



G101 Reye's Syndrome: A Proposal of Two Fatal Cases

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The goal of this presentation is to examine the clinical and histopathological aspects of two cases of Reye's Syndrome.

This presentation will impact the forensic science community by describing the necessity of a complete methodological forensic approach by means of autopsy and histopathological examinations to diagnose Reye's Syndrome. The anamnestic element, like this case shows, can be fundamental to direct the diagnosis in the correct way.

Reye's Syndrome (RS) is a descriptive term covering a group of heterogeneous disorders. Various factors were considered in the pathogenesis of RS, such as viral, toxic, drug-related, and metabolic. The link between aspirin ingestion and RS has not been scientifically demonstrated. Neither has the link between salicylate ingestion and a range of inherited metabolic disorders that can mimic RS (Reye-like diseases). Epidemiological data seems to show a significant reduction in the incidence of RS after the Centers for Disease Control and Prevention (CDC) issued warnings in the U.S. in 1980 against aspirin administration to children.

The first signs of RS are usually persistent vomiting and diarrhea. Other early symptoms may include irregular breathing, listlessness, drowsiness, lethargy, and coma. Fever is not usually present. The causes of symptoms associated with RS relate to dysfunction of the liver and a resultant increase in serum ammonia levels and other toxins. These toxins cause increased pressure in the brain and swelling, leading to brain dysfunction, and can progress to death.

Case 1: A 10-month-old girl was admitted to the hospital for a deep coma state (Glasgow Coma Scale (GCS) 3) during acute bronchitis and persistent fever. Her temperature was 39.7°C, her pulse was 125 beats per minute, and she was noted to be gasping. Her parents revealed they gave aspirin to their daughter for about four days prior to admission because of the flu. There was no previous history suggestive of a metabolic disorder.

A chest Computerized Tomography (CT) scan demonstrated the presence of pulmonary edema and incomplete flow of lower lobes. A brain CT scan showed diffuse, hypoxic-ischemic changes and slight cytotoxic edema of the parenchyma.

Chemical analysis showed elevated Serum Glutamic-Oxaloacetic Transaminase-Serum Glutamic Pyruvic Transaminase (SGOT-SGPT) without jaundice, elevated blood ammonia level, metabolic and respiratory acidosis (pH 7), hypoglycemia, and coagulopathy (PT%: 66.5, PT sec: 13.56). The child died ten days after admission. The autopsy revealed hepatomegaly and diffuse brain edema with massive bronchopneumonia. The etiopathogenetic definition was outlined by histological examinations of all organ samples, using Haematoxylin and Eosin (H&E) and immunohistochemical staining methods that revealed the presence of a diffuse microvesicular accumulation of fat in hepatocytes, diffuse vasospastic cerebral edema with small endovascular hemorrhages, myocardial myocytolysis, and widespread stasis of all the remaining organs. The death was attributed to multi-organ failure.

Case 2: A 12-year-old female presented with fever, asthenia, lack of appetite, and diarrhea for a week. Aspirin was administered daily. Seven days later, the patient suddenly lost consciousness in bed. On admission to the hospital, the child was in a deep coma with fixed mydriasis, areflexia, absence of response to any stimulus, and decerebrate posturing. Blood chemistry showed high SGOT (1555U/L) and SGPT (1395U/L), hypoglycemia (10mg/dL), and elevated prothrombin time (130sec). She died a few hours after admission. The HbSag test was negative. The autopsy revealed yellow complexion due to jaundice, brain edema, subserosal petechiae, lung congestion and signs of hemorrhages, intensely yellow liver with creamy consistency, and diffuse subcapsular hemorrhages. The etiopathogenetic definition was outlined by histological examinations of all organ samples, using H&E and immunohistochemical staining methods that confirmed stasis of all organs and diffuse hemorrhagic foci. The liver showed typical features of massive centrilobular necrosis and vacuolar degeneration of liver cells. Death was attributed to RS.

In conclusion, RS should be suspected when this pattern of symptoms appear during or, most commonly, after a viral illness with (or with out?) aspirin ingestion prior to presentation. Not all of the



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symptoms have to occur, nor do they have to be displayed in this order. Many diseases have symptoms in common and a complete autopsy with histopathological examination can aid in the correct determination of the cause of death.

Reye's Syndrome, Liver Failure, Cerebral Edema