

# Analysis of causes that led to Eliza Jane Scovill's cardiac arrest and death

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## Abstract

My review of the medical evidence presented in this case and the pertinent medical literature clearly shows that Eliza Jane's death was not caused by *Pneumocystis carinii* Pneumonia (PCP) as alleged by the medical examiner, or any other type of pneumonia. Eliza Jane's lungs did not show an inflammatory response to medically justify a diagnosis of pneumonia. Pneumonia is a term that refers to inflammation and consolidation of the pulmonary parenchyma.

Eliza Jane's death resulted from an acute allergic reaction to amoxicillin, which caused severe hypotension (due to the leakage of significant amount of fluid outside the blood vessels), shock, and cardiac arrest. The autopsy revealed that she had pericardial and pleural effusion and ascites. In addition, her organ weights (lungs, heart, liver, and kidneys) were increased significantly. The weight of Eliza Jane's lungs, heart, liver, and kidneys were 184%, 131%, 121%, and 146% of the expected average normal weight for her age, respectively. Also, her liver was significantly enlarged and the hepatocytes show micro-and macrovesicular steatosis. Amoxicillin has been known to induce immune mediated toxic changes in the liver.

Eliza Jane suffered from an upper respiratory tract infection for about three weeks prior to her death on May 16, 2005. My investigation indicates that her respiratory infection was probably caused by Human Parvovirus B19 (HPVB19) infection. HPVB19 has been known to cause upper respiratory tract infection, encephalitis, and aplastic anemia in children and adults. Eliza Jane had non-specific microscopic lesions in the brain consisting of microglia and multinucleated giant cells. These lesions could be caused by HPVB19.

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