

# Closed cervicocranial trauma, "false aneurysm" of the internal carotid artery and brain infarction

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**ABSTRACT** - A 17-year-old man developed acute hemiparesis 6 months after a motor cycle accident. In the accident he had a closed trauma on the contralateral side of the head and the neck, with multiple bone fractures. Aortocervical angiography, performed after the infarction, revealed a 2.5 cm long aneurysmatic dilatation in the internal carotid artery, the presumably source of embolic infarction. This and the 24 other cases gathered from the literature support the notion that closed neck trauma may create "false aneurysm" which again may cause neurological deficits.

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Aneurysms are relatively rare in the internal carotid artery (1-5) but somewhat more frequent in the common carotid and external carotid artery (3). Lacerating neck trauma appears to be a logical and rather frequent cause of carotid artery aneurysm (2, 6, 7), whereas a closed cervicocranial trauma is only seldom recognized in the previous history (6-8).

We describe a case where a cerebral infarct developed in a 17-year-old man, 6 months after a road accident. Similar cases with the association of closed trauma and internal carotid artery aneurysms are reviewed from the literature.

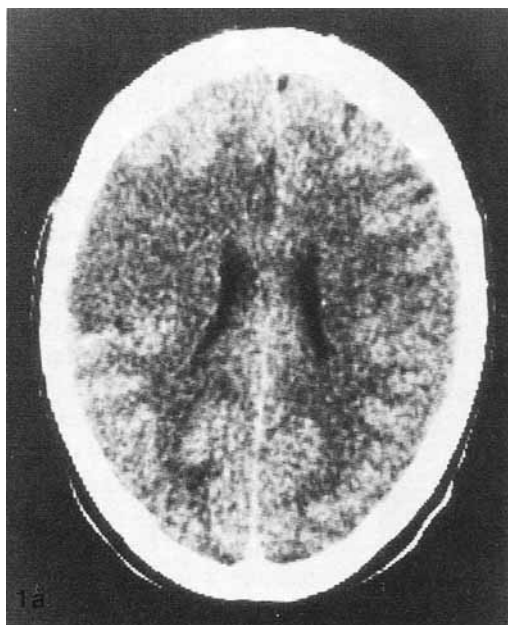
## Case report

A healthy 17-year-old man crashed his motor-cycle off the road in May 1985. During the accident he wore a helmet strapped under his chin. He was conscious when found and had no neurological deficit on examination. He had a dislocated fracture of the mandible with one tooth injured and 6 teeth injured in the maxilla, a dislocated fracture of the left humerus, a distension of the left acromioclavicular ligament

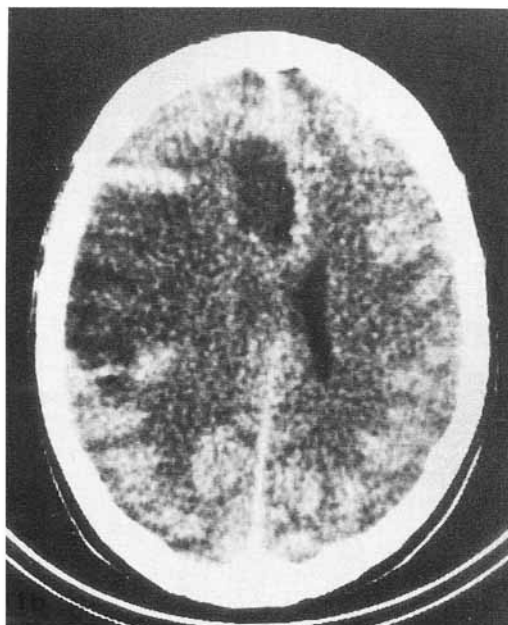
and a contusion of the left lung. He made a good recovery and was discharged from the hospital after 2 weeks.

Six months later he was admitted to the hospital with an acute leftsided hemiparesis. CT-scan showed an infarction (Fig. 1a-b). Aortocervical angiography revealed a 2.5 cm long aneurysmatic dilatation of the right internal carotid artery about 5 cm beyond the bifurcation (Fig. 2). Other branches of the aortic arch were normal.

Investigations of risk factors and other etiologies were unrewarding. There were no cerebral infarcts in the family history. The patient had smoked about 10 cigarettes per day, but for a period of 3 years only. His blood pressure, blood sugar, serum cholesterol and triglyceride values were normal. ECG, M-mode and 2-dimensional echocardiography and continuous monitoring of the heart rhythm gave normal results. Physical examination, radiological and laboratory investigations excluded Marfan's syndrome, Takayasu arteritis, homocystinuria, generalized infection including endocarditis, and other predisposing causes.



*Fig. 1a.* CT-scan on the day of arrival shows a slightly hypodense area of infarct at the territory of the right middle cerebral artery.



*Fig. 1b.* Two days later the infarcted area has become more hypodense and expansive.

Surgery was not considered possible and the patient was placed immediately on permanent anticoagulant therapy. His condition improved to some extent. One year after the infarction he still has a spastic hemiparesis. He is able to walk without support, but can use his left hand for grasping only.

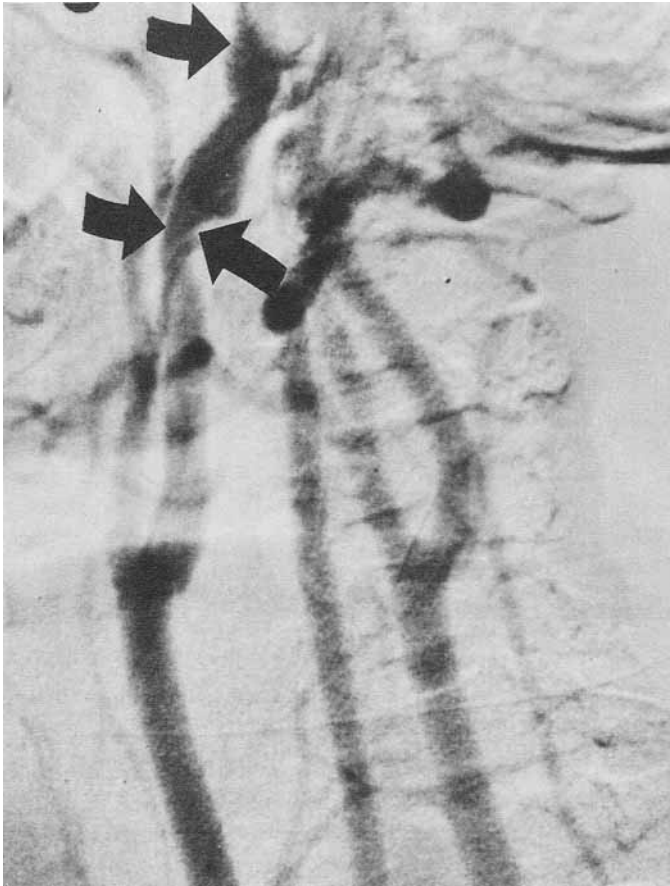
## Discussion

A dissecting aneurysm of the internal carotid artery was an obvious origin for an embolus that caused a cerebral infarct in our patient. To evaluate the possible association of the aneurysm and the previous trauma we reviewed the literature

Table 1  
Clinical findings in 24 cases of traumatic carotid artery aneurysms

Sex	men 19, women 5
Age	mean 31.2 yrs (range 15–65 yrs)
Type of the trauma	traffic accident 16 (hyperextension in at least 7) hyperextension of the neck 4, fall 3, direct blow to the neck 1
Localization of the lesions	head and neck 17, head and chest 1, head and trunk 3, trunk and extremities 3
Time latency between the trauma and the symptoms	immediate 11, hours 2, days 3, weeks 2, months 3, years 3
Primary symptoms and signs	unconscious or stuporous 9, amnesic, desorientated, confused 3, local signs (pain in the neck, a pulsatile mass, a Horner's syndrome, epistaxis) 8, signs of carotid ischemia 8
Angiographic findings	aneurysm in the left carotid artery 15, in the right carotid artery 7, aneurysm bilaterally 2; extracranially 22, intracranially 2
Therapy	operation 14, anticoagulant therapy 6, antiaggregative therapy 2
Outcome	dead 4, severe neurological deficit 2, minor neurological deficit 4, symptomfree 14
Follow-up time	mean 2.5 yrs (n = 14)

Clinical information obtained from the references<sup>1,4-12</sup>.



*Fig. 2.* An irregular aneurysmatic dilatation can be seen in the distal part of the right internal carotid artery (arrows). The lesion extends to the base of the skull (arrowhead) but the upper limit can not be depicted.

and found 24 cases where a closed trauma to the craniocervical region preceded the finding of an aneurysm in the internal carotid artery (1, 4–12) (Table 1). The pathogenesis is probably a stretch injury the vessels being compressed over the bony prominences of the atlas and the axis as well as over the styloid process during hyperextension and rotation of the head (5, 6, 8, 10–14). This type of stretch injury may be more likely in younger individuals, since the carotid artery becomes more tortuous with advancing age (14). Another suggested mechanism is impingement of the carotid artery between the angle of the mandible and the upper cervical transverse process. This may lead to a transmural crush damage (7). It may have been a contributing factor causing extra compression during the trauma. One previous case had a somewhat similar mechanism (1).

Most traumatic aneurysms are apparently “false aneurysms”, where the aneurysm is not made up of the 3 layers of the normal vessel wall, but only of the lining endothelium and organized clot giving rise to occlusion or thrombosis of the carotid artery (1, 2, 9, 13). In some cases the aneurysms have a saccular appearance. It is presumed that the pathogenic mechanism in these particular cases might be an acute crush damage with subsequent rupture and hemorrhage or vasospasm. The onset of clinical symptoms appears to be more acute in these cases (6, 7). It is, however, difficult to divide the traumatic aneurysms into definite subgroups since information on vascular pathology is available from a few cases only, and they may not be so representative for the whole clinical spectrum. We therefore gathered information from all the traumatic aneurysms

appearing in the extracranial portion of carotid arteries (Table 1).

In most cases local neurological signs or symptoms from the corresponding carotid territory develop within a few days from the trauma, but occasionally the time interval may be months or even years (7, 9–11, 13). Pain associated with a pulsatile mass in the neck and Horner's syndrome are suggestive local signs. Carotid bruit, however, is present only infrequently (7). We were unable to find any data of the value of Doppler ultrasound technique in the diagnosis. Aortocervical angiography therefore remains as a decisive diagnostic method. Traumatic aneurysms are actually difficult to distinguish from other aneurysms by angiography (6) except that they are usually located distally in the extracranial portion.

Recognition of a traumatic aneurysm is of considerable importance for several reasons. Surgery should always be considered and its long term results are usually excellent (4–6, 14). Some cases, like ours, was beyond surgical therapy because of the size and location of the aneurysm. These cases might benefit from long term anti-coagulant therapy. The diagnosis of traumatic carotid artery aneurysm also carries a great significance from the medicolegal point of view.

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