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EXPANSIVE NEGATIVE T WAVES WITHOUT MYOCARDIAL DAMAGE MAY BE A PREMONITORY SIGN OF TRANSIENT CEREBRAL ISCHEMIA

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Expansive negative T waves were observed in a patient with chronic respiratory failure. The electrocardiogram (ECG) revealed expansive negative T waves in leads I, II, aVL, and V_{3-6}, and the ECG changes persisted for three months. Cardiac enzymes did not rise throughout the course, although these ECG findings suggested acute myocardial ischemia. Seven days after the onset of the ECG changes, a transient ischemic attack occurred in the vertebrobasilar territory. Although a relationship between these ECG changes and cerebral ischemia is unknown, such ECG changes in the absence of myocardial damage might be caused by ischemia of the vertebrobasilar territory, especially the hypothalamus, and might occur as a premonitory sign of transient ischemic attacks.

Key words: ECG / TIA

CASE REPORT

A 62-year-old Japanese woman without a past history of seizures, symptomatic cerebrovascular accident, or migrane, was admitted to our hospital because of severe dyspnea and leg edema on April 2, 1996. She had been treated for pulmonary tuberculosis from November 1995 at our outpatient clinic. From three weeks before admission, dyspnea had gradually increased. The electrocardiogram (ECG) obtained on admission showed normal sinus rhythm, deep S waves in leads I,II and III, small q waves in lead III, and counterclockwise rotation of chest leads (Fig 1). She had no abnormal findings on hematology and biochemistry tests. The platelet count was 167,000, the prothrombin time was 11.4sec (control value; 12.6sec), the partial thromboplastin time was 27.5sec (control value; 28.2sec), and the bleeding time was 1.5min. Echocardiography revealed normal left ventricular systolic function without valvular diseases. No evidence of left arterial thrombus was seen. On admission, the arterial partial pressure of oxygen was 36.7mmHg and the arterial partial pressure of carbon dioxide was 68.2mmHg. As the retention of carbon dioxide increased to 136.4mmHg with oxygen therapy, she was intubated, sedated, and ventilatory assistance was begun. On April 15, 1996, although she was asymptomatic, the ECG monitor showed giant negative T waves and the 12-lead ECG showed negative T waves in leads I,II,aVL ,and V_{3-6} (Fig 1). It might appear to negate that myocardial ischemia was present, since she had neither symptoms nor an abnormal increase of cardiac enzymes [serum creatine kinase, 36 (normal, 0-110 IU/L); aspartate aminotransferase, 19 (normal, 6-32 IU/L); and lactate dehydrogenase, 295 (normal, 200-400 IU/L)]throughout the course. Although the ECG changes persisted, the depth of the negative T waves gradually decreased. On the 22nd hospital day (April 23, 1996) (Fig 1), she suddenly lost consciousness and assumed decerebrate posture. On examination, the position of the eyes was normal and oculocephalic reflexes were positive bilaterally. Both pupils were miotic and sluggishly reactive to light. The deep tendon reflexes of the extremities were equally exaggerated and Babinski's sign was present on both sides. Thirty minutes later, these symptoms and signs completely disappeared. There were no significant changes of the ECG or arterial blood gases analysis compared with the previous day. Computed tomography scan of the brain showed no abnormal findings two days after the event. Coronal T_2-weighted magnetic resonance imaging (MRI) showed high signal intensity lesions in the right putamen and corpus callosum and showed a low signal intensity in the right putamen, suggesting small infarcts and a small old hemorrhage, respectively. The MRI of brainstem appeared slightly atrophic, however no abnormal intensity was observed. There were no abnormal signal in the hypothalamus and cingulate gyrus in this MRI examination. Magnetic resonance angiography revealed no stenotic lesions of the large vessels. Based on these findings, we concluded that a transient ischemic attack of the vertebrobasilar territory. Coronary angiography was performed. Left ventriculography revealed normal cardiac function and coronary arteriography showed no stenosis with ergometrine infusion. A routine electroencephalogram (EEG), performed about a month after the episode, revealed medium voltage 6-7 Hz background activity, without paroxysmal epileptic waves. Reactive to photic stimulation and hyperventilation were normal. The ECG returned to normal range on August 7 (Fig 1). Ventilation/perfusion lung scans were normal and paired serum viral titers for coxsackie, echo, adeno and influenza viruses showed no infection.

DISCUSSION

It has been known for sixty years that brain lesions may be accompanied by abnormalities of the electrocardiogram (ECG). These changes may be seen in patients with aneurysms, subarachnoid hemorrhage (SAH), brain tumors, and other neurologic disorders. Abnormalities of the central nervous system can produce marked changes of ventricular repolarization. There have been many reports on the SAH, which included QT interval prolongation, ST segment elevation or depression, changes of T and U wave morphology,

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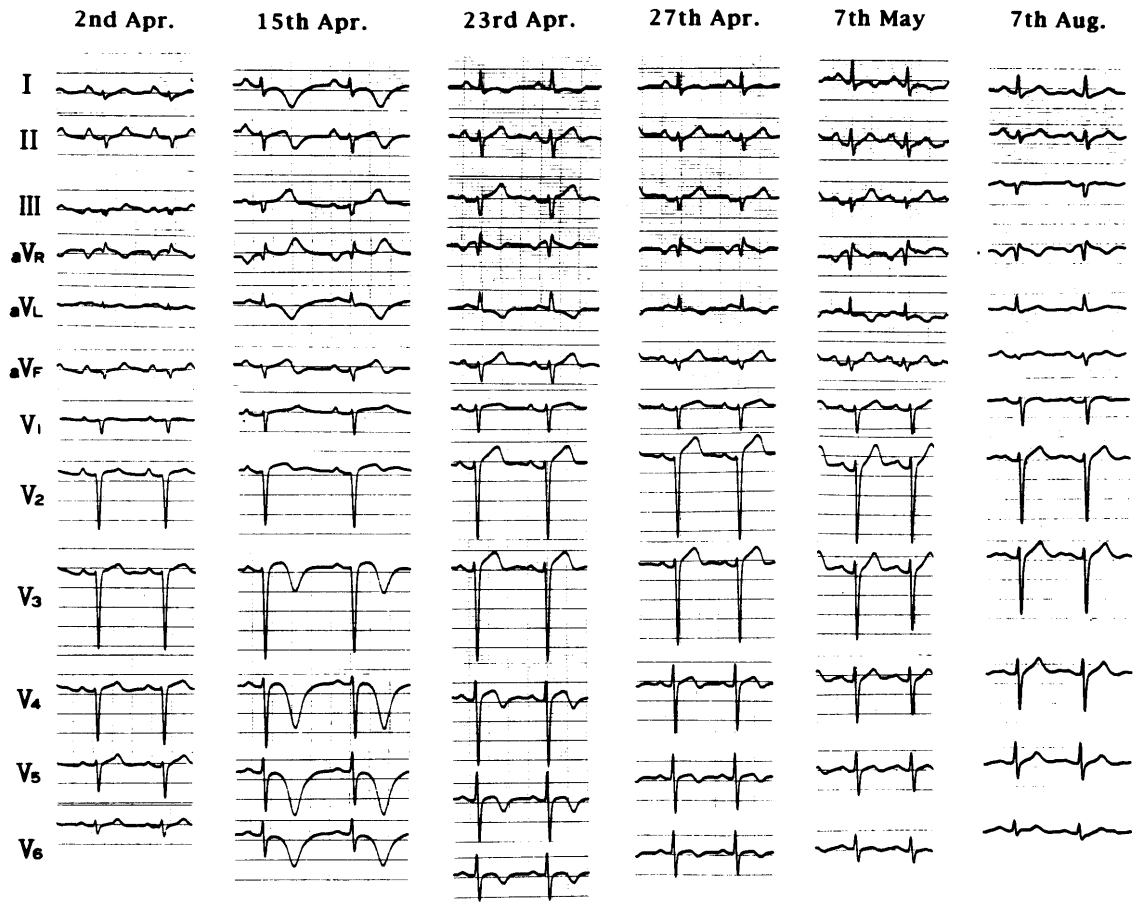

Fig. 1. The serial changes of the electrocardiogram.

and various arrhythmias (1-4). In our patient, ECG changes occurred at a time when she had neither symptoms nor neurological defects. After seven days the attack occurred. Serum enzymes, electrolytes, and blood gases were all within normal limits. Although there are a few reports on the relationship between ECG changes and cerebral infarction, it seems difficult to conclude that the ECG changes in our case resulted from the old lesions revealed by magnetic resonance imaging. In previous studies, ECG changes were concluded to be caused by neurally mediated myocardial damage. Myocardial damage has been demonstrated in patients with SAH by histologic examination at autopsy (5,6). Ischemic stimulation to the hypothalamus after SAH is thought to increase local norepinephrine release in the heart and thus cause myocardial damage by coronary vasoconstriction or by the direct toxic effect of norepinephrine (3,6,7). As no changes of cardiac enzymes were observed in our patient, neural factors unrelated to myocardial damage may also influence the ECG. On the 7th day after the onset of ECG changes, the attack occurred. Actually, we diagnosed her as a TIA with much difficulty, because her clinical symptoms were atypical as a TIA of vestibrobasilar territory. She had had no clinical history of neurological diseases including attacks of epilepsy and of losing consciousness. EEG examination could not point out epileptic waves, although the examination was performed only once and about a month after the attack. However, MRI showed old vascular lesions, which might suggest brain ischemia in our patient. The arteries to the hypothalamus are derived from the circle of Willis, including the posterior cerebral arteries. Davis *et al.* (8) reported that hypothalamic stimulation may cause ECG changes without associated myocardial damage, whereas elevated catecholamine levels may result in myocardial damage. It may be possible that our patient was like their case, since brain ischemia of vestibrobasilar territory was suspected to be responsible for this attack. We proposed that the ECG changes could be a premonitory sign of the brain ischemia, though it is unknown how these lesions could produce such changes.

The ECG changes may also be due to humoral factors or to a direct effect on repolarization, so further studies need to be done.

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