CASE REPORTS

Abnormal Q Wave, ST-Segment Elevation, T-Wave Inversion, and Widespread Focal Myocytolysis Associated With Subarachnoid Hemorrhage

Hironosuke Sakamoto, M.D.1, Hiroshi Nishimura, M.D.2, Kouji Imataka, M.D.
Keiko Ieki, M.D., Toshinobu Horie, M.D.3
and Jun Fujii, M.D.

A 74-year-old Japanese woman with subarachnoid hemorrhage was admitted to our hospital. During her hospitalization, serial electrocardiograms showed the combination of abnormal Q waves, ST-segment elevation, and T-wave inversion, which strongly suggested acute myocardial infarction. However, postmortem examination revealed widespread focal myocytolysis of the myocardium which was unrelated to vascular distribution.

(Ipn Circ J 1996; 60: 254–257)

SUBARACHNOID hemorrhage is often accompanied by electrocardiographic abnormalities, including QT interval prolongation, ST-segment elevation or depression, changes in T- and U-wave morphology, and various arrhythmias1–5 However, the combination of abnormal Q waves, ST-segment elevation, and T-wave inversion, which is consistent with a diagnosis of acute myocardial infarction, is extremely rare? We describe a patient with subarachnoid hemorrhage in whom serial electrocardiograms showed these 3 abnormalities, which strongly suggested acute myocardial infarction. However, postmortem examination revealed widespread focal myocytolysis of the myocardium.

Key words:
Electrocardiography
Acute myocardial infarction
Catecholamine

CASE REPORT

A 74-year-old Japanese woman who had experienced sudden severe headaches and vomiting 3 days earlier was admitted to our hospital. On admission, her blood pressure was 210/82 mmHg, with a regular pulse rate of 56 per minute. She had a left-sided hemiplegia, corresponding to a low-density area in the right lentiform nucleus on a computed tomographic scan (Fig 1A). She lost consciousness on hospital day 3. A computed tomographic scan showed blood in the right and left lateral ventricles from an unspecified source (Fig 1B). A follow-up computed tomographic scan performed on day 4 revealed a massive subarachnoid hemorrhage located primarily in the anterior interhemispheric fissure and the right Sylvian fissure (Fig 1C). She died on hospital day 9.

An electrocardiogram obtained on admission showed normal sinus rhythm and high-

(Received February 18, 1995; accepted August 11, 1995)
The Institute for Adult Diseases, Asahi Life Foundation, 1-9-14, Nishishinjuku, Shinjuku-ku, Tokyo; 1Second Department of Internal Medicine, Gunma University School of Medicine, Maebashi; 2Third Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Tokyo; and 3Saitama Ohara Cardiovascular Center, Saitama; Japan
Mailing address: Hironosuke Sakamoto, M.D., Second Department of Internal Medicine, Gunma University School of Medicine, 39-22, Showa-machi 3-chome, Maebashi, Gunma 371, Japan

254 Japanese Circulation Journal Vol.60, April 1996
Fig 1. Computed tomographic scans of the brain obtained on admission (A), day 3 (onset of subarachnoid hemorrhage) (B) and day 4 (C).

electric QRS complexes (Fig 2A). An electrocardiogram obtained on day 3, when she became unconscious, showed sinus tachycardia, reduction of R-wave amplitudes in lead V3, elevated ST segments with large, upright T waves in leads I, II, aVF and V3 to V6, and prolonged QT intervals (Fig 2B). Abnormal Q waves in lead V3 and inverted T waves in leads I, II, III, aVL, aVF and V3 to V6 developed on day 4 (Fig 2C). Inversion of T waves increased on day 5 (Fig 2D). These electrocardiographic changes strongly suggested the presence of an acute transmural anterolateral, apical, and inferior myocardial infarction.

Enzyme studies on admission, day 3, day 4, and day 7 gave the following results: serum creatine kinase, 48, 23, 117, and 48 IU/L (normal, 13–118 IU/L); aspartate aminotransferase, 13, 8, 44, and 19 IU/L (normal, 6–33 IU/L); and lactic dehydrogenase, 192, 175, 325, and 434 IU/L (normal, 148–250 IU/L), respectively. The serum level of cardiac myosin light chain I on day 8 was 6.0 ng/ml (normal, <2.5 ng/ml).

Postmortem examination confirmed a ruptured aneurysm at the junction of the left anterior cerebral and anterior communicating arteries and an infarction in the right cerebral hemisphere. The heart weighed 335 g. The left ventricle was slightly hypertrophied. There was no macroscopic evidence of significant coronary artery stenosis or myocardial infarction. Histologic examination revealed multiple foci of myocytolysis with loss of myocardial cells, collapse of the
Fig 3. Photomicrograph of the left ventricular myocardium showing multiple foci of myocytolysis of the myocardium (hematoxylin-eosin, × 100).

supporting stroma, and a scant mononuclear cellular infiltrate frequently containing phagocyted lipofuscin pigment throughout the total circumference of the left ventricle (Fig 3). There were no polymorphonuclear leukocytes in the cellular infiltrate. The foci of myocytolysis were surrounded by normal myocardial cells. Although the abnormal Q wave in lead V3 suggested that the greatest intensity of myocardial damage would be in the apical area of the left ventricle, the distribution of the foci of myocytolysis was not related to the vascular distribution. The intramural location of these foci was evenly distributed without an epicardial-endocardial gradient.

DISCUSSION

This patient is the first reported case of subarachnoid hemorrhage to show a clinicopathologic correlation between the classic electrocardiographic pattern of acute transmural myocardial infarction, characterized by abnormal Q waves, ST-segment elevation, and T-wave inversion, and widespread focal myocytolysis of the myocardium. Although these electrocardiographic findings were documented in one previous case with subarachnoid hemorrhage, postmortem examination in that case showed a classic myocardial infarction in the upper anterior portion of the interventricular septum9. Electrocardiographic abnormalities in association with focal myocytolysis were detected in 2 previous cases with subarachnoid hemorrhage, one of which had abnormal Q waves and ST-segment elevation7 while the other showed only abnormal Q waves8.

The clinicopathologic findings in the present case suggest that the development of an infarction-like electrocardiographic pattern in patients with subarachnoid hemorrhage may occur as a result of focal myocytolysis. Connor9 found an 8% incidence of focal myocytolysis in the hearts of patients dying of intracranial disease. Focal myocytolysis must be distinguished from lesions most frequently associated with myocardial infarction. The characteristic features of myocytolysis are loss of sarcoplasm from a small area of muscle, with retention of the sarclemma, stroma, muscle nuclei, and lipofuscin granules7. These degenerating myocardial cells are engulfed by a few mononuclear cells. The polymorphonuclear leukocytic
Electrocardiogram and Subarachnoid Hemorrhage

infiltration characteristic of myocardial infarction is not seen in this condition. Most of the foci of myocytolysis are found in the left, rather than the right, ventricle, but the distribution of damage in the muscle is random. There is no predilection for the subendocardial zone, which is typical in myocardial infarction. The foci of myocytolysis are surrounded by normal myocardial cells, and the total volume of damaged muscle appears to be small. This may account for the small rise in serum cardiac enzymes and the inability to macroscopically detect the injury despite electrocardiographic changes which suggest extensive myocardial infarction.

Myocarditis can provoke ST-segment elevation or depression, T-wave elevation, flattening, or inversion, and, occasionally, abnormal Q waves. Atrial and ventricular arrhythmias and atrioventricular and intraventricular conduction defects may occur. Patients with myocarditis demonstrate a wide spectrum of gross and histological changes. Grossly, the heart in acute cases is flabby, with focal hemorrhages; in chronic cases, the heart is enlarged and hypertrophied. The histological hallmark of myocarditis is an interstitial infiltration of inflammatory cells with necrosis and degeneration of adjacent myocardial cells. The inflammatory infiltrate is composed of polymorphonuclear leukocytes, lymphocytes, macrophages, plasma cells, eosinophils, and giant cells.

The exact mechanisms by which a classic electrocardiographic pattern of acute myocardial infarction and focal myocytolysis of the myocardium occur in patients with subarachnoid hemorrhage remain unclear. Most authors consider that this phenomenon is due to sympathetic overstimulation. Subarachnoid hemorrhage may stimulate sympathetic centers in the hypothalamus, leading to either myocardial or systemic release of catecholamines. The released catecholamines could damage myocardial cells either by inducing constriction of the myocardial microcirculation, thus leading to focal ischemia, or by a direct toxic effect. Tachycardia during the episode of electrocardiographic changes in our case may support this notion of sympathetic overstimulation.

REFERENCES


Japanese Circulation Journal Vol.60, April 1996