Acute Pulmonry Oedema rare causes and possible mechanisms

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摘要

Abstract

Acute pulmonary oedema usually has a fatal outcome. In this clinical report, we present rare cases of pulmonary oedema that were associated with Japanese B encephalitis, lymphangitis in breast carcinoma, fat embolism due to long-bone fracture, and the rupture of cerebral mycotic aneurysms. A total of 18 patients in the four disease categories were collected in two teaching hospitals in Taipei and Hualien. Upon admission, routine and specific examinations were taken and all patients showed clear lungs by chest X-ray; however, signs of acute pulmonary oedema occurred within 7 days. After resuscitation, all patients died of acute pulmonary oedema. In patients with fat embolism, the levels of non-esterified plasma fatty acids, cGMP, 5-hydroxytryptamine (serotonin) and nitrates/nitrites were increased during pulmonary oedema. Immunohistochemical staining revealed virus infection and neuronal death, predominantly in the medial, ventral and caudal medulla in cases of Japanese B encephalitis. The pulmonary oedema due to central sympathetic activation in Japanese B encephalitis may be related to destruction of depressor mechanisms in the medulla. The rupture of mycotic aneurysms is known to cause cerebral compression that results in acute pulmonary oedema. Blockade of lymphatics, capillaries and venules in breast carcinoma with lymphangitis causes the development of rapid lung oedema. The pathogenesis of pulmonary oedema is much more complicated in fat embolism. Mediators such as cGMP, 5-hydroxytryptamine, nitric oxide and presumably other chemical substances may also be involved.