

TRAUMATIC THROMBOSIS OF THE INTERNAL CAROTID ARTERY

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Obstruction of the internal carotid artery by thrombus has been recognized with increasing frequency in recent years, because both carotid arteriography and inspection of this vessel post mortem have been done more often. Thrombosis following injury to the neck is well recognized and was described after both world wars, but in nearly all these cases there was laceration of the neck and an open wound in relation to the artery. But occasionally carotid thrombosis occurs after trauma which has not broken the skin of the neck, and it is with such cases of closed traumatic thrombosis of the internal carotid artery that this article is concerned. These have been regarded as rarities, and a rough idea of their frequency comes from Greco (1935) who recognized one such case whereas he saw 22 with open wounds, and from Caldwell and Hadden (1948) who described one case of carotid thrombosis due to closed trauma and eight with open wounds among some 25,000 wounded soldiers admitted to an American army hospital. Murray (1957) recently made a survey of the world literature and listed 10 cases from wounds of the neck, excluding cases such as Sedzimir (1955) described in which the thrombosis originated in the intracranial part of the internal carotid artery and was associated with a fracture of the skull base.

In addition to these analysed by Murray (1957), cases have been described by Cairns (1942), who also referred to another recognized by Zannoni (1933), by Sedzimir (1955) quoting an Australian case, and by Rosegay (1956) who describes one in America. Two further cases are now added, one recently seen in the Military Hospital, Wheatley, and the other associated with that hospital three years ago when the patient was sent to Oxford for rehabilitation after initial diagnosis at Cardiff.

Of these six cases, five survived, while in Murray's paper (1957) nine out of 10 died. The increasing use of arteriography makes it probable that many more such favourable cases will be recognized. The

six patients who survived, one mentioned by Murray (1957) and five additional to his list, are briefly described.

Case Reports

Case 1 (Schneider and Lemmen, 1952).—A 42-year-old woman sustained a fracture of the right clavicle with contusion of the right supraclavicular area in a car accident. She was normally conscious on arrival at the local hospital but was unable to move her left leg although the left arm was normal. She soon became lethargic and 12 hours later was semiconscious with a left hemiplegia and failure of conjugate eye movement to the left. Next day arteriography showed a block of the right internal carotid 3 cm. above its origin. Three months later she had a left hemiplegia, a left homonymous hemianopia, and was markedly labile emotionally.

Case 2 (Cairns, 1942).—A 23-year-old airman was knocked unconscious for perhaps three minutes in a crash landing. On arrival at hospital he could give a clear account of the accident but in a voice already so husky that, together with his difficulty in swallowing and breathing, a tracheotomy set was prepared although never used. There were superficial grazes in the left carotid triangle and a laceration, bleeding freely, on the right. However, it was on the former side that the damage occurred, for, about 30 hours later, despite apparent improvement, he suddenly became aphasic with paralysis of the right side of the face and of the right arm and with weakness of that leg. Craniotomy and air encephalography were both normal although six weeks later this latter investigation, on repetition, showed some dilatation of the left lateral ventricle. Thirty-eight days after the injury arteriography showed a block of the left internal carotid artery 2 cm. from its origin. One month later there was gross global dysphasia, a homonymous field defect in the right upper quadrant, right hemihypaesthesia, and some slight recovery of movement of the limbs on the right.

Case 3 (Sedzimir, 1955).—A 32-year-old man also improved over the year after his car accident so that he was then able to walk with an appliance although his left arm was useless. He had occasional grand mal attacks. His injuries had all been thought to be frontal and, again, loss of consciousness at the time of the accident was brief before he walked half a mile for help. Rational on entering hospital, he became unconscious

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with the development of a left hemiplegia over a few hours during the next day. Angiography, on the basis of a diagnosis of subdural haematoma, showed a low complete obstruction of the right internal carotid.

Case 4 (Wheatley Hospital No. N 2092).—A 20-year-old soldier was knocked out in a dance-hall brawl. There were small cuts on the lower lip and the left side of the chin while there was also slight swelling of the left side of the neck, although it never felt stiff. He was unconscious on admission to hospital and did not regain consciousness for some 12 hours when he was unable to speak or to write. He also had a marked right facial palsy and some weakness of the right hand, but not to a degree that could explain the agraphia. This hand and his writing showed most improvement so that, although he began to say words 10 days after the injury, 10 weeks after the accident there was still considerable dysphasia, but no nominal dysphasia or apparent defect of internal speech, together with a right facial weakness. Six weeks after the accident the fields were normal. Arteriography at this time showed a proximal obstruction of the left internal carotid.

The last two cases both show carotid obstruction distally, recalling the cases of Sedzimir (1955) but without any evidence of skull fracture such as he found.

Case 5 (Rosegay, 1956).—A 29-year-old man was knocked out in a fight but soon came round to notice weakness and numbness of the left arm and leg which progressed so that next day there was a severe left spastic hemiparesis. At a stage when recovery had already begun in the leg, arteriography showed a block of the right internal carotid artery just distal to the origin of the posterior communicating artery. The leg continued to improve but the hand remained virtually without function. This case again showed no delay between the trauma and the onset of neurological deficit.

Case 6 (Wheatley Hospital No. A 25187).—A 21-year-old professional boxer lost a fight on points but did not receive particularly severe punishment or any noticeable injury to his neck. About three-quarters of an hour later, he vomited and then rapidly developed motor aphasia and a right hemiparesis. He came under the care of Mr. C. Langmaid, at Cardiff, and arteriography showed the obstruction again to be high in the left internal carotid, at the base of the skull. Recovery in the right leg and in speech had begun 10 days after the fight while six weeks later, when he came to Oxford for rehabilitation, there was only mild nominal dysphasia, slow speech, a moderate right facial weakness, severe spastic paralysis of the arm, a moderate spastic paresis of the leg (but not severe enough to need a toe-spring), and a mild left hemianaesthesia, but no visual loss. Three years later, he is completely independent but there is still almost complete lack of movement in the right forearm and hand. Interestingly, in view of the transitory aphasia, he is a left-handed man with a history of left-handedness in the family.

Discussion

These six surviving cases may be grouped with the other cases of closed traumatic thrombosis of the internal carotid artery listed by Murray (1957) or mentioned earlier in the paper (Zannoni, 1933), so that there are 16 cases for discussion.

The youngest patient was 16 and the oldest 45 years of age, while 10 were aged 20 to 30 years. This age group is probably more liable to trauma than are others, but the incidence is nevertheless remarkably high. As might be expected on similar grounds, males form the bulk of the patients.

The initial injury is not usually severe and later examination shows only some abrasions of the face or neck, contusion or mild swelling of the neck, or, occasionally, a fracture of the jaw, clavicle, or first rib (in one instance each). The cause may be an accident while travelling, a blow in a fight or from a flying missile, or, in one bizarre case (Northcroft and Morgan, 1944) the embrace of a rope which trailed from a passing lorry to wrap itself round a pedestrian's neck, throw him to the ground, and then unwind again. This initial injury might cause unconsciousness for a few minutes but in most cases the patient was regarded at first as having sustained only minor injuries, and, indeed, might walk some distance to a hospital for an abrasion to be dressed, or merely go straight home. The onset of symptoms is most commonly in the first six hours but may occur some 30 hours after the accident, as shown in Table I.

TABLE I
ONSET OF SYMPTOMS AFTER ACCIDENT

Time after accident (hr.) Number of cases	Immediate 2	0-6 6	6-12 3	12-24 3	24-36 2
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Two cases were unusual, for one patient (Case 4) was unconscious for 12 hours after the accident when he recovered with signs of left cerebral dysfunction while the other (Case 5) had weakness and numbness of the left arm immediately he recovered from a few minutes of unconsciousness. Clarke and Harris (1958) write that an interval between the trauma and the development of altered consciousness is a constant feature of traumatic carotid thrombosis. In Case 4 the 12 hours of unconsciousness may have resulted from a cause other than the blockage of an internal carotid artery, which lesion could have developed during that period to be responsible at its end for aphasia, agraphia, and palsy of the right face and hand. And in Case 5, it was malfunction of the left arm that was present and progressive, for there was no period of disturbed consciousness apart from a few minutes immediately after the accident. Both these patients survived.

There seem two possible sites of obstruction. Of 10 cases with adequate necropsy reports, nine showed damage to the intima and usually the media of the artery in the 2 to 3 cm. above the bifurcation of the common carotid with corresponding thrombus formation, while in one extravasation of blood into the carotid sheath at this level was the most prominent feature apart from the thrombus itself. In the six survivors, where there is only arteriographic evidence, the block was near the origin of the internal carotid in four and high in its course in two, where it was described respectively as at the base of the skull (Case 6) and just distal to the origin of the posterior communicating artery (Case 5).

Obstruction low in the internal carotid does not itself indicate where the thrombus originated, for spread may be retrograde, but, as just mentioned, in nearly all the necropsy cases there was clearly intimal damage near the proximal end of the clot. Indeed, distal spread is the rule for, of 10 cases, spread to the beginning of the middle cerebral artery had occurred in nine while in five of them the anterior cerebral artery was also involved. Occasionally thrombus was also seen in the posterior cerebral or even basilar artery. The effect upon cerebral nourishment of the spread of a thrombus to break the continuity of the circle of Willis and perhaps extend along a cerebral artery must be considerable. If extension blocks the middle cerebral artery alone the anterior cerebral artery could still be fed from the opposite side. Of 16 cases considered, motor involvement of the arm and leg seemed equally severe in 10, in two the leg was worse than the arm, and in four the arm was the worse. Not surprisingly, of these six patients with more partial lesions, five were survivors so that clinical and post-mortem findings cannot be compared. In the one exception (Clarke, Dickson, and Smith, 1955), where the arm was affected almost 24 hours before the leg, the thrombus was found at necropsy to extend to the origin of the middle and anterior cerebral arteries. Sedzimir (1955) describes an interesting case where thrombotic obstruction of the internal carotid artery, originating from the region where it passes upwards from the cavernous sinus, and associated with rhinorrhoea and a fracture of the cribriform plate, caused paresis of the right arm several hours before coma occurred and some days before the leg was paralysed. Here again the thrombus extended only to the point of bifurcation of the internal carotid artery.

The survivors show no special features to distinguish them from those who died, either in the nature of the accident, the severity of apparent damage at an early stage, or the time before neuro-

logical symptoms appeared. They do include one case (Cairns, 1942) where a cerebral deficit appeared suddenly and as late as 30 hours after the original injury. Cairns felt that distal embolism from a carotid clot accounted for the sudden onset in this young pilot of hemiplegia and aphasia and supported this view by reference to the case described by Zannoni (1933) where at necropsy there was a slightly adherent clot in the middle cerebral artery not in continuity with clot firmly adherent at the proximal end of the internal carotid artery.

The severity of the residual damage in these cases varies from a right facial weakness with a dysphasia not severe enough to prevent the patient from conveying his meaning (Case 4) to a man with dysphasia, hemiplegia, hemihypaesthesia, and an upper quadrantic homonymous field defect (Case 2). In only one of the six were grand mal attacks a sequel (Case 3).

The cases presented here may be contrasted with two described by Sedzimir (1955) in which the distal end of the internal carotid artery was occluded after trauma, which also caused fracture of the cribriform plate on one side and rhinorrhoea. In these two patients, aged 54 and 42, respectively, there was a long period of unconsciousness immediately following the injury (five days and continuous to death). The onset of focal signs came relatively late, on the seventh day in the first case and after two days in the other. Both these patients died. The rare cases in which injury occurs through the soft palate (Fairburn, 1957) have not been considered here.

Summary

Two further cases of thrombosis of the internal carotid artery after non-penetrating injury are described. They both survived and are grouped with four other surviving cases to contrast with a recent list in which nine out of 10 died. Some features of the lesion are discussed.

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