G20 Fatal Spontaneous Non-Traumatic Subdural Hematoma and Terson Syndrome

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After attending this presentation, attendees will learn that a ruptured cerebral aneurysm can cause a compressive acute subdural hematoma without concomitant subarachnoid hemorrhage.

This presentation will impact the forensic science community by expanding the attendees knowledge base by increasing awareness of causes of non-traumatic subdural hematomas and retinal hemorrhages.

This presentation will inform attendees of something that they do not know. While most acute subdural hemorrhages are the result of trauma, forensic pathologists must be aware that a ruptured cerebral saccular aneurysm can cause a spontaneous non-traumatic subdural hemorrhage along with associated retinal hemorrhages (Terson syndrome).

Cerebral saccular aneurysms frequently rupture into the subarachnoid space, accounting for 70-80% of non-traumatic subarachnoid hemorrhages (SAH); however, aneurismal rupture also may result in concomitant intraparenchymal, intraventricular, or subdural hemorrhage. Most acute subdural hematomas (SDH) in adults are due to traumatic head injuries, although less common causes include coagulopathies, non-traumatic intracranial hemorrhage, intracranial hypotension, or post-surgical complications. A ruptured cerebral berry (saccular) aneurysm causing only an acute SDH is rare, representing < 1.5 - 2% of all ruptured aneurysms in several large case series. In 1881, Litten first described intra-retinal hemorrhage associated with SAH. However, Terson's description in 1900 of vitreous hemorrhage following SAH is now associated with this syndrome. Although originally defined by the presence of vitreous hemorrhage in association with SAH, Terson syndrome now encompasses any intraocular hemorrhage associated with intracranial hemorrhage and elevated intracranial pressures.

A case of 46-year-old woman who died suddenly and unexpectedly at her residence is presented. Found on the bathroom floor, she had no obvious injuries. According to investigations by the medical examiner and law enforcement, she had a vague past medical history significant for hypertension but did not consume alcoholic beverages or use illicit drugs. Subsequent toxicological analysis did not reveal any licit or illicit drugs.

At autopsy, she appeared well nourished and had a body weight, length ,and body mass index of 49.1 kg, 160 cm, and 19.1, respectively. Postmortem monocular indirect ophthalmoscopy revealed bilateral retinal hemorrhages. The right and left fundi exhibited 25-35 and 15-20 flamed-shaped and dot retinal hemorrhages over the posterior poles, respectively.

A 1.5 cm subscalpular contusion was left of the vertex over the parietal area. No subgaleal extravasated blood or skull fractures were present. Diffuse liquid and clotted subdural blood covered the cerebral convexities (R > L) and weighed 67 gm. The calvarial dura had adherent non-organizing blood over the right and left frontoparietal regions. The leptomeninges were thin and translucent without any extravasated blood. Compression of the midbrain involved the inferomedial temporal lobes and 2 x 1.5 x 0.3 cm dusky area of hemorrhage was in the inferomedial right temporal lobe (medial to the groove caused by transtentorial herniation). The arteries of the circle of Willis were in the usual anatomic configuration and patent. A 0.5 x 0.2 x 0.2 cm ruptured saccular aneurysm projected from the callosal side of the bifurcation of the left pericallosal and callosal marginal arteries. The brainstem contained Duret hemorrhages in the pons and midbrain.

Ophthalmological examination revealed bilateral diffuse optic nerve sheath hemorrhages and extravasasted blood within the perineural fat. The right and left fundi had 75-100 and 25-35 flame-shaped and dot retinal hemorrhages, respectively. These involved all four quadrants and extended past the equator but did not abut the ora serrata. The fundal hemorrhages were in all retinal layers and scant blood was in the vitreous of both globes.

A non-traumatic SDH can occur due to the rupture of cerebral saccular aneurysm. Most of these aneurysms are located on the internal carotid artery followed by the middle cerebral artery and anterior communicating artery, but only rarely arise from the distal anterior cerebral artery. Four mechanisms have been proposed by which blood from a ruptured cerebral aneurysm causes a SDH:

1. Successive small hemorrhages allow adhesions to develop and the final rupture dissects between the subarachnoid and subdural layers
2. The arachnoid membrane is breached by the rapidly accumulating blood from the rupturing aneurysm
3. A massive hemorrhage ruptures the cortex and breaches the arachnoid membrane
4. A carotid artery aneurysm located between the arachnoid layer and dura mater ruptures causing a SDH

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Subarachnoid hemorrhage almost invariably develops following the rupture of a cerebral aneurysm and only extremely rarely does a SDH occur without an associated SAH. While most acute subdural hemorrhages are the result of trauma, forensic pathologists must be aware that a ruptured cerebral saccular aneurysm can cause a spontaneous non-traumatic SDH along with associated retinal hemorrhages (Terson syndrome).

Acute Non-Traumatic Subdural Hematoma, Cerebral Aneurysm, Retinal Hemorrhages

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