Closed Cervical Cranial Trauma Associated with Involvement of Carotid and Vertebral Arteries*

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Injury of the carotid and the vertebral system may occur as a result of closed impacts to the head and neck. Injury of these vessels is more common with perforating and penetrating wounds of the neck and head. Tear or thrombosis of the carotid artery associated with perforating wounds of the neck, thrombosis of the carotid artery with fracture of the mandible, and tear of carotid and vertebral arteries caused by stab wounds have been described frequently. Injury of the vertebral artery by perforating wounds is seen less commonly. In this paper, the literature on this subject will be reviewed, and a report will be made of 5 patients who have had carotid and vertebral vessels involved as a result of closed impacts of the neck and head.

Review of Literature

The intima of the left internal carotid artery was torn and rolled in the case described by Verneuil in 1872. The patient had a head injury and a swelling of the left sternocleidomastoid muscle. A right hemiplegia, coma, and death resulted. There was complete occlusion of the internal carotid artery caused by a mural thrombus extending up and into the middle cerebral artery and branches. There was extensive softening of the middle third of the left cerebral hemisphere. The author ascribed the arterial tear to torsion and bending of the neck at the time of the head injury.

The patient of Northcroft and Morgan sustained a bruise of the left side of the neck when a rope hanging from a passing vehicle caught the patient about the neck. He became unconscious following a lucid interval and was paralyzed in the right half of the body. A possible intracranial hemorrhage was suspected, but operation was negative for the presence of such a lesion. An autopsy showed a dissecting aneurysm of the left internal carotid artery with complete occlusion of the internal carotid artery. Subintimal hemorrhage caused by an injury resulting in a complete occlusion of the internal carotid artery near the bifurcation was also described by Hutchinson and Yates. Schneider and Lemmen have discussed 2 examples of thrombosis of the carotid artery secondary to nonpenetrating injuries of the neck.

In closed injuries of the head a carotid-cavernous fistula may be caused. Such a fistula may be associated with a fracture extending into the paranasal sinuses. Occasionally, the fistula may bleed into the paranasal sinuses causing severe epistaxis. Cairns, Christensen, Davis, Hallberg, Hamilton, Jacques, and Ogura and Senturia have described severe epistaxis in association with a tear of the carotid artery and/or aneurysm at the base with bleeding into the sphenoid sinus. The earliest case appears to be the one described by Guibert in 1895. Rousseau and Spillman and Vandooren et al. have described instances of severe epistaxis following head injury caused by a tear of the internal carotid artery intracranially along a fracture line extending into the paranasal sinuses. Arteriovenous communication between the carotid and the cavernous sinus ruptured into the nose with fatal epistaxis in the case of Seftel et al.

Angiography has been used to study patients with severe post-traumatic epistaxis. Schlosshauer and Vostein found the carotid angiogram to be negative in 1 of their 2 cases.
In the case described by Denecke and Harttert the angiogram also was negative. In many cases, the epistaxis is delayed but was immediate in about one-fourth of the patients. The patients in the cases of Takeda and Kawakita, and Christensen, and in 1 of the cases of Cairns died following an epistaxis soon after injury.

Birley and Trotter in 1928 described traumatic aneurysm of the intracranial portion of the internal carotid artery treated by ligation of the common carotid artery. The patient described by Voris and Basile was treated by trapping the fistula between a ligation of the internal carotid artery in the neck and a clip on the intracranial portion of the internal carotid artery through a craniotomy. A similar case was described by Weaver et al. The triad of unilateral blindness, orbital fracture, and massive epistaxis after head injury was described by Maurer et al. The ruptured aneurysm of the carotid artery also bled into the sphenoidal air sinus through a fracture of the base of the skull crossing the sphenoid sinus. There was unilateral blindness and a fracture of the roof of the orbit in their case.

Involvement of the vertebral artery has been described following manipulation of the neck by chiropractic or other methods of management. Pratt-Thomas and Berger, in 1947, described 2 patients aged 32 and 35 who lost consciousness during a chiropractic treatment. In both instances there was thrombosis of the basilar artery and in 1 the posterior inferior cerebellar artery also was thrombosed. In the case of Ford and Clark a treatment was given the patient by his wife for a pain in the neck. Following this twisting of the neck in the 37-year-old patient there was vertigo, tinnitus, and right homonymous hemianopia. He died 60 hours later and autopsy showed thrombosis of the left posterior cerebral, the basilar, and the left posterior inferior cerebellar arteries. In a patient with thrombosis of the basilar artery seen at autopsy, there was unconsciousness and coma after an operation for a left stellate ganglionectomy. With the patient’s head turned to the right, there developed a left-sided thrombosis of the vertebral artery and involvement of the basilar artery. The vertebral artery was thrombosed as it entered the dura mater in the base of the skull. The arteries in the neck lower down apparently were normal. Manipulation of the neck was not followed by death in the cases of Kunkle et al., Schwarz et al., and Green and Joynt. Thrombosis of the vertebral artery was suspected by the evidences of a lateral medullary syndrome. The thrombosis may have been caused by the rotation of the head in the region of the atlas and the atlanto-occipital membrane with compression of the artery. Yates found evidences of hemorrhage in the adventitial lining of one or both vertebral arteries in 24 out of 60 infants who died in the perinatal period. Often the hemorrhages were severe enough to cause narrowing of the lumen of the vessel and in 1 instance, 12 days post partum, there was a vertebral thrombosis.

Carpenter’s patient fell 10 feet to the ground from a tree, becoming paralyzed in both lower extremities. He was rendered unconscious for about 2 minutes. Soon after entrance into the hospital, he had intense vertigo, vomiting, and a horizontal nystagmus which was made worse on lateral gaze. Roentgenograms showed a compression fracture of the 7th cervical vertebra. The C6-C7 disc had ruptured, the superior surface and the posterior two-thirds of the body of C7 was displaced downward and posteriorly so that it projected 4 to 5 mm. into the spinal canal at the midline. There was a fracture of the right lamina of the 7th cervical vertebra. The patient, the following morning, had weakness of the left upper extremity and labored respiration. He died suddenly while being prepared for tracheotomy 29 hours after injury. Autopsy revealed an infarct of the left cerebellar hemisphere and the left side of the brain stem. There was a firm thrombus in the left vertebral artery extending into the origin of the anterior spinal artery. The clot also extended into the posterior inferior cerebellar artery. The vertebral artery was thrombosed at the level of C6 transverse process and foramen. The left
lateral mass of C7 was intact, the spinal cord was flat and bruised at the level of the 1st and 2nd thoracic segments and the entire thickness of the cord was pulped at this level. The left vertebral artery was lacerated and there was tearing of the muscularis at the level of C6-C7. The lumen was filled with antemortem clot.

Osteophytic proliferation in the cervical spine causing deformation of the vertebral artery may facilitate injury of these vessels in trauma to the neck.

Brass\textsuperscript{3} described a rupture of the basilar artery following a moderate head injury with fracture of the left orbital roof. There was bloody spinal fluid and 15 days after injury the patient died. Autopsy showed a tear of the basilar artery with evidences of repair compatible with the assumption that trauma and rupture took place coincidentally.

**Case Reports**

**Case 1.** A.B., 57 years old, was struck on the head and neck on Nov. 13, 1957. He was paralyzed immediately in the right half of the body and aphasia developed.

He was seen by us on Dec. 5, 1957. The right hemiplegia and aphasia were still present. Angiograms showed atherosclerosis of the left carotid bifurcation. The right carotid bifurcation appeared normal (Fig. 1).

A 5-year follow-up has shown some improvement in paralysis. The upper limb still is affected and the speech is impaired. His disability probably is ascribable to forward embolism in the distribution of the middle cerebral artery caused by the trauma of the head and neck.

**Case 2.** C.L., 64 years old, was admitted to the hospital on Feb. 6, 1957. He had been choked by a son 3 weeks earlier. For about 5 minutes, he was quite normal; then, suddenly, he collapsed with right hemiplegia and aphasia. The aphasia improved to almost normal speech and the hemiplegia improved to hemiparesis. Angiography showed stenosis of the left carotid bifurcation (Fig. 2).

He was treated by anticoagulation with no change in his condition. He was readmitted 4 months later and an endarterectomy was performed on the left carotid bifurcation. About 10 days later, another angiogram showed the vessel

![Angiograms of left carotid artery showing atheromatous involvement of bifurcation with satisfactory intracranial visualization of the branches. A.B., aged 57, was attacked, struck on head and neck, with immediate onset of right hemiplegia and aphasia. Angiograms were obtained 23 days after injury.](image)
to be completely occluded with no appreciable change in the patient's condition. He was found to have sensitive bilateral carotid sinuses.

The patient expired Dec. 26, 1961 following an operation for a bleeding gastric ulcer at the age of 69. Compression of the neck may have dislodged some emboli which involved the distribution of the left middle cerebral artery, causing small-vessel involvement in this distribution.

Case 3. L.C., 39-year-old white male, was admitted to the hospital on Aug. 5, 1962. He gave a history of having been struck on the left anterior region of the neck about 2 weeks earlier by a car door. On the day of admission the patient had a transient attack of numbness involving the right arm and hand with dysphasia.

Examination revealed an alert and cooperative patient. He had normal speech, the neck was supple, and there were no evidences of injury upon the neck. Cranial nerves were intact, the visual fields were full on confrontation, and fundusoscopic findings were normal. Power of extremities, coordination and sensation were intact. Reflexes were equal on the two sides; the plantar responses were down-going. There was no astereognosis. Carotid compression on the right side produced moderate dizziness; on the left side it produced only slight dizziness. Thrombosis of the carotid artery caused by trauma was thought to be likely. Electroencephalogram was normal; bilateral carotid angiography showed complete occlusion of the left internal carotid artery.

On Aug. 10, 1962, the left carotid bifurcation was explored. Just below the crossing of the hypoglossal nerve, the wall of the internal carotid artery was hemorrhagic and the vessel was thought to be occluded. On opening the vessel, it was found that the intima had fractured and the lumen was partly occluded by the flapping edges of the intimal tear. There was no bleeding from the distal end of the vessel. A small catheter was passed distally for several inches, and, after removal of several clots from the distal end of the carotid artery, a good flow was obtained from the brain end. The opening in the vessel then was enlarged, the torn intima distally was sutured to the wall of the vessel and proximally was excised.

An angiogram was performed after the arteriotomy showing that there was patency of the vessel; the caliber of the lumen appeared to be quite small. There also was seen very good collateral circulation through the ophthalmic artery. Periarterial sympathectomy at the time of expo-
sure may have contributed to the ophthalmic collateral supply.

Postoperatively this patient has done well with no focal neurological abnormalities. He may be readmitted in about 3 or 4 months for another angiographic study (Figs. 3 and 4).

Case 4. K.D., a white male aged 53, sustained quadriplegia following a diving accident in shallow water. There was fracture-dislocation at the level of the 4th and 5th cervical vertebrae with compression fracture of the body of the 4th. Vertebral angiography by the retrograde brachial route showed some bending of the left vertebral artery but no actual compression of its lumen (Fig. 5). The right vertebral artery appeared normal. There were no cerebellar or brain-stem signs noted in this case.

Case 5. J.K., a colored male aged 58, sustained quadriplegia following an automobile accident. He had a fracture dislocation of the 5th and 6th cervical vertebrae. Vertebral angiography by the retrograde brachial route showed the right vertebral artery to be intact and of normal size. The left vertebral artery, on the other hand, was smaller and was found to be completely shut off at the level of the 5th cervical vertebral level (Fig. 6). It was our impression that the shut-off was caused by the injury. It is interesting to note that this patient showed no cerebellar or brain-stem signs, probably because the vertebral artery was small and the blood supply to the brain-stem area was not compromised or impaired by its loss.

Discussion

The possible involvement of extracranial vessels supplying the brain by penetrating and perforating wounds is very understandable. The involvement of these vessels in closed impacts of the neck and head also is a possibility. In the case of the carotid artery, a contusion of the wall of the vessel, and a clinging clot which propagates is one method

Fig. 3. Case 3. L.C., aged 39, was struck on the left anterior region of neck, July 22, 1962. Angiogram on Aug. 6, 1962 (a) showed complete occlusion of left internal carotid artery. Following carotid exploration on Aug. 10, 1962, postoperative angiogram (b) showed patency of internal carotid artery but a very narrow vessel. Whether this is a spasm or caused by mural clot is difficult to say. The periarterial sympathectomy performed on the internal carotid at the bifurcation during arteriotomy should have helped if this were a spasm.
of occlusive involvement. Another cause may be a fracture of the intimal lining with a curling of the intima with eventual thrombosis and forward propagation of the clot. Subintimal hemorrhage in the presence of an atheromatous disease of the bifurcation may be sufficient to complete the occlusion. In the presence of arteriosclerosis, hemorrhage from the media may cause a dissecting aneurysm which shuts off the lumen of the vessel. Boldrey et al.\(^2\) pointed out that there is an obvious proximity of the internal carotid artery to the lateral mass of the 2nd cervical vertebra in many instances. They feel that spontaneous thrombosis of the carotid artery may be aided by this compressing mass against the internal carotid artery.

The problem of forward embolism is somewhat more difficult to understand. In our Case 1, the injury was followed immediately by right-sided paralysis. Five minutes after the choking injury of the neck in Case 2, there were right hemiplegia and aphasia, and this story is well documented in the history obtained from the patient and the relatives. Both of these patients had atheromatous disease of the left carotid bifurcation. The injury could have mobilized some clots, in the vicinity of the atheromatous mass, with forward embolism of the small arteries in the distribution of the middle cerebral artery. This appears to be the likely mechanism. In Case 2, the neck had been compressed, but we doubt that the compressive ischemia was the factor since, for 5 minutes or so after the choking incident, this patient had no evidence of weakness or paralysis, but the paralysis of the right half of the body and aphasia developed later.

In Case 3, the obvious tearing of the intimal lining with a clot which propagated into the internal carotid artery toward the brain was seen at operation. It was, however, interesting to note that this patient had no paralysis and only a transient weakness with some dysphasia before he entered the hospital, indicating that he had adequate collateral supply. Whether or not the vessel will remain open is problematical although after the intimeotomy, and the tacking of the intimal lining to the adventitia, the angiogram showed patency of the internal carotid artery. Whether the vessel remains patent or not, he will probably remain asymptomatic. It should be emphasized that no evidence of severe involvement of the nervous system developed as a result of the complete occlusion of the vessel for several days. Such a case may explain the possible finding of asymptomatic occlusion of the carotid artery in some patients and raises the question that some of these patients might well have had a minimal to moderate injury to the neck which was not considered serious since there was no associated evidence of dysfunction of the carotid artery in the neurological status of the patient.

The mechanism of injury in dysfunction of the vertebral artery is through stretching and tearing of the intimal and medial tissue of the vessel in some cases of closed fracture-
Fig. 5. Case 4. K.D., aged 53, sustained quadriplegia after diving into shallow water. (a) and (b) show fracture dislocation of 4th and 5th cervical vertebrae, with compression of body of 4th cervical vertebra.

Fig. 5c and d. Case 4. Angiograms showing vertebral arteries. There is some bending of left vertebral artery at site of fracture, but no significant stenosis.
dislocations of the neck, particularly in the vicinity of the 5th, 6th, and 7th cervical vertebrae. In the case of Carpenter, stretching of the artery with tearing of the intima and media was the cause of the propagating clot and eventual occlusion of the basilar artery with unilateral cerebellar infarct. With closed fractures of the clavicle, there may be a tear of this vessel. Fracture of the spine may be associated with a tear or an occlusion of this vessel. In 1 of our cases, there was obvious shut-off of the vessel which caused no neurologic abnormality indicating that, in many such cases, one or the other vertebral artery may be injured with occlusion. Particularly if this happens to be the

Fig. 6. Case 5. J.K., aged 58, sustained quadriplegia in automobile accident. (a) Shows fracture dislocation of 5th and 6th cervical vertebrae; (b) is a well visualized and adequate right vertebral artery; (c) presents an occluded left vertebral artery at level of C6. This vessel is much smaller than the contralateral vertebral artery.
smaller of the two vessels, the patient may go on asymptotically.

A traumatic carotid aneurysm rupturing into the paranasal sinuses causing severe epistaxis is an important clinical picture. In the presence of severe epistaxis, angiographic study is certainly indicated if there is a history of injury. When an aneurysm is located, its treatment by ligation of the carotid artery in the neck or by intracranial approach may be decided upon as the lesion has been uncovered and its position in the cranial cavity ascertained. If such an epistaxis is associated with a carotid-cavernous fistula, ligation of the common carotid first, later, ligation of the internal carotid artery, still later, ligation of the internal carotid artery intracranially may be done in a step-like fashion. As concerns unilateral blindness, severe epistaxis, and orbital fractures being associated with aneurysmal lesions of the carotid complex on the affected side, it should be pointed out that unilateral blindness frequently may be present following a head injury with normal vascular channels shown in the angiogram. On the other hand, unilateral blindness with epistaxis may be different. It should raise the question of an aneurysmal lesion in such a case.

In the management of the patient who suddenly exhibits focal abnormalities following insignificant trauma of the neck and head, the need for angiographic evaluation becomes obvious. This may eliminate the need for unnecessary intracranial explorations and point to the lesion in the neck which should be treated first.

The medicolegal implications of this paper should be analyzed critically. It is our impression that a careful history of the case is an important must. Another consideration is the proximity of the trauma to the appearance of the focal abnormality. In the cases described above, there can be no question of the relationship between trauma and the resultant difficulty. In many instances which go to court, however, cases are presented in which, following an injury to the head, the stroke occurs 2, 3 and 4 months later, and to prove that this is an example of a stroke caused by the initial trauma frequently must fail because the proximity of the stroke to the injury is not well established. Frequently, there is an intervening period of several weeks during which the patient has been able to work and carry on in a relatively normal fashion. It seems to us very unlikely that, in such a case, an injury sustained several weeks earlier could have been the cause of the stroke seen several weeks later.

Summary

Our experiences with 5 patients with traumatic involvement of the carotid and vertebral arteries in the neck are described. The injuries were nonpenetrating in every instance.

Three cases of involvement of the carotid artery and 2 cases in which the vertebral artery was involved are discussed.

Intimal and medial tear, and subintimal hemorrhage, may all result in occlusive thrombosis.

The need for arteriographic study is obvious and may enable one to recognize the lesion and manage it surgically.

References

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