G24 Two Fatal Cases of Hidden Pneumonia in Young People

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The goal of this presentation is to review two fatal cases of hidden pneumonia in young people.

This presentation will impact the forensic science community by demonstrating how accute respiratory distress syndrome (ARDS) can result in death. Also young people generally, in cases of pneumonia, can be treated and consequently prevent death. Therefore, these cases illustrate the importance of early diagnosis of this condition.

ARDS is a severe lung disease characterized by inflammation of the lung parenchyma leading to impaired gas exchange with concomitant systemic release of cytokines and inflammatory mediators frequently resulting in multiple organ failure (MOF). This condition usually requires a rapid application of mechanical ventilation and admission to an intensive care unit.

When the endothelium of lung capillaries and the alveolar epithelium are damaged, plasma and blood spread in the interstitial and intralveolar spaces. Such a change induces decreased lung compliance, pulmonary hypertension, reduced functional capacity, modified ventilation/perfusion ratio, and hypoxemia. ARDS can occur within 24 to 48 hours of an attack of acute illness. In such a case the patient usually has shortness of breath and tachypnea.

Typical histological presentation involves diffuse alveolar damage (DAD) and hyaline membrane formation in alveolar walls.

If the underlying disease is not diagnosed and treated, the condition of the patient will worsen resulting in shock and/or MOF potentially resulting in sepsis.

Supposedly over 30% of ARDS cases are due to “sepsis syndrome,“ which is characterized by leukocytosis or leukopenia, fever, hypotension and leading to the identification of a potential source of systemic infection via positive blood culture for pathogenic agents.

The rate of survival in case of severe ARDS with appropriate and early treatment is 50%. However, if the severe ARDS induced hypoxemia is not recognized or treated, or if the disease reaches is not diagnosed until the terminal phase, cardio-respiratory arrest occurs in more than 90% of patients.

Case 1: A 29-year-old man was found lifeless at home by his girlfriend. Death scene investigation was unremarkable. He took psychotropic drugs, and he was known to be an abuser of alcohol and drugs. Family history was negative for sudden death. A complete postmortem examination was performed four days after death. External examination was insignificant. The internal examination revealed polyvisceral congestion, microthrombosis, cerebral and pulmonary oedema. Free citrine liquid was found on both sides of the pleural cavities.

Marked congestion and release of foamy material on sectioning of both lungs was observed. Hydrostatic docimasia for large and small fragments was positive in all fields such as an index of bilateral consolidation. The histological lung examination, performed with routine haematoxylin-eosin staining, revealed diffuse alveolar damage, endobronchial and endoalveolar infiltrates of polymorphonuclear neutrophilic leukocytes and focal emphysema. No fungal infections were detected using slides by PAS and Grocott staining. Gram staining didn’t reveal evidence of bacteria. Toxicology was negative for drugs and alcohol.

Case #2: A 31-year-old man was with a history of pharingodinia, fever, and cough taken to the hospital. The clinical symptoms progressed to acute onset of increasing shortness of breath rapidly progressing to acute respiratory failure with haemoptysis. Chest x-ray demonstrated bilateral diffuse airspace opacification; the high resolution CT confirmed the presence of bilateral diffuse airspace consolidation associated with liquid in pleural cavities. The patient, with a severe leukopenia, was admitted to the intensive care unit, but died after a few hours. Two blood cultures were positive for group A beta-hemolytic *Streptococcus*. No other pathogenic agents were present. An autopsy was performed within 48 hours. The internal examination revealed an increase in lung weight and findings were consistent with intense congestion attributable to a bilateral pneumonia. The histological examination of lung specimens showed a pattern of diffuse alveolar damage and the presence of intralveolar bacterial and fungal colonies. In the kidneys a thrombotic microangiopathy compatible with DIC was found.

In conclusion, the cause of death was, in both cases an acute cardio- respiratory failure secondary to acute bilateral pneumonia with DAD and consequently ARDS, sepsis and DIC.

Hidden Pneumonia, Diffuse Alveolar Damage, Adult Respiratory Distress Syndrome

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