Neurogenic Pulmonary Edema and Large Negative T Waves Associated with Subarachnoid Hemorrhage

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Abstract

We describe a 72-year-old woman with hypertension who developed acute neurogenic pulmonary edema and giant negative T waves on electrocardiography (ECG) due to subarachnoid hemorrhage. The patient was alert and complained of precordial chest discomfort, dyspnea and shoulder stiffness. Echocardiography demonstrated normal left ventricle contraction with hypertrophy. Computed tomography (CT) and subsequent cerebral angiography revealed subarachnoid hemorrhage and saccular aneurysm at the anterior communicating artery. It is important to consider the possibility of subarachnoid hemorrhage when a patient shows pulmonary edema and ECG abnormalities even without typical clinical signs of subarachnoid hemorrhage.

Key words: neurologic insults, neurologic pulmonary edema, ECG abnormalities, computed tomography

Introduction

Pulmonary edema following a neurologic insult is often called “neurogenic pulmonary edema” (1). The mechanisms of its production have been thought to be noncardiac, as opposed to conventional pulmonary edema caused by left heart failure. However, recent investigations indicate that a neurologic insult also induces cardiac complications including electrocardiographic abnormalities, cardiac arrhythmias, and myocardial damage (2-4). Here we describe a 72-year-old woman who developed acute neurogenic pulmonary edema and giant negative T waves on electrocardiography (ECG) in association with a subarachnoid hemorrhage.

Case Report

A 71-year-old woman who had hypertension was referred to our hospital with precordial chest discomfort, dyspnea and shoulder stiffness. On admission, she was alert, her pupils were isocoric with prompt reaction to light and full-range of ocular movement. Motor and sensory functions were normal. Her blood pressure was 180/72 mmHg, heart rate was 88/min, and no heart murmur was audible. Laboratory analysis showed a white blood cell count of 13,200/μl, C-reactive protein (CRP) of 4.3 mg/dl, aspartate aminotransferase (AST) of 22 mU/ml, alanine aminotransferase (ALT) of 11 mU/ml, lactic dehydrogenase (LDH) of 513 mU/ml, and creatine kinase (CK) of 79

Figure 1. Chest X-ray shows diffuse pulmonary infiltrates in the lung indicating the presence of pulmonary edema.
mU/ml. An arterial blood gas analysis showed pH of 7.32, PO$_2$ of 77 mmHg, PCO$_2$ of 50 mmHg, HCO$_3^-$ of 26 mmol/l, and O$_2$ saturation of 93%. Chest X-ray revealed diffuse pulmonary infiltrates in the lung indicating the presence of pulmonary edema (Fig. 1). Electrocardiography showed negative P waves and ST segment depression with deeply inverted T waves (giant negative T waves) in I, II, III, V$_1$, V$_2$, and V$_3$-V$_6$ (Fig. 2). Echocardiography demonstrated normal left ventricle contraction with hypertrophy. Because the ECG in patients with neurologic insults including subarachnoid hemorrhage frequently shows ST segment and T wave changes, we performed computed tomography (CT). Brain CT (Fig. 3) and subsequent cerebral angiography revealed the presence of subarachnoid hemorrhage and a saccular aneurysm at the anterior communicating artery. The patient underwent a successful urgent operation for subarachnoid hemorrhage. The pulmonary edema in this patient disappeared after the operation.

**Discussion**

The association between pulmonary edema called “neurogenic pulmonary edema” and central nervous system damage such as head trauma (5), brain tumor (6), cerebral hemorrhage (7), increased intracranial pressure (8), and ruptured aneurysm (9) has been reported. However, the pathogenesis of neurogenic pulmonary edema has been unclear. Several experimental investigations suggest that a sudden increase in intracranial pressure or hypothalamic lesions may elicit a massive sympathetic discharge, with redistribution of blood to the pulmonary circulation resulting in high pulmonary capillary pressures and

![Figure 2. ECG shows ST segment depressions and deep symmetric T wave inversions (giant negative T waves) in leads I, II, III, V$_1$, V$_2$, V$_3$-V$_6$.](image)
increased permeability (10, 11). In fact, experimental studies in animals (12) demonstrated that a large amount of norepinephrine is released after neurologic insult and it may play a substantial role in neurogenic pulmonary edema because of its ability to increase the post capillary pulmonary vascular resistance (1).

Several previous reports have demonstrated that patients with neurogenic pulmonary edema after subarachnoid hemorrhage tend to have a more severe illness than those with subarachnoid hemorrhage alone. Yabumoto et al. (9) described that although there are no remarkable differences in age, sex, site of aneurysm, and mortality between the patients with subarachnoid hemorrhage with or without neurogenic pulmonary edema, the prognosis is still grave. In their report the situation was not unlike that of subarachnoid hemorrhage alone; of a total 12 patients, 3 died, 1 deteriorated, and 2 became hemiparetic because of vasospasm. Mayer et al. (2) also reported that cerebral infarction due to vasospasm occurred in 4 of 5 patients who had neurogenic pulmonary edema after subarachnoid hemorrhage and it was the immediate cause of death in the 2 patients who died. Furthermore, patients with pulmonary edema have been reported to have a tendency for more severe conditions of subarachnoid hemorrhage (2, 13). Interestingly, the patient described here with neurogenic pulmonary edema was alert and did not have the typical symptoms of subarachnoid hemorrhage. In addition, because the patient showed giant negative T waves on ECG which suggested myocardial ischemia, it was difficult to diagnose subarachnoid hemorrhage.

Abnormalities of ECG in patients with subarachnoid hemorrhage have also been described (2-4). However, in some cases, these ECG changes can mimic those observed in acute myocardial ischemia. Thus, we should consider the possibility of neurologic insult when we see pulmonary edema and ECG abnormalities even when the typical symptoms of subarachnoid hemorrhage are not present, and perform a CT examination of the head.

References