

G40 False Positive Diagnosis of Subarachnoid Hemorrhage and Subdural Hemorrhage by Computerized Tomography

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The goal of this presentation seeks to raise awareness of the potential misdiagnosis of subarachnoid hemorrhage and subdural hemorrhage by computerized tomography. Attendees should understand possible reasons for misdiagnosis as well as their medicolegal and clinical implications.

This presentation will impact the forensic community and/or humanity by presenting three cases of misdiagnosed subarachnoid hemorrhage and one of subdural hemorrhage.

Death investigators should be aware of potential discrepancies between radiologic and autopsy findings. Subarachnoid hemorrhage (SAH) involves bleeding into the space between the pia and arachnoid membranes and subdural hemorrhage (SDH) is characterized by bleeding into the space between the dura and arachnoid membranes. SDH is generally associated with trauma while SAH has well-recognized traumatic and non-traumatic (e.g. ruptured cerebral aneurysm) etiologies. Computerized tomography (CT) is frequently used in the evaluation of cases of suspected head trauma and has a high sensitivity in the diagnosis of SAH and SDH. Misdiagnosis by CT of SAH has been infrequently reported but may have significant clinical and medicolegal consequences. Potential sources of misdiagnosis include hypoxic encephalopathy, meningitis and reviewer inexperience. This report addresses three adult cases where CT misdiagnosis of SAH occurred as well as a pediatric case where CT misdiagnosed a SDH.

The **first case** involved a 25-year-old man who presented to the emergency department with vomiting and abdominal pain following an alcohol binge. He had altered mental status and went into cardiorespiratory arrest shortly after presentation. He was resuscitated but remained comatose and succumbed to multisystem organ failure three days after admission. A head CT after resuscitation was interpreted as showing diffuse SAH. No SAH was identified at autopsy. Neuropathologic examination revealed changes consistent with hypoxic encephalopathy with cerebral edema. Cause of death was acute liver failure due to acute and chronic alcoholism.

The **second case** involved a 49-year-old man who presented in cardiorespiratory arrest to the emergency department after being found unresponsive at home. Past medical history was notable for a high cervical spine injury approximately one and half years prior to death. Because of the injury the decedent was ventilator dependent. After resuscitation he remained comatose and died one day later. A CT scan of the head after resuscitation was interpreted as showing diffuse SAH but no SAH was identified at autopsy. Neuropathologic evaluation demonstrated hypoxic encephalopathy with cerebral edema. The cause of the decedent's initial cardiopulmonary arrest was related to complications of his remote neck injury and this was listed as the proximate cause of death.

The **final adult case** involved a 33-year-old man with a history of substance abuse who collapsed at a fast food restaurant and was taken unresponsive to the hospital where he was resuscitated. He regained vital signs but had sustained a hypoxic brain insult and never regained consciousness before expiring 14 hours later. A head CT after resuscitation was interpreted as showing diffuse SAH. This was not present at autopsy and neuropathologic examination again showed only signs of hypoxic encephalopathy with cerebral edema. Cause of death was related to acute drug intoxication.

The **pediatric case** involved a 5 week-old infant who was brought to the hospital in extremis by her father after she developed labored breathing. She had been previously healthy but over the preceding hours was

described as progressively lethargic. She was intubated in the emergency department but became profoundly bradycardic during a head CT. She received inotropic support but expired four hours after presentation. Head CT was interpreted as showing a small right frontal SDH with blood at the posterior aspect of the interhemispheric fissure. No SDH was identified at autopsy, which revealed bacterial leptomeningitis and this was given as the cause of death. The father was released from police custody following autopsy as he had been arrested on suspicion of child abuse (shaken baby syndrome).

Forensic Science, Subarachnoid Hemorrhage, Subdural Hemorrhage