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【 PICTURES IN CLINICAL MEDICINE 】

Neurogenic Pulmonary Edema without Norepinephrine Elevation

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Key words: neurogenic pulmonary edema, traumatic brain injury, norepinephrine

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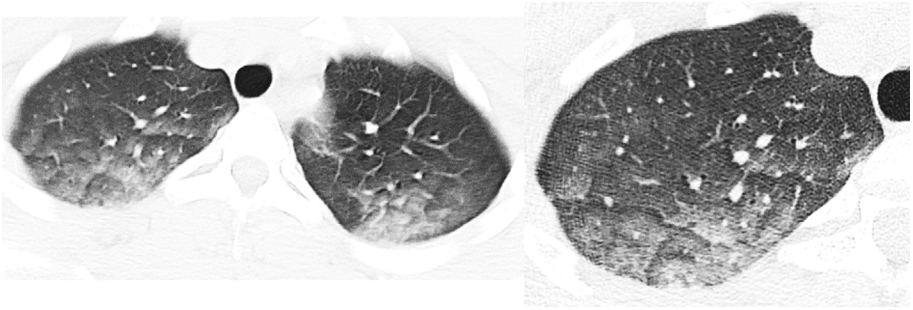
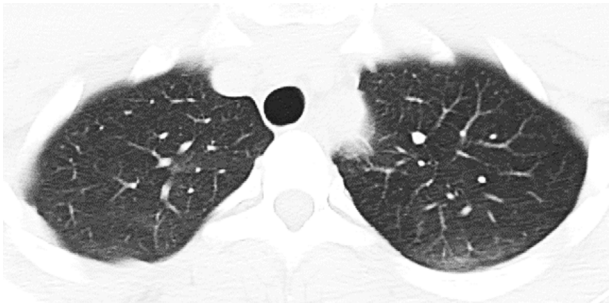


Picture 1.



Picture 2.

A 22-year-old medical student transiently lost consciousness, possibly due to orthostatic dysregulation in the operating room during a surgical internship; his head and neck struck the floor heavily. He was brought to the emergency room immediately, where he regained consciousness. His vital signs were normal, and chest auscultation and a physical examination revealed no abnormalities. Although his oxygen saturation was 99%, bilateral infiltration was detected on computed tomography (CT) (Picture 1). The patient's laboratory findings, including his plasma norepinephrine (311 pg/mL) were unremarkable just after the accident. The results of electrocardiography and echocardiography were normal, and his inflammatory marker levels were not elevated. Thus, cardiac failure and pneumonia were excluded. Based on the presence of bilateral infiltrations after a central nervous injury without other common causes, we diagnosed neurogenic pulmonary edema (NPE) after traumatic brain injury. The bilateral infiltration on CT improved 1 day later (Picture 2).

NPE is a syndrome characterized by the acute onset of pulmonary edema following a central nervous system insult. The radiological findings of NPE are bilateral and predominant at the apices in approximately 50% of cases; they typically disappear within 1-2 days (1), which was compatible with this case. Although the mechanism underlying the development of NPE is thought to involve catecholamine storms after injury (2), NPE did not involve norepinephrine in a rat model (3). Indeed, the patient's norepinephrine did not increase. Further investigations are needed to determine the mechanism underlying the development of NPE.

The authors state that they have no Conflict of Interest (COI).

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