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CASE REPORT

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Hyperextension and Rotation of Head Causing Internal Carotid Artery Laceration with Basilar Subarachnoid Hematoma

ABSTRACT: Hyperextension of the head can cause injury to the vessels at the base of the brain. These lacerations are believed to be caused by stretching of the vessels due to the abrupt movement of the head and rotational acceleration of the brain within the cranium, and they usually occur in the intracranial portions of the vessels, producing a subarachnoid hemorrhage. This is the case of a 35-year-old man who received a blow to the face that forcefully hyperextended and rotated his head to the left. Autopsy revealed an intracranial right internal carotid laceration extending from a calcified atherosclerotic plaque. This unusual injury may be due to a combination of blunt force applied to the head and the alteration of the vessel's structural and functional capacities secondary to atherosclerosis.

KEYWORDS: forensic science, forensic pathology, hyperextension, subarachnoid hemorrhage, internal carotid artery laceration, rotational injury, New York City, atherosclerosis

A 35-year-old man was witnessed to be held by two other men and struck in the face with an object by a third man. The impact caused his head to abruptly extend backward and rotate to his left. He immediately lost consciousness and fell to the ground. Emergency medical services arrived shortly after the assault and found him pulseless and without respiration. They attempted resuscitation, but he was declared dead by neurologic criteria. His body was kept alive at the admitting hospital for 17 h pending organ donation. He had no medical history of hypercholesterolemia or hyperlipidemia.

Thirty-eight hours after he was pronounced dead, an autopsy was performed at the Brooklyn branch of the Office of the Chief Medical Examiner of the City of New York. External examination revealed a 7.5 by 2.5-cm abraded contusion extending across the right jaw and face (Fig. 1). There was a smaller abrasion on his right forehead.

Anterior, posterior, and lateral neck dissections were performed, including the distal extracranial portion of each vertebral artery. The anterior cervical vertebrae were examined as the neck organs were removed. Bilateral posterior-lateral neck incisions were made to expose the lateral cervical vertebrae. The vertebral arteries were dissected at the distal aspects. A posterior neck dissection was performed and the spinal cord was removed. There were no areas of hemorrhage and no fractures present in the neck structures. The skull had no fractures, and there was no epidural or subdural hemorrhage. The brain was removed without creating artifactual lacerations of the arteries at the base of the brain. There was a basilar subarachnoid hematoma (Fig. 2). Inspection of the vessels at the base of the brain revealed a laceration of the right internal carotid artery (Fig. 3). It consisted of a 0.3-cm defect extending from a 0.6-cm intimal tear of the anterior aspect of the artery approximately 0.6 cm distal to its intracranial origin near the clinoid process. The arterial defect extended from a partial separation of the intima at a calcific atherosclerotic plaque (Fig. 4). Neuropathologic evaluation also revealed brain swelling and venous sinus thrombosis. There were no cerebral artery aneurysms, intracerebral hemorrhages, or vascular malformations.

Internal examination of the viscera revealed cardiac hypertrophy (460 g) and left ventricular hypertrophy (1.4 cm). The coronary arteries showed slight atherosclerosis with up to 40% occlusion. The toxicology report was negative for commonly used prescription and illicit drugs, including cocaine and its metabolites.

Discussion

Subarachnoid hemorrhage (SAH) is the most common intracranial lesion seen in cases of blunt force head injuries. The clinical sequelae of such injuries vary, but in a small number of instances the injuries have proven to be rapidly fatal. When viewed in the context of fatal head trauma in general, Freytag (1) suggested that SAH was the sole traumatic intracranial finding in less than 1% of cases. Identifying SAH as a traumatic cause of death can be difficult, particularly in those instances in which the SAH is the sole intracranial finding, because the degree of external injury is some-

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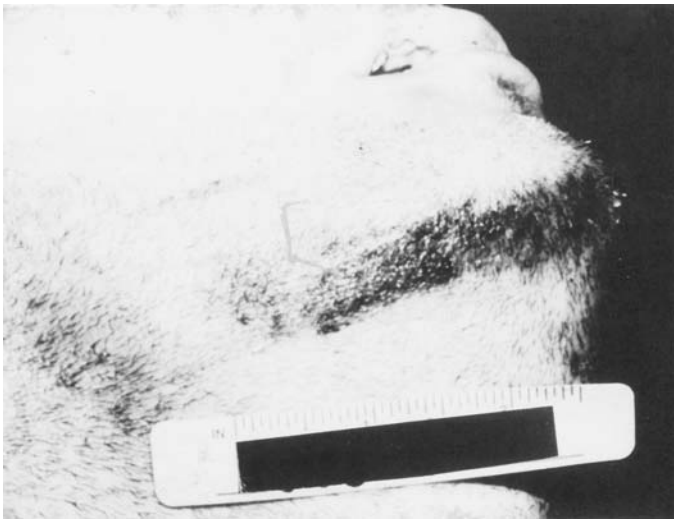

FIG. 1—*Abraded contusion on right jaw and face.*

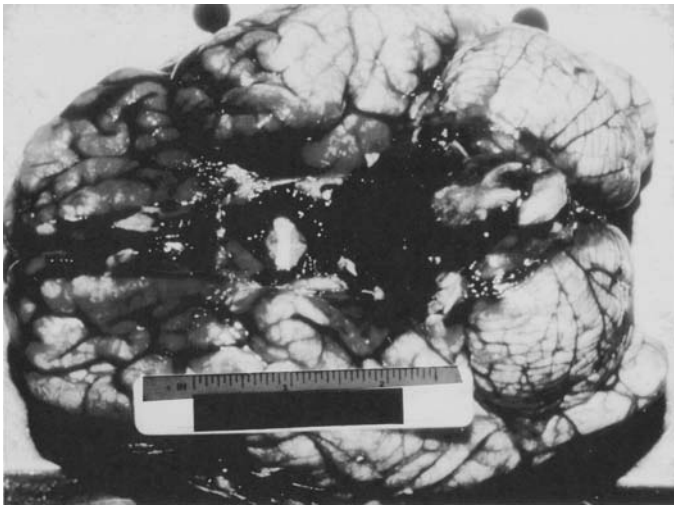

FIG. 2—*Basilar subarachnoid hematoma.*

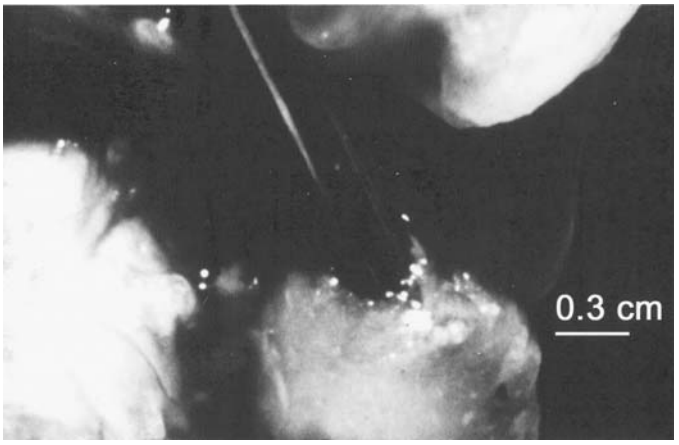

FIG. 3—*Laceration of right internal carotid artery.*

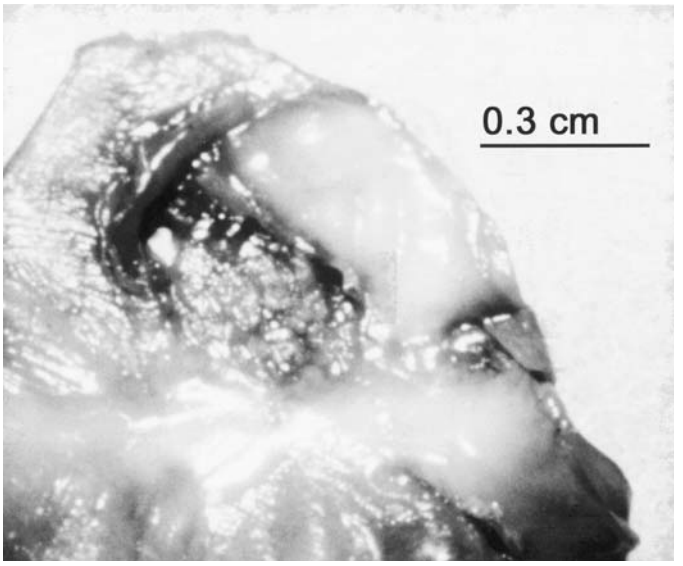

FIG. 4—*Calcified atherosclerotic plaque and laceration of right internal carotid artery.*

times so slight that the visible signs of trauma are frequently judged to be of insufficient magnitude to account for the development of the hematoma.

In a series of cases involving traumatic SAH, Tatsuno and Lindenberg (2) found that 82% of such cases involved blows to the head or neck. In their series, fatal basal SAH tended to occur in young, apparently healthy, males, with over 50% occurring in males between ages 20 and 39.

The same authors (2) also found that identification of the bleeding source is not always possible and were unable to identify a specific arterial source in 59% of the cases they studied. In another series of 20 cases of traumatic basal SAH, the arterial source of the SAH was not demonstrated in 35% of the cases studied (3). Contostavlos (4) has described several cases where impacts to either the neck or head lead to a laceration of the distal portion of the extracranial vertebral artery, causing dissection and subarachnoid hemorrhage.

In this same series of 20 cases, where the source of bleeding could be identified, it was found that 50% of the cases had demonstrable laceration of a vertebral artery, with one case of a lacerated basilar artery. Krauland (5) similarly reported identifying the vertebral artery as the source of hemorrhage in 12 of 31 cases, or 39%. Gray et al. (6) examined 14 cases of traumatic basal SAH specifically due to tears in the vertebral arteries and noted that 78% of these lesions were in the intracranial portions of the vessels. In their survey, Tatsuno and Lindenberg (2) noted that of the 14 cases in which an identifiable source for the hemorrhage could be found, four cases were noted to have tears in the intracranial internal carotid arteries (28%), four cases revealed lesions in the vertebrobasilar arteries (28%), and two cases showed defects in the stem of the middle cerebral artery (14%).

In this case report, a 35-year-old man had a SAH arising from a clearly demonstrable tear in the intracranial portion of the right internal carotid artery, with an atheromatous plaque near the disrupted area of the vessel. We postulate that the forces acting on the head during abrupt hyperextension and rotation, in combination with the alteration and possible compromise of the vessel wall by atherosclerosis, are responsible for this unusual and fatal injury.

The internal carotid artery runs in the carotid sheath, anterior to the longis cervicis muscle and the transverse processes of the upper three or four cervical vertebrae. It then passes directly anterior to the anterior surface of the axis vertebra, where it then passes either anterior or medial to the articular mass of the atlantis vertebra. After this, it enters the petrous part of the sphenoid bone of the skull via the carotid canal. Within the bone, the internal carotid is relatively fixed, and it courses anterosuperiorly to exit the bone adjacent to the clinoid process, where it enters the cavernous sinus. It then gives off the opthalmic artery before joining the Circle of Willis and branching into the anterior cerebral and middle cerebral arteries and posterior communicating arteries.

This proximity to the atlanto-occipital joint makes the extracranial portion of the internal carotid artery vulnerable to stretch injury when the head is abruptly hyperextended or rotated. The portion of the internal carotid that is fixed within the sphenoid bone is pulled upwards or laterally, and the portion of the artery that passes close to the atlas and axis is stretched against their lateral masses and can therefore tear (7). It is also by this mechanism that sudden, sharp increases in blood pressure secondary to compression occur in the carotid vessels during abrupt hyperextension or rotation of the head.

The intracranial portion of the internal carotid artery is also subject to potentially damaging forces when the head is abruptly hyperextended and/or rotated from a resting position. When the head is suddenly moved, the brain oscillates within the cranial vault due to its inertia. After the head begins to move, the inertia of the brain initially causes it to move in a direction opposite the direction of force of the blow to the head and the head's movement. Then, as the head begins to snap back to its original position, the inertia of the brain has been overcome, and the brain moves in the direction of the blow as the head moves opposite. This oscillation of the brain is out of phase with the movement of the head, causing the arteries at the base of the brain to be subject to shearing forces. These forces are likely to be greatest when the blow to the head causes it both to be hyperextended and to move laterally (2). Therefore, the intracranial portion of the internal carotid artery, where it is attached to the mobile brain as it joins the Circle of Willis above where it is fixed within the sphenoid bone, is subject to these shearing forces during hyperextension and rotation of the head and can sustain a tearing injury. In the case we present here, the intracranial right internal carotid artery sustained a 0.3-cm tear adjacent to its exit from the cavernous sinus at the right anterior clinoid process after witnesses had seen the victim sustain a blow to the right face with a blunt object that hyperextended and rotated his head leftward.

In addition to the above-described forces, the compromised integrity of the arterial wall may also have rendered the artery vulnerable to injury. The intimal surface of the victim's right internal carotid, adjacent to the 0.3-cm tear, showed a 0.6-cm laceration adjacent to an atherosclerotic plaque, with partial separation of the plaque. This type of lesion appears to be a somewhat unusual finding, as another study (2) found no evidence of atherosclerosis in 33 of their 34 cases. They postulated that this finding was due to the young age of most persons who are typically subjected to such trauma. However, others have suggested that even in a younger population, atheromatous plaques may play an important role in determining the nature of arterial injury and its effects on the lumen (8). Studies of sclerotic vessels have demonstrated that fibrotic vessels have lower elastic moduli than normal vessels (9). Moreover, distensibility of the sclerotic vessels was essentially equal to that of normal vessels under low to moderate pressure conditions; but the difference in distensibility became much more important at pressures above 120 mm Hg (9). Pressure of this magnitude can occur during acute compression of the neck vessels secondary to trauma.

Animal models that recapitulate the progression of vascular disease have been used to examine vessel wall characteristics during early stages of hypercholesterol-induced atherosclerosis. Hironka et al. (10) have shown that there is an alteration in vascular responsiveness of the aorta in rabbits during the very early stages of atherosclerotic disease. Perhaps this phenomenon can be extended to humans with mild vascular disease, and normal compensatory mechanisms that ordinarily modulate vessel response to stretching and pressure increases become inadequate, making vessels more vulnerable to injury.

Finally, it has been postulated that plaques in the carotid arteries of middle-aged Americans may contribute to alteration or focal destruction of arterial wall structural elements (11). This disruption of structural integrity of vessels at the site of atherosclerotic plaques, coupled with decreased elasticity and impaired ability to compensate for rapid increases in intraluminal pressure, may act synergistically to focally predispose vessels to laceration secondary to trauma.

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