Massive Hemoptysis after Generalized Tonic Clonic Seizure Requiring Mechanical Ventilation

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A 38-year-old woman presented with massive hemoptysis (> 200 mL/24 hours) occurring abruptly after generalized tonic clonic seizure. She experienced similar episodes of hemoptysis on three later occasions. Although the coexistence of hemoptysis and seizure has been reported, albeit rarely, as a clinical manifestation of postictal neurogenic pulmonary edema, massive hemoptysis after seizure is an extremely rare event with no recurrent cases of such episodes having ever been reported. The coexistence of hemoptysis and seizure increases the difficulty in diagnosis for the clinician. We describe the differential diagnosis among the diseases capable of causing seizure and hemoptysis.

Key Words: Seizure, hemoptysis

INTRODUCTION

Massive hemoptysis is a common yet serious clinical condition which is typically caused by pulmonary tuberculosis, bronchiectasis and aspergillosis in Korea. Its occurrence after seizure is a rare clinical manifestation gave diagnostic difficulties to the clinician and is rarely reported in the clinical setting of neurogenic pulmonary edema (NPE).¹⁻⁵ We report a case presenting recurrent episodes of hemoptysis, consisting of two episodes of massive hemoptysis followed by two later episodes of non-massive hemoptysis after generalized tonic clonic seizure, and discuss the differential diagnosis of diffuse alveolar hemorrhage.

CASE REPORT

In March 17, 1998, a 38-year-old woman visited the emergency room presenting with massive hemoptysis (> 200 mL/24 hours) and dyspnea which occurred abruptly after generalized tonic clonic seizure. She denied smoking and drinking alcohol. She had no history of any other medical illnesses including heart diseases, tuberculosis or connective tissue disease. Because she was a shaman, her compliance to prescribed medication was poor. She had experienced a series of complex partial seizures 3 years previously. Those seizures had occurred about once a month with intermittent irregular antiepileptic medications. Repeated EEG recording showed intermittent left temporal spikes and brain MRI revealed a small lesion of a few millimeters in diameter/length, of unknown nature, in the left basal temporal cortex.

For 15 days prior to visiting the emergency room, she had ceased to take the valproic acid, which had been prescribed since her first partial seizure 3 years previously. Her typical seizure comprised a generalized heat flushing sensation followed by staring, lip smacking and loss of consciousness with secondary generalization. The clinical seizure diagnosis was complex partial seizure with secondary generalization, while the epilepsy diagnosis was left temporal lobe epilepsy. Her blood pressure was 140/80 mmHg, pulse rate 100 beats/min, respiratory rate 32/min and body temperature 37.2°C. In auscultation, coarse crackles were heard over the whole lung field and her heart beat was regular without murmur. Arterial blood gas study on room air revealed pH 7.38, PaCO₂ 38 mmHg, PaO₂ 88 mmHg, HCO₃⁻ 22 mEq/L and SaO₂ 76%. Labora-

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tory study confirmed whole blood cell count 6,700/mm³ (neutrophil, 79.7%), hemoglobin 11.5 g/dl, platelet count 256,000/mm³, aspartate aminotransferase 161U/L, alanine aminotransferase 121U/L, blood urea nitrogen 16.0mg/dl, creatinine 0.8mg/dl, prothrombin time 13.5 seconds (normal range, 11-15 sec.), activated partial prothrombin time 33 seconds (control, 29-44 sec.), creatinine kinase 150IU/L (normal range, 30-165 IU/L) and MB fraction 1 IU/L. Urine analysis was normal. Sputum gram stain and culture for bacteria revealed a few polymorphonuclear neutrophil leukocytes and α-streptococcus as the predominant organism. Direct sputum examination produced negative results for acid-fast organisms and fungi. Chest X-ray showed diffuse infiltration on both lung fields. Her EKG showed normal findings. Echocardiogram revealed elevated right ventricular systolic pressure (RVSP): 41mmHg with no regional wall motion abnormalities and normal left ventricular ejection fraction. The patient was intubated and mechanically ventilated with F̌O₂ 100%. After 6 hours of mechanical ventilation, arterial blood gas study was as follows: pH 7.28, PaCO₂ 44 mmHg, PaO₂ 191 mmHg, HCO₃⁻ 20 mEq/L and SaO₂ 99%. On fiberoptic bronchoscopy, scanty quantities of fresh blood were seen around both main and lobar bronchi, and no endobronchial lesion was observed. Hemosiderin-laden macrophages were not seen on the bronchial washing specimen. The results of bronchial washing and sputum examinations were negative for acid-fast organisms, fungi and bacteria. Studies for rheumatoid factor, ds anti-DNA, ANA and ANCA showed negative results. Valpric acid and empirical antibiotics were administered intravenously. No other medications, including corticosteroid or diuretics, were administered. The blood level of valpric acid was 15.6μg/mL (therapeutic range: 50-100μg/mL). On the 2nd day after admission, chest CT scan revealed diffuse, ground glass opacities on both lung fields (Fig. 1). On the 3rd day, the patient was weaned from the mechanical ventilator. The chest radiographic finding was significantly improved (Fig. 2). Repeated echocardiogram showed normal RVSP. She was discharged without any complaints of dyspnea or hemoptysis on the 7th day.

Similar clinical manifestations, including dyspnea and hemoptysis within several minutes of each other followed by generalized tonic clonic seizure, were repeated on three more occasions, each time several days after the patient again
discontinued taking the anticonvulsive medica-
tion; massive hemoptysis in Aug. 1998, and
non-massive hemoptysis in Mar. 1999 and Aug.
2001. Within one to two days of admission, the
diffuse infiltrations on chest X-ray were cleared
and the initially increased RVSPr were returned
to normal on serial echocardiography, merely
with the recommencement of anticonvulsive
medication, in combination with mechanical
ventilation on one occasion and oxygen via mask
on the other two occasions. In Aug. 2001, re-
peled brain MRI results were normal and no
evidence of endobronchial lesions was seen on
fiberoptic bronchoscopy. Studies for acid-fast
organisms, fungi, bacteria and cytology were all
negative.

DISCUSSION

Massive hemoptysis is a serious condition
which requires prompt decision making from the
clinician. Its etiology has been reported as pul-
monary tuberculosis, bronchiectasis, lung abscess,
mycetoma, lung cancer, and broncholithiasis.6
Because bilateral, diffuse, alveolar infiltration was
observed on chest CT scan, diffuse alveolar ho-
morhage syndrome and pulmonary edema were
initially included in the differential diagnosis. In
the presenting case, the absence of anemia and
hemosiderin-laden macrophages on the bronchial
washing specimen, the rapid improvement of
clinical manifestation, and the relatively longer
interval between episodes of hemoptysis ex-
cluded the clinical possibility of diffuse alveolar
hemorrhage syndrome, and also cardiogenic
pulmonary edema was excluded with the
findings of transthoracic echocardiogram and the
level of cardiac enzyme. We could further excludethe possible presence of isolated pul-
monary capillaritis as a cause of diffuse alveolar
hemorrhage because of the recurrent episodes
of massive hemoptysis after the seizures, and the
rapid improvement demonstrated on chest
radiography without the use of immunosup-
pressive agents in the presenting case.7 Diffuse
alveolar hemorrhage has been rarely reported in
cases of malignancy or bronchial artery rupture,
and both possibilities were easily excluded in the
presenting case.8,13

Considering the temporal relationship of the
hemoptysis to the seizure, the postictal NPE may
have been the possible hemoptysis cause. Since
the first reported case of postictal NPE by
Shanahan in 1908, only 43 cases have been re-
ported in the world, none of which involved
massive hemoptysis.1,5,14 The fact that all of these
postictal NPE cases were reported with small
amounts of hemoptysis after seizure and fine
crackles on auscultation of the chest during the
course of the disease may exclude the possibility
of hemoptysis caused by postictal NPE in the
presenting case which showed massive hemop-
tysis on two occasions and coarse crackles on
both lung fields. Serial echocardiogram at the
time of emergency room admission was per-
formed on the presenting case. The dynamic
clearing of pulmonary infiltration observed on
serial chest radiography was associated with
rapid normalization of the right ventricular
systolic pressure on all four occasions of admis-
sion. The left ventricular function on echocar-
diogram and the level of cardiac enzyme were
both normal. These findings suggest that pul-
monary vascular hypertension after seizure,
rather than increased pulmonary capillary per-
meability, may cause the sudden rupture of the
pulmonary capillary and directly induce diffuse
alveolar hemorrhage and massive hemop-
tysis.2,15–19 Pacht et al reported the first case of
postictal NPE with gross hemoptysis.3 Although
they did not clarify the level/severity/ degree of
the sudden hemoptysis, gross hemoptysis may be
caused solely by the sudden rupture of the
pulmonary capillary, rather than by postictal
NPE.

After excluding the possibility of postictal NPE,
the following three cases with hemoptysis after
seizure have been reported: one with necrotizing
pneumonia caused by aspiration during seizure,
another with lymphangioleiomyomatosis with
tuberculous sclerosis, and a third with cystic fi-
brosis.20–22

We report the first case of recurrent episodes of
massive hemoptysis and diffuse alveolar hemor-
rhage after seizure, in a patient with no under-
lying lung disease.

Yonsei Med J Vol. 43, No. 4, 2002
REFERENCES