H45 Shallow-Water Blackout: A Rare Case of Death During Pool Free Diving

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Learning Overview: The goal of this presentation is to present a rare case of prolonged laryngospasm in a fit diver who was found submerged and lifeless on the bottom of a public swimming pool while he was training in free diving.

Impact on the Forensic Science Community: This presentation will impact the forensic science community by presenting the usefulness of a multidisciplinary forensic approach by autopsy, histological/immunohistochemical, and toxicological investigation in cases of sudden and unexpected death in the water, particularly in all cases in which common signs of drowning are not found on the body.

The sudden and unexpected death of a young, fit diver in a public swimming pool represents a challenge to the forensic pathologist. The lack of internal and external drowning signs requires the consideration of a differential diagnosis that includes at least four syndromes (i.e., preexistent cardiac disease, abnormalities in electrical conduction, epilepsy, and hypoxic blackout). Hypoxic blackout can occur at any dive depth, including at constant depth, on ascent from depth, or at the surface following ascent from depth. “Shallow water blackout” is a term referring to the loss of consciousness that occurs when hypoxia is expedited by hypocapnia caused by voluntary hyperventilation before the dive, and where alternative causes of unconsciousness have been excluded.

During free diving, divers can rely only on the oxygen found in their lungs, blood, and tissues. Oxygen (O_2) tends to decrease, while carbon dioxide (CO_2) increases in the blood level, stimulating the brain centers associated with breathing. These impulses will induce an urgency to surface and to inspire, while powerful diaphragmatic contractions begin. This moment has been designated the “inspiration break point.” During voluntary diving, if a diver engages in prolonged presubmersion hyperventilation, the pre-dive CO_2 levels will fall and the subsequent CO_2 rise during submersion may not be sufficient to provoke the stimulus to surface before the O_2 blood levels fall and the individual loses consciousness. At this point, the inspiration break point occurs and the unconscious submerged victim is at high risk of drowning.

Presented here is a case report of a Caucasian, 32-year-old man, 171cm in length, found submerged and lifeless on the bottom of a swimming pool where he usually went to train. He had no family history of cardiovascular disease. He was retrieved after a few minutes, but cardiopulmonary resuscitation attempts were ineffective. A multidisciplinary forensic approach, including autopsy, histologic, and toxicologic investigation, was performed. The autopsy external examination showed no injuries. Internal examination, however, revealed subarachnoid hemorrhage and diffuse petechiae overlying the pleural surfaces of the congested and edematous lungs. Once sectioned and compressed, much serousanguinous fluid was extruded. The larger bronchial branches revealed foamy liquid contents. There were no gastric contents. Hematoxylin and Eosin (H&E) -stained histologic sections revealed polyvisceral stasis, thin cerebral hemorrhagic suffusion, and massive pulmonary edema. The toxicologic examination did not reveal drugs or alcohol in the serum. No diatoms were observed in either the water samples or the marrow.

In sum, these anatomic and pathologic findings would support a death attributable to a hypoxemic-hypercapnic condition induced by prolonged laryngospasm due to the apnea during immersion. The autopsy showed very little volume of liquid in the airways, but histologic evidence of acute pulmonary edema. The lack of ingested liquid would be compatible with a laryngeal spasm and cardiac arrest, the consequence of a voluntary respiratory arrest in prolonged apnea conditions.

Apnea, Sudden Death, Hypoxic Blackout

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