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.

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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 V1 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
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 V2, and QTc of 400 milliseconds (corrected according to Bazett’s 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 V2, 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 V2 through V5, and discrete deflections at the end of R waves in
leads III and aVF (Fig. 3, panel A) were present on the admission. Although the patient underwent successive embolization of the aneurysm, there was recurrent bleeding into the subarachnoid space, which was finally stopped by neurosurgery. Repeated head CT scans showed severe brain edema. The ECG revealed a sinus rhythm of 60 bpm, QTc of 430 milliseconds, a further increase in ST-segment depression to 0.2 mV in V2 through V5, flat T waves in I and V5, biphasic T waves in V1 through V4, a negative T wave in aVL, and the amplitude of the S wave increased in V5 and V6. In addition, distinct J waves appeared in leads II and aVF with amplitude of 1 to 2 mm falling to the isoelectric line or with a horizontal elevation of duration 20 to 30 milliseconds (Fig. 3, panel B).

Discussion

The presence of ECG changes in the course of brain injury with accompanying intracranial hypertension was extensively described in the 1940s.5 The most frequent morphologic ECG findings in patients with subarachnoid hemorrhage are QT prolongation; ST-segment ischemic-like changes; large, inverted, or flattened T wave; or prominent U wave.6 Some of these classic changes, such as T-wave changes, ST-segment ischemic-like changes, and QT prolongation, were also found in our patients, but the presence of the J wave without coexisting hypothermia was another feature observed in our group. Examination of ECGs recorded during the cerebral injury that was developing revealed dynamic morphologic changes resulting in the formation of completely new J waves. Within a few hours of the onset of intracranial hypertension, the typical S waves changed into J waves in some leads. In addition, in some other leads, there appeared discrete deflections at the end of R waves, which were more subtle than typical J waves. In the first case, even negative J waves were present.

In the third case, the distinct J waves appeared a few days after onset of subarachnoid hemorrhage, when the intracranial pressure increased. The patient’s first ECG was recorded at the time of a minor increase in intracranial pressure, whereas the second ECG was taken when massive brain edema had developed. It is plausible that the increase in intracranial pressure was responsible for the “amplification” of J-wave expression in this subject.

The J wave is a result of higher density of $I_o$ channels in epicardium compared to endocardium, which leads to prominent $I_o$-mediated action potential notch in epicardium but not endocardium. The difference provides transmural gradient, which occurs during ventricular activation and is manifested in ECG as a J wave.7 Appearance of J wave in ECGs in patients with increased intracranial pressure may result from changes of autonomic tone observed under these condition.8

In all presented ECGs, the J waves were not accompanied by upward concaved ST segments, which would be characteristic for early repolarization syndrome.2 Similarly, in the second ECG of the first patient, presence of retrograde P waves in the course of nodal rhythm is doubtful. Retrograde P waves should be negative in leads II, III, and aVF because of retrograde atrial depolarization in nodal rhythm.

Conclusions

J waves of various magnitudes were present in the ECG of our patients after the development of intracranial hypertension. It is reasonable to believe that the expression of these J waves was related to the severity of intracranial hypertension, but further studies are needed to justify this hypothesis.

References

Apparent Osborne waves

Osborne waves. Figure A is a 12-lead electrocardiogram (ECG) taken from a 36-year-old male presenting to the emergency department with noncardiac chest pain, no medical history, and normal vital signs, including a temperature of 37°C. On initial inspection, there appear to be Osborne waves, seen best in the latter portion of lead II and in V4 (arrows). Upon further scrutiny of the preceeding P-P intervals, however, the cardiac rhythm is sinus bradycardia with slight slowing, leading to a junctional escape rhythm. The sinus P waves occur either before the QRS complexes with too short a PR interval to indicate sinus conduction (diamonds) during the QRS complex or shortly after the QRS complex. It is this finding of the P waves occurring within the QRS complexes but ending after them that is causing the apparent Osborne wave and is in fact an artifact. Osborne waves, or J waves, are usually seen in hypothermia, where its height reflects the degree of hypothermia. They are usually accompanied by bradycardia and a long QT interval, are best seen in the inferior and lateral leads, and are often seen with shivering artifact. They are not infrequently mistaken for ST elevation or intraventricular conduction delays; they are generally not recognized in computer readings.

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