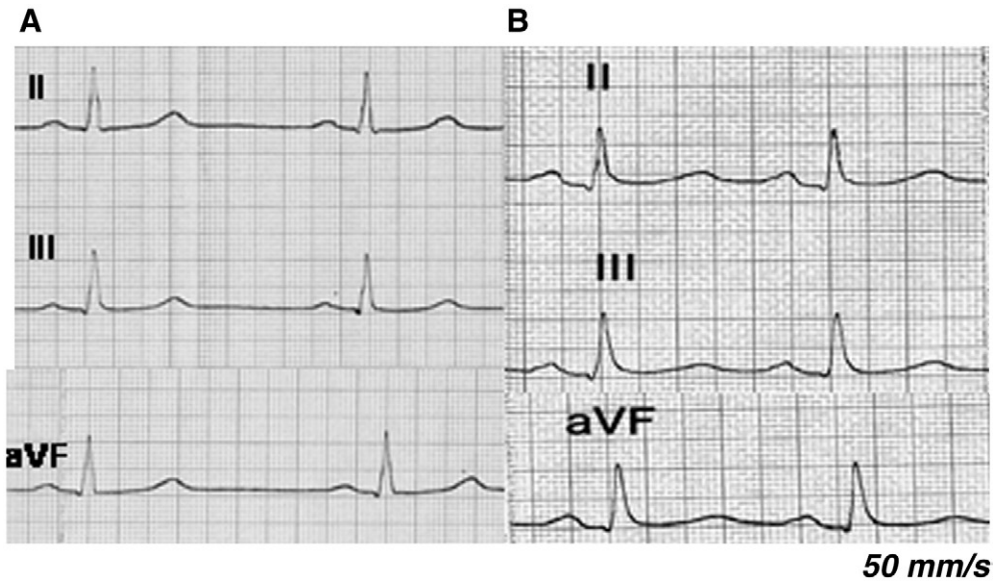

Fig. 2. Twelve-lead ECG recordings (paper speed, 50 mm/s) in the second patient who was referred for elective neurosurgery of a tumor localized near the foramen ovale. Panel A shows leads II, III, and aVF of the ECG before the surgery and panel B shows the same leads recorded when the postoperative period was complicated by a subarachnoid hemorrhage. In panel B, note the appearance of J waves in lead II and subtle deflections at the end of R waves in leads III and aVF.