G56 Sudden Death From Atypical Pneumonia in a Healthy Adolescent

Sabina Di Donato, MD, Ospedale San Carlo - U.O. Medicina Legale, Via Potito Petrone, s.n.c., Potenza, 85100, ITALY; Margherita Neri, PhD, Department Forensic Pathology, University of Foggia, Viale degli Aviatori 1, Foggia, 71100, ITALY; and Rocco A. Maglietta, MD,*
C.R.O.B. - Rionero in Vulture (Pz), via Padre Pio, 1, Rionero in Vulture (Pz), 85100, ITALY

After attending this presentation, attendees will become familiar with the possibility that a completely asymptomatic atypical pneumonia may induce sudden death, even in a previously healthy adolescent, with absence of histological signs of diffuse alveolar damage.

This presentation will impact the forensic science community by making attendees aware of the insidious development of atypical pneumonia in immunocompetent subjects, focusing the possible responsible mechanisms of sudden death in such cases, in the absence of ARDS and histological signs of diffuse alveolar damage.

The most common causes of atypical pneumonia are Mycoplasma pneumoniae, Chlamydophila pneumoniae and Legionella pneumophila which cause fifteen percent to as much as fifty percent of cases of community-acquired pneumonia. Other organisms include viruses; few cases are due to zoonotic agents like *Chlamydophila psittaci*, *Coxiella burneti* and *Francisella tularensis*. Clinical and pathological patterns range from mild upper respiratory infection to severe lower respiratory tract disease. Atypical pneumonia generally is benign, with systemic complaints often more prominent than respiratory ones; fever, headache and myalgia are common. Although the clinical course is often self- limited, these pathogens can cause severe community-acquired pneumonia. The inflammatory reaction is localized in the alveolar septa that appear thickened, edematous, with infiltrates of leukocytes. In severe cases, fibrinous thrombi inside alveolar capillaries and haemorrhagic necrosis of alveolar walls are visible. Alveoli may contain scant exudate. Fibrin and hyaline membranes line the denudated alveolar walls, due to diffuse alveolar damage. Superimposed bacterial infection is common.

Case Report: A 16-year-old boy who spent all the day with his family, went to sleep after dinner. His brother checked on him after one hour and found him agonizing in an anomalous prone position, with the legs out of bed. Immediately he turned the body supine, called the ambulance, and tried to resuscitate him. When the doctor arrived, after the attempting with reanimation maneuvers, pronounced the adolescent dead. The boy had a negative history (except for a mild headache) and a negative family history for sudden death. He was a basketball player on the school team and was not known as a drug abuser. Death scene investigation was unremarkable. External examination was insignificant except for the presence of a little superficial wound on the right frontal scalp. The internal examination revealed polyvisceral congestion, cerebral and pulmonary edema, free fluid in the pleural cavities, and release of foamy material on sectioning of both lungs. The left ventricle showed a concentric hypertrophy (anterior wall 2 cm, lateral 2.4 cm, posterior 2 cm, septal 2.2 cm). The histological examination showed a pattern of massive diffuse interstitial pneumonia with markedly thickened alveolar septa with extreme congestion of capillaries, the presence of abundant eosinophilic material, and infiltrates of leukocytes. In the adjacent fields there were some amorphous eosinophilic material and erythrocytes inside the alveoli. The immunohistochemical stains revealed that the pulmonary infiltrates consisted of lymphocytes, histiocytes and plasma cells. There were some foci of leukocytes within the epicardium, and focal areas of patchy myocardial fibrosis and perivascular fibrosis were visible, with a mild degree of myocardial hypertrophy. The encephalon showed leukocytic meningitis with subarachnoid infiltrates of lymphocytes and mild perivascular edema. The immunohistochemical analsysis (RSV, HSV1, HSV2,VZV,CMV, HHV A and B, Parainfluenza Virus 1, 2 and 3, Adenovirus, Aspergillus spp., P. carinii, T. gondii) gave negative results. Additional tests were carried out to identify possible pathogenic agents through microbiological studies. Toxicological screening was negative. Molecular genetic analysis was conducted and excluded underlying heritable diseases. The decedent's parents indicated that the boy did not have the scalp injury before going to sleep, so it's possible that the boy suddenly fell to the bed, striking the bedside table and arresting in the anomalous position described by his brother. To explain the occurrence of sudden death in this case, two possible mechanisms of acute respiratory failure are hypothesized: (1) the underlying respiratory acidosis (well tolerated by a young active boy, by means of an induced tachypnoea) and hypoxemia may have conducted to tachycardia and deteriorating hemodynamics. This instability may have elicited a lethal ventricular arrhythmia supported by a mechanism of re-entry, considering that the boy's heart showed diffuse areas of patchy fibrosis; and (2) the irritation of the adjacent cerebral cortex by inflamed meninges may have caused epileptic seizures. Seizure activity can disrupt normal physiological regulation and control of respiratory and cardiac activity (similar to mechanisms operating in cases of sudden death in epilepsy), precipitating the unstable equilibrium present at lung level (reduction in gas exchange due to massive interstitial pneumonia), causing an acute respiratory insufficiency.

Sudden Death, Atypical Pneumonia, Meningitis

 ** Presenting Author*