TRAUMATIC INTERNAL CAROTID ARTERY THROMBOSIS SECONDARY TO NONPENETRATING INJURIES TO THE NECK

A PROBLEM IN THE DIFFERENTIAL DIAGNOSIS OF CRANIOCEREBRAL TRAUMA

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IN PATIENTS who have concomitant injuries of the head and neck the physician’s attention is directed to the cranium when neurologic symptoms are present. This is particularly true when the trauma to the neck is of a blunt nonpenetrating type, and the neurologic manifestations simulating severe craniocerebral injury are not recognized as arising from thrombosis of the internal carotid artery. We wish to report 2 such cases recently encountered which demonstrate the challenge that this type of lesion presents to the physician in diagnosis and treatment.

While only an occasional case of thrombosis of the carotid artery secondary to nonpenetrating injuries of the neck has been reported in the literature, the presence of embolism or thrombosis associated with penetrating wounds of the neck or cheek has been more frequently recognized, especially in war wounds of the last century. In addition, since cerebral angiography has been used more widely in the diagnosis of neurological abnormality, certain patients with no apparent trauma are found to have a spontaneous carotid artery thrombosis.

Verneuil (1872) was probably the first to report a case of thrombosis following a nonpenetrating injury of the neck:

His patient had been found under a railroad car in an agitated and shock-like state. Examination disclosed a contusion to the vertex of the skull, ecchymosis of the groin, and a 5–6 cm. perineal laceration. During the next 24 hours a right hemiplegia and profound coma developed, and the “contour of the left sternocleidomastoid muscle was effaced by a swelling.” A diagnosis of hemorrhage of the brain was made. Postmortem examination 5 days after the injury demonstrated the following findings. The brain appeared grossly normal. Thrombus formation in the left internal carotid artery was found about 2 cm. distal to the bifurcation of the common carotid artery. While the external wall of the vessel was intact, the intima and the media at this point were torn so that their edges were rolled under, forming a nidus for thrombus formation. The thrombosis extended into the most peripheral branches of the middle cerebral artery. Cut section of the brain revealed softening of the left cerebral hemisphere. Verneuil postulated that there had been a sudden wrenching of the neck with no blow directly to the artery because there was no contusion or hematoma. He emphasized the importance of the differential diagnosis “between diverse accidents that can follow traumatic lesions of the head.”
In 1935, Greco\textsuperscript{11} reviewed the literature on injuries to the carotid artery in the neck with thrombosis, and listed 22 cases in which this phenomenon occurred secondary to penetrating wounds of the face and neck. To these he added another case of thrombosis of the internal carotid artery that followed a nonpenetrating injury to the neck:

While riding his bicycle, a 23-year-old male was struck by a horse-drawn carriage which knocked him down, but he did not lose consciousness. He was dazed and had no recollection how the trauma had occurred. Although the patient was able to continue his bicycle ride, shortly after the episode he had transient generalized malaise and blurring of vision. These symptoms subsided only to recur again an hour later with headache and vomiting; later he became unconscious. Bradycardia and a right hemiplegia developed about 16 hours after the accident. Examination revealed superficial abrasions over the left chin and lower lip. No anisocoria was noted; the right cremasteric and right patellar reflexes were absent, but a right extensor planter reflex was elicited. Palpation of the carotid vessels in the neck was not performed. Lumbar puncture was normal. A diagnosis of possible left middle meningeal hemorrhage was made. Left temporoparietal craniotomy revealed no abnormality except minimal pulsation of underlying brain. Postmortem examination 60 hours after injury disclosed encephalomalacia of the left cerebral hemisphere. A small hematoma was found over the left carotid sheath just to the left of the thyroid cartilage. No extravasation of blood into the neck was seen. At the origin of the internal carotid artery a linear transverse laceration was noted which had involved the intimal and medial layers of the vessel and there was retraction of their margins for a distance of 8 mm. Strongly adherent thrombus at this site had greatly reduced the lumen of the vessel, and at the level of the foramen lacerum the vessel was completely occluded by this process. The thrombus extended into the branches of the internal carotid artery.

Moniz,\textsuperscript{19} in 1941, presented a case of severe trauma to the head which was so marked that traction on the carotid artery apparently tore the intima and resulted in a thrombosis to the vessel.

In 1944 Northcroft and Morgan\textsuperscript{22} presented a case of traumatic thrombosis:

A young soldier was walking along the road when a lorry passed him. A loose rope hanging from the vehicle wound around the patient’s neck, throwing him to the ground, and then rapidly unwound itself without dragging the patient. He did not lose consciousness, and was able to walk to a nearby dispensary where a single stitch was placed in a superficial scalp laceration in the right parietal area. A contusion was observed on the left side of his neck. On the following day the swelling in the left sternocleidomastoid area was larger; right Jacksonian seizures and a right hemiplegia developed. Roentgenograms of the skull showed no fracture. A left extradural hemorrhage was considered. A burr hole in the left side of the skull revealed only “healthy brain.” The left lateral ventricle was tapped and the pressure was normal. The patient died 19 hours after operation. Postmortem examination revealed external marks of the rope on the neck. Two-thirds of the left sternocleidomastoid muscle was found to be ruptured. The left internal carotid artery contained a thrombus which extended into the middle cerebral, part of the left anterior cerebral, and the left posterior communicating arteries.
TRAUMATIC INTERNAL CAROTID ARTERY THROMBOSIS

There is some question whether Case V reported by Erikson\textsuperscript{10} should be included in this group or be classified under spontaneous thrombosis of the internal carotid artery with coincidental head trauma:

For several years a 28-year-old male had suffered from periodic headaches. He sustained a blow to the head which was followed in a few hours by increasing apathy and a flaccid left-sided hemiplegia. Right carotid angiography demonstrated obstruction in the internal carotid artery just below the base of the skull. Subsequent postmortem examination revealed an adherent thrombus, 2 cm. long, in the internal carotid artery at this point. Distal to this point was a thrombus, 3 cm. in length and reddish gray in color, lying free from the vessel wall. A large cerebral infarct was found in the right cerebral hemisphere.

Closely associated with nonpenetrating wounds of the neck are minor lacerations with carotid thrombosis as illustrated by Case 4 of Caldwell and Hadden.\textsuperscript{2}

A 20-year-old male was injured when his truck overturned on an ice-covered highway. Following a lucid interval he became comatose 4 hours after injury. Examination disclosed a (1) compound comminuted fracture of the right mandible near the angle, (2) simple fracture of the left clavicle, (3) superficial laceration of the anterior triangle in the right neck. Five hours following injury the jaw fracture was reduced. Skull roentgenograms showed no fracture. The left eyelid was ptosed and there was twitching in the left facial muscles. The left leg and arm were paralyzed.

Exploratory trephines revealed normal dura and cortex. Thirty-four hours after injury the patient died. Autopsy revealed a thrombosis of the right common, external and internal carotid arteries.

CASE REPORTS

We would like to report 2 cases of internal carotid artery thrombosis which occurred secondary to nonpenetrating injuries to the neck and simulated epidural or subdural hematoma.

\textit{Case 1.} L.O., a 42-year-old right-handed white female, was involved in an auto accident at 10:00 a.m. on March 19, 1951. She was thrown out of the car as it overturned; the vehicle struck the right side of her head. It was not possible to ascertain whether she had been unconscious; however, on arrival at a local hospital she was conscious and it is alleged that she was able to move her left upper extremity but not the lower extremity. When she became progressively more lethargic, she was referred to the University Hospital 12 hours later with a diagnosis of “acute head injury.”

\textit{Examination} disclosed a B.P. of 120/70, respirations of 28, and pulse rate of 120. Abrasions and contusions were observed lateral to the right orbit, in the right supraclavicular region, and anteriorly over the right chest. There was a palpable fracture of the right clavicle. There was dullness to percussion over the left chest and breath sounds were reduced over the left base. The patient was lethargic but was well oriented. The miotic pupils were equal. There was forced conjugate deviation of the eyes toward the right (Fig. 1) with inability to move the eyes to the left of the midline. During the 12 hours following injury a complete left hemiplegia
had developed with a left extensor plantar reflex. Roentgenograms of the skull showed no fracture. Further roentgenography showed fractures of three ribs of the right chest, and fractures of the right clavicle (Fig. 2) and the pelvis. There was a right pneumonitis with hemothorax. Urinalysis showed no abnormality.

Ophthalmodynamometric study revealed the retinal artery pressures to be 25/40 O.D. and 65/115 O.S. Lumbar puncture disclosed a pressure of 180 mm. of water, 5 WBC, and a total protein of 80 mg./100 cc.

Course. A right stellate block with 10 cc. of 2 per cent procaine was performed with no change in her hemiplegia.

On the following day open arteriography demonstrated an occlusion of the right internal carotid artery 3 cm. distal to the bifurcation of the common carotid. There was reflux filling of the right vertebral artery during the injection (Fig. 3). Definite transmitted pulsations were observed in the exposed segment of the internal carotid artery. Superior cervical sympathectomy was performed at the time of angiography with excision of 3–4 cm. of the chain and ganglia.

Five days after the angiography conjugate deviation of the eyes to the left of the midline was possible. Three months later marked emotional lability, left hemiplegia with a left extensor plantar reflex, and left homonymous hemianopsia were present.

Comment. The differential diagnosis in this case rested between an expanding extra- or subdural hematoma, a right frontal intracerebral hematoma, cerebral fat embolism to the smaller vessels of the right frontal lobe with infarction, and thrombosis of the right internal carotid artery. Dis-

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**Fig. 1. Case 1.** There is conjugate deviation to the right and a right facial weakness. The dressing covers the incision made for the right carotid arteriography. Contusion over the right clavicle is not readily visualized.

**Fig. 2. Case 1.** The site of fracture of the right clavicle is demonstrated.
proportion between the patient's alertness and orientation and severe neurological deficit did not suggest an intracranial hemorrhage. Her vital signs and her state of consciousness did not indicate the presence of a rapidly expanding intracranial lesion. The diagnosis of fat embolism could not be established. The diagnosis of internal carotid thrombosis was made clinically because of the right supraclavicular contusion, the relatively alert state of mind initially compared to the degree of neurologic disability, and the differences in the retinal artery pressures. The diagnosis was confirmed by carotid angiography.

Case 2. P.H., a 24-year-old right-handed white male, was injured in an automobile accident at 1:00 A.M. on Nov. 23, 1951. He was semiconscious when first examined, and a left hemiplegia developed with deepening coma during the next 36 hours. He was admitted to University Hospital at 3:00 P.M. on Nov. 24, 1951.

Examination revealed a B.P. of 195/100, pulse rate of 62–84, and respiratory rate of 20 per min., irregular. There were multiple abrasions of the right side of the neck, face, and forehead. The right lower extremity rested in a Thomas splint with marked swelling of the middle third of the thigh. On auscultation there were a moderate number of extrasystoles, and moist rales were heard at both lung bases. The patient was semicomatose but responded to painful stimuli with movement of the right side of his body, and only slight movement of the left. There was hypertonicity with hyperactivity of deep reflexes on the left. Abdominal and cremasteric reflexes were absent on the left, and extensor plantar reflexes were elicited bilater-
ally. The right pupil was larger than the left, but both reacted to light. Funduscopic examination revealed blurred disc margins bilaterally with distension of the retinal veins. The carotid pulse was palpable bilaterally in the neck.

Roentgenograms of the skull displayed no fracture, but there was a fracture of the middle third of the right femur. Urinalysis revealed a trace of albumin and many fat particles by staining techniques. A tentative diagnosis of right extradural hematoma was made.

**Course.** Burr holes were made over the right temporal, frontal, and posterior parietal regions. Upon incising the dura there was a herniation of the brain at each burr hole. An unsuccessful attempt was made to tap the right ventricle from the posterior parietal trephine opening. Another burr hole was therefore made at the homologous site on the left side; upon tapping the left ventricle the cerebrospinal fluid escaped under markedly increased pressure. Ventriculogram showed a shift of the left lateral and 3rd ventricles from right to left; there was no air visible in the right lateral ventricle (Fig. 4). Right percutaneous carotid arteriography on three injections demonstrated a thrombosis of the internal carotid artery about 3 cm. above the bifurcation of the common carotid (Fig. 5), with filling of the external carotid artery. Because of the pronounced cerebral edema a Penfield type of subtemporal decompression was done on the right and a Cushing procedure on the left. Upon incising the dura there was a marked herniation of the brain through the dural openings. A Kirschner wire was placed through the right tibia to immobilize the fractured right femur. A polyethylene catheter was inserted under the carotid sheath and a continuous procaine block of the cervical sympathetic chain was carried out using 1 per cent xylocaine.

Prior to operation multiple particles identified as peanuts were recovered by intratracheal suction, and thick mucopurulent material was aspirated frequently postoperatively. For 12 hours after operation the blood pressure remained at 120/90 and then gradually fell to shock levels despite all attempts to maintain it with blood and vasopressor agents. The pulse continued to be rapid and thready. The temperature rose to 105°F. and the patient succumbed at 8:00 P.M. on November 25.

**Postmortem Examination.** There were numerous abrasions on the forehead over the orbits, below the left eye, and over the bridge of the nose. Moderate swelling was present on the right side of the neck beneath the mandible. There was an extravasation of blood into the right sternocleidomastoid muscle, and around the carotid sheath of the common carotid artery in the region of the bifurcation. The needle puncture of the previous arteriography was 4 cm. proximal to the lowermost
point of the extravasation. The right carotid artery was occluded just proximal to its bifurcation by thrombus (Fig. 6). This thrombus extended into the anterior, middle and posterior communicating arteries. Convolutions of both cerebral hemispheres were flattened and hyperemic with more pronounced swelling and marked ecchymosis over the right frontoparietotemporal cortex (Fig. 7). Distinct right tentorial and cerebellar pressure cones were found. Section of the brain showed diffuse softening of both gray and white matter with multiple infarcted areas in the right cerebral hemisphere. An 80 per cent atelectasis was observed in the right lung. Crepitus was palpable over the right femur.

Comment. There are several important lessons to be learned from this case. The patient presented the clinical picture of acute increased intracranial pressure. There was the failure primarily to consider thrombosis of the internal carotid artery in the differential diagnosis. An expanding intracranial lesion was suggested by the dilated right pupil and the bilateral extensor plantar reflexes indicating the formation of a tentorial pressure cone. Since no surface lesion could be demonstrated by burr holes and no intracerebral hematoma in the right hemisphere could be localized by the ventriculogram, an arteriogram was made thereby demonstrating the thrombosis
of the internal carotid artery. Diagnosis in this instance could be made only by the dye study, since there was no demonstrable change to palpation of the carotid pulse in the neck.

The marked cerebral edema could have been due to cerebral hypoxia secondary to the thrombosis and an almost complete atelectasis of the right lung. Continuous stellate block was employed to promote the collateral arterial supply and increase oxygenation of the hemisphere. In retrospect an

Fig. 6. Case 2. Postmortem specimen showing the severe edema and ecchymosis of the right cerebral hemisphere. See Fig. 7 for magnification of outlined area.

early bronchoscopy and tracheotomy probably would have been of aid in maintaining better aeration of the lung, and might have increased the patient’s chance of survival.

PATHOLOGY

Legal limitation in the autopsy examination prevented microscopic examination of the bifurcation region of the common carotid artery. Studies of Verneuil,\textsuperscript{33} Greco,\textsuperscript{11} and Caldwell and Hadden\textsuperscript{2} showed that there was a tear
in the intima with associated damage to the media and a curling up of these layers to form a nidus for thrombus formation. The first author believed that this was due to a wrenching movement of the neck with no direct trauma to the vessel, but Greco regarded the blow to the vessel as the etiological factor. Arteriosclerotic changes may be contributing factors to forma-

Fig. 7. Case 2. Magnification of outlined area in Fig. 6. RIC, right internal carotid artery. LIC, left internal carotid artery. RON and LON, right and left olfactory nerves. OC, optic chiasm. The thrombus is seen in the right carotid artery.

tion and propagation of thrombus. Associated with injury to, or near, large arteries is spasm involving some part of the artery or entire arterial beds. Spasm in the artery as well as in the arterial bed is usually caused by abnormal reflexes since extensive arterial thrombosis is not a common finding in the initial stage of this syndrome.

Ecker\(^7,8\) has described a syndrome of spasm of the internal carotid artery. It consists of: (1) dysfunction of a cerebral hemisphere, (2) brain swelling in the acute stage, (3) internal hydrocephalus in a late stage and (4) narrowing of the cerebral arteries. He presented 4 cases which illustrate the association of this syndrome with nonpenetrating wounds of the neck and blunt injuries of the head.

**DIAGNOSIS**

If there is a penetrating wound of the neck, traumatic internal carotid artery thrombosis is more likely to be recognized, but if there is merely a
contusion with a minor degree of swelling and ecchymosis in the supraclavicular fossa this diagnosis may be overlooked. In patients who present clinical signs and symptoms simulating those of acute head injuries the diagnosis of thrombosis of the internal carotid artery may be suspected when a combination of neurologic signs and trauma to the neck are observed.

In the majority of cases of acute thrombosis of the internal carotid artery the signs and symptoms may develop within a period of 6 to 24 hours,\(^1\) as contrasted to the longer interval for spontaneous thrombosis. The clinical manifestations of these lesions depend upon several factors: (1) the extent of the thrombosis, (2) the competency of circulation in the circle of Willis, (3) the presence of vasospasm, (4) the presence of the lesion in the dominant or minor hemisphere, (5) the severity of associated cranioencephalic injury, (6) the degree of secondary hypoxia. These may result in alterations in the state of consciousness, hemiplegia, aphasia, blindness, visual field defects, and Horner’s syndrome.\(^2,3\)

If the patient is cooperative the measurement of the retinal artery pressure by Baillart’s ophthalmodynamometer may give a definite indication of the presence of this lesion.\(^1,5,31\) Using this technique the first pulsation of the retinal artery as observed by funduscopic examination with pressure on the globe corresponds with the diastolic retinal pressure (35–40 mm.) and the last beat with the systolic pressure (70–75 mm.). In thrombosis of the internal carotid artery the diastolic and systolic pressures are reduced when compared to the normal eye. Since the accuracy depends upon the cooperation of the patient this procedure is of limited value in an agitated or irrational individual.

Caldwell and Hadden\(^2\) have indicated that “absence of the temporal pulse on the involved side is good confirmatory evidence that the common carotid and possibly the external carotid artery is occluded.” However, palpation of the vessels in the neck is of no value in diagnosis of the process in the internal carotid artery. In the occluded artery, pulsations may be transmitted to the thrombosed segment from the more patent portion.

The presence of increased intracranial pressure with thrombosis of the carotid artery is now well recognized. Caldwell and Hadden,\(^3\) in their review of the literature on thrombosis secondary to penetrating wounds of the carotid artery, noted a definite elevation of cerebrospinal fluid pressure on lumbar puncture. Riechert\(^25\) and Moniz et al.\(^20\) observed papilledema in cases of spontaneous thrombosis of the carotid artery suggesting increased intracranial pressure. The development of unilateral cerebral edema was recognized in a shift of the ventricular system toward the contralateral side in 3 cases 3, 6 and 12 days after the onset of signs of thrombosis by Elvidge and Werner\(^9\) with confirmation by Raney\(^24\) and Caldwell and Hadden\(^3\) in autopsy material. In spite of subtemporal decompression, this increased intracranial pressure may be so marked as to produce definite temporal and cerebellar pressure cones, as illustrated in Case 2.

Angiography presents the most accurate method of diagnosing throm-
bosis of the internal carotid artery during life. Johnson and Walker have emphasized that if percutaneous arteriography is used the angiogram should be taken in such a fashion that the bifurcation of the common carotid artery is shown, so that one can observe the extent of filling of both the external and internal branches. We believe that it is also advisable to include the needle tip in the angiogram in the percutaneous method, in order to rule out faulty injection technique, such as injection into the external carotid artery or extra-arterial tissues. If the diagnosis of carotid thrombosis is reasonably certain it is better to use the open method of arteriography. This allows direct visualization of the internal carotid artery permitting more accurate injection, enables an estimate of the degree of injury to the vessel and surrounding tissues, and, if thrombosis is demonstrated, cervical sympathectomy may be done immediately.

Although bilateral angiography in these cases of occlusion of the carotid artery may demonstrate the collateral circulation through the anterior communicating and anterior cerebral arteries this appears to be definitely contraindicated in the traumatic group. The risk of such a procedure is recognized in spontaneous carotid artery thrombosis, and it is no doubt more dangerous in acute occlusion where the collateral circulation has probably been placed on a more acute strain.

The problem of the differential diagnosis between an expanding intracranial hematoma and internal carotid artery thrombosis may be a difficult one. If there is progressive impairment of the state of consciousness, or there are signs of a tentorial pressure cone or changes in the vital signs suggesting increased intracranial pressure, burr holes are indicated under local anesthesia to rule out the possibility of an acute subdural or extradural hematoma. If no pathological condition is found and the brain shows signs of being under increased pressure, a ventriculogram may disclose signs of unilateral hemispheric lesion. Angiography will permit differentiation between carotid artery thrombosis and an intracerebral lesion.

TREATMENT

Prompt diagnosis of arterial thrombosis with as little delay as possible in instituting treatment should be the goal of every surgeon. Constant observation of all patients with suspected arterial injury and immediate institution of therapy are factors of paramount importance to success in treatment of this lesion. If the diagnosis of thrombosis of the internal carotid artery can be made early by percutaneous arteriography, anticoagulant therapy may be of value in controlling the progression of the intravascular clot, but it will not affect the thrombosis that has already taken place. If the clotting of blood is progressive it will reduce the efficiency of collateral arterial circulation by actual extension into the collateral arteries. Treatment in these cases should be directed toward increasing the blood flow through the collateral channels, which involves overcoming the reflex vasoconstriction distal to the site of thrombosis. This may be attempted by continuous cervical sympa-
thetic blocks administered by inserting a polyethylene catheter into the region of the sympathetic chain as suggested by Poppen. Cervical sympathectomy is more permanent and probably more satisfactory. Papaverine as a supplementary vasodilator should be administered.

Because the thrombosed vessel is considered to cause a reflex spasm of the distal vessels, excision of the thrombosed vessel has been performed on several occasions. In their review on spontaneous internal carotid artery thrombosis Johnson and Walker stated that this procedure was used in 28 cases in the literature with improvement in 7 patients. However, they question the advisability of the procedure, for recanalization of the thrombosed carotid artery has been known to occur.

In those cases in which thrombosis has been extensive and signs of increased pressure and cerebral edema are present, early bilateral subtemporal decompression may be of some benefit in preventing pressure on the medullary and midbrain centers by prevention of cerebellar and temporal pressure cones. In the unconscious patient cerebral edema may be reduced by maintaining an adequate airway by intratracheal suction or trachetomy. Supplementary oxygen will afford a better chance of adequate oxygenation of cerebral tissue.

**SUMMARY**

Traumatic internal carotid artery thrombosis secondary to nonpenetrating injuries to the neck should be considered in the differential diagnosis of cranio-cerebral trauma. Five cases of concomitant head injury and traumatic internal carotid artery thrombosis of this type were found in the literature. These have been described and we have added 2 similar cases. The diagnosis and treatment of this lesion have been presented in detail.

**REFERENCES**

12. HUNT, J. R. The role of the carotid arteries, in the causation of vascular lesions of the brain, with remarks on certain special features of the symptomatology. Amer. J. med. Sci., 1914, n.s. 147: 704-713.