Case Report

Neurogenic Pulmonary Oedema Misdiagnosed as Acute Myocardial Infarction in a Comatose Patient

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Abstract

Introduction: We report a case of neurogenic pulmonary oedema (NPO) following massive left cerebral infarct, which was initially misdiagnosed as acute myocardial infarction (AMI). Clinical Picture: This 52-year-old man presented with acute loss of consciousness with normal brain computed tomography (CT). He was treated as non-ST-elevation AMI complicated with pulmonary oedema based on findings of chest radiograph (bilateral pulmonary oedema), electrocardiogram (marked ST-T changes in leads V3 to V6), and cardiac enzymes [elevated creatinine kinase (CK) and CK-MB]. However, coronary angiogram and serial cardiac enzymes were inconclusive. Anisocoria developed after admission and a repeat brain CT was evident for large left cerebral infarct. Treatment: Decompressive craniectomy was carried out. Outcome: Mortality. Conclusions: The diagnosis of NPO can be challenging when it occurs without abnormal findings on preliminary brain CT. It can be mistaken for cardiogenic pulmonary oedema secondary to AMI.

Ann Acad Med Singapore 2007;36:684-6

Key words: Cerebral infarction, Neurogenic pulmonary oedema, Stroke

Introduction

Neurogenic pulmonary oedema (NPO) is a well recognised complication of subarachnoid haemorrhage (SAH) and severe traumatic brain injuries (TBI).[1] The incidence of NPO was reported to be 6% in a series of 457 patients with SAH.[2] However, the diagnosis of NPO can be challenging when it occurs without abnormal findings on preliminary brain computed tomography (CT). It can be mistaken for acute heart failure, pneumonia, or acute respiratory distress syndrome (ARDS). We report a case of NPO following massive left cerebral infarct which was initially misdiagnosed as cardiogenic pulmonary oedema due to acute myocardial infarction (AMI).

Case Report

A 52-year-old man was brought to our emergency department (ED) because of acute loss of consciousness for about 30 minutes. His family reported a brief episode of breathlessness just before he collapsed. No chest pain and no focal neurologic symptoms were noted before the event. He had a past history of hypertension under regular medical treatment. On ED arrival, marked lip cyanosis was found and the patient was in deep coma with a Glasgow Coma Scale score of $E_1V_1M_1$. His vital signs were blood pressure (BP) 228/130 mm Hg, pulse rate 135 beats/min, respiratory rate 8 breaths/min and temperature 36.2ºC (97.2ºF). He was immediately intubated under rapid sequence induction and connected to a mechanical ventilator. Arterial blood gases (ABGs) obtained before intubation showed pH 6.94, P_aCO_2 77 mm Hg, P_aO_2 58 mm Hg (80% FiO_2), and HCO_3 16.8 mmol/L. Physical examinations revealed isocoric pupils (3 mm/3 mm) with prompt light reflexes. His neck veins were not distended. Auscultation showed bilateral moist rales of the lungs. Heart sounds were normal and there were no abdominal bruits. Extremities were flaccid, cyanotic, cold, and sweaty, but there were no pitting oedema.

A chest radiograph obtained after intubation showed bilateral pulmonary oedema (Fig. 1). Electrocardiogram (ECG) revealed left ventricular hypertrophy (LVH) and

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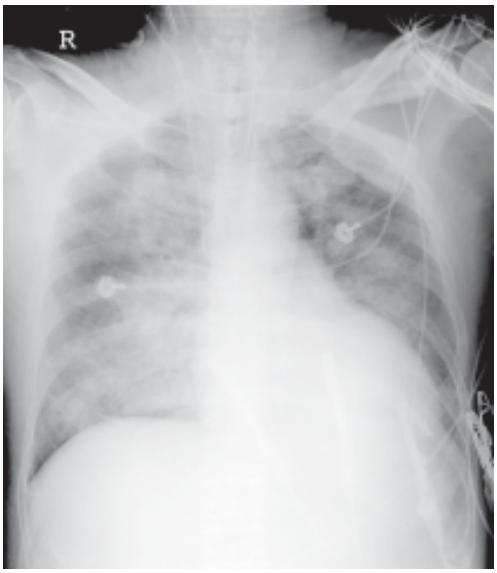

Fig. 1. Chest radiograph on presentation (with endotracheal tube, CVP catheter, and ECG leads in place).

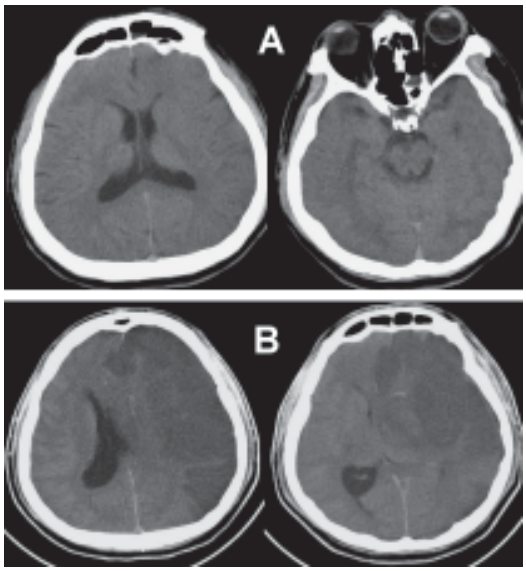

Fig. 3. The patient's brain computed tomography on presentation (A) and 37 hours after admission (B).

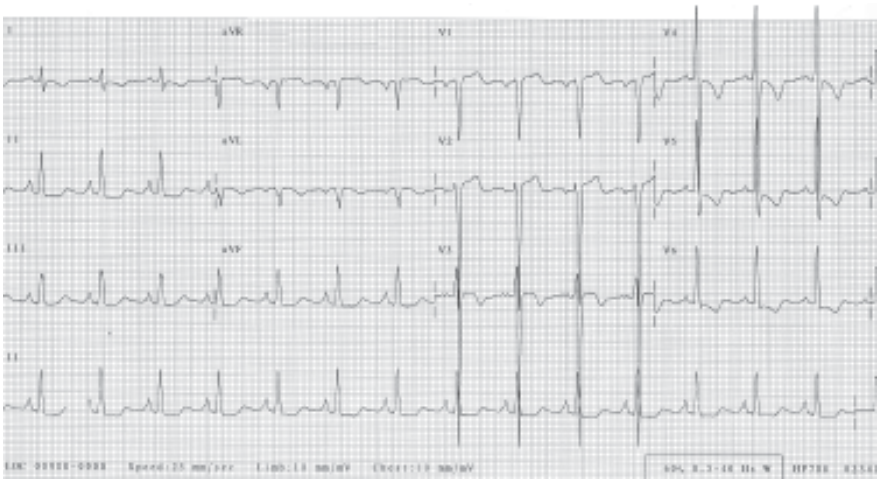

Fig. 2. Twelve-lead ECG on presentation.

marked ST-T changes in leads V3 to V6 (Fig. 2) which were considered as "ischaemic" changes. Echocardiography was not performed due to equipment failure. A non-contrast enhanced brain CT was performed which was reported as normal (Fig. 3A). Other significant laboratory findings were blood glucose 363 mg/dL, creatinine kinase (CK) 256 U/L (normal, 39 to 308 U/L), CK-MB 165 U/L (normal, 7 to 25 U/L), and troponin-I 0.03 ng/mL (normal <0.05 ng/mL). Diabetic emergencies such as hyperosmolar syndrome and ketoacidosis were excluded by normal blood osmolarity and the absence of ketone bodies in blood and urine. Drug screening test was not performed because intoxication was considered unlikely through history taking. At this stage, the diagnosis of non-ST-elevation AMI complicated with pulmonary oedema was suspected and the patient was treated accordingly. In order to prevent further hypoxic brain insults, his BP was controlled cautiously with nitroglycerin (NTG) infusion, targeting at a reduction in systolic BP of no more than 15%. His ABGs were rapidly normalised by using PEEP-assisted ventilator and diuretics. After consulting with the in-house cardiologist, a primary cardiac catheterisation was carried out, showing patent coronary arteries and normal heart contractility, with an estimated ejection fraction of 68%. Spontaneous reperfusion was assumed, and he was admitted to the intensive care unit (ICU) for further treatment.

Under the diagnosis of AMI, the patient was treated with NTG pump, diuretic (bumetanide), beta blocker (carvedilol), angiotensin-converting enzyme inhibitor (captopril), and antiplatelet (clopidogrel). However, subsequent measurements of cardiac enzymes (6 hours after presentation) did not support the diagnosis of AMI: CK 376 U/L (initially 256 U/L), CK-MB 22 U/L (initially 165 U/L) and troponin-I 0.44 ng/mL (initially 0.03 ng/mL). Although a follow-up

chest radiograph showed dramatic resolution of the pulmonary oedema and there was no evidence of hypoperfusion to the liver and kidneys, the patient was in persistent deep coma ($E_1V_EM_1$), which was considered to be the result of hypoxic brain insults. The ICU nurse detected anisocoria 37 hours after admission and a brain CT was repeated which showed substantial hypodensities in the left cerebral hemisphere with marked midline shift (Fig. 3B). Emergent decompressive craniectomy was performed but the patient died of refractory ventricular arrhythmias during the surgery.

Discussion

Florid pulmonary oedema can develop acutely in patients who have sustained sudden neurologic injury, including SAH, intracranial haemorrhage, TBI, acute hydrocephalus, brain tumours, phenothiazine overdose and seizure.[3] Although both high-pressure and increased-permeability abnormalities have been reported as the origins of NPO, the exact neurologic pathways responsible for initiating NPO remain uncertain. Smith et al[4] have propounded that transient left heart failure or neurally mediated pulmonary venoconstriction are contributory factors in the pathogenesis of NPO in humans. On the other hand, animal models of NPO[5,6] demonstrated a change in pulmonary vascular permeability, suggesting that central mechanisms can alter the barrier function of lung endothelium. Although the hydrostatic and permeability theories seem to develop through separate mechanisms, their combined effect is probably synergistic on the accumulation of extravascular lung water.

Ischaemic stroke is a rare cause of NPO. To our knowledge, our patient is one of the very few cases of NPO following non-haemorrhagic cerebrovascular accidents which have been reported in the English literature.[7] One major pitfall in the diagnosis of NPO in this case is the lack of focal neurologic deficits (patient in coma and respiratory failure on presentation) and the lack of abnormal findings on brain CT scan in the early course of a stroke. Physicians were also misled by the "abnormal" ECG findings and an increased activity of CK-MB isozyme, which indicated a direct myocardial injury. However, the presumably abnormal ECG findings (lateral ST-T changes) were probably strain patterns in patients with LVH. Consequently, the patient was treated as a case of cardiogenic pulmonary oedema complicated by hypoxic encephalopathy rather than the actual diagnosis of NPO caused by massive cerebral infarction. It has been described that both ischaemic and haemorrhagic strokes may cause cardiac abnormalities such as release of cardiac enzymes, changes in ECG, or clinical or echocardiographic evidence of left ventricular dysfunction.[8-10] Apak et al[11] stated that measurement of the serum levels of cardiac troponin-T is of clinical importance in evaluating myocardial injury and provides a useful aid in estimating the volume of stroke lesions. Although the precise underlying mechanism could not be determined, excessive catecholamine discharge caused by hypothalamic stress was considered to be responsible for the development of myocardial dysfunction in these patients.[12,13]

Another controversy in the management of this patient is that whether a coronary angiogram was necessary when there is suspected presence of significant hypoxic encephalopathy. In this case, the cardiologist preferred an early and aggressive treatment strategy because isocoric pupils with prompt light reflexes were found on arrival, which may indicate a good outcome in the setting of hypoxic-ischaemic brain insults.

In summary, our patient presented with signs of myocardial injury, which may be seen in patients with NPO following massive ischaemic stroke. Such cases can be easily misidentified as cardiogenic pulmonary oedema due to ischaemic heart disease when the preliminary brain CT appears "normal" and focal neurologic symptoms or signs are lacking.

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