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# Traumatic internal carotid artery injuries: do we need a screening strategy? Literature review, case report and forensic evaluation.

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**Running head: Traumatic internal carotid artery injuries**

**Abstract**

Internal carotid artery dissection (ICAD) represents the cause of ictus cerebri in about 20% of all the cases of cerebral infarction among the young adult population. ICAD could involve both the extracranial and intracranial internal carotid artery (ICA). It could be spontaneous (SICAD) or traumatic (TICAD). It has been estimated that carotid injuries could complicate the 0,32% of cases of general blunt trauma and the percentage seems to be higher in cases of severe multiple trauma. TICAD is diagnosed when neurological symptoms have already occurred and it could have devastating consequences, from permanent neurological impairment to death. Thus, even if it is a rare condition, a prompt diagnosis is essential. There are no specific guidelines regarding TICAD screening. Nevertheless, TICAD should be taken into consideration when a young adult or middle-aged patient presents after severe blunt trauma. Understanding which kind of traumatic event is most associated with TICAD could help clinicians to direct their diagnostic process. Herein, a review of the literature concerning TICAD has been carried out to highlights its correlation with specific traumatic events. TICAD is mostly correlated to motor vehicle accidents (94/227), specifically to car accidents (39/94), and to direct or indirect head and cervical trauma (76/227). As well, a case report is presented to discuss TICAD forensic implications.

**Keywords**

Internal carotid artery dissection; trauma; diagnostic screening.

## Introduction

Internal carotid artery dissection (ICAD) occurs when the blood penetrates the arterial wall because of internal elastic lamina discontinuation. The collection of the blood between the tunica media and tunica adventitia could create a false lumen, also called pseudoaneurysm or false aneurysm. ICAD represents the cause of ictus cerebri in about 20% of cases of cerebral infarction among the young adult population [1, 2]. ICAD could be spontaneous (SICAD) or traumatic (TICAD). SICAD occurs in absence of a traumatic event and usually correlates to genetic syndromes, to recent infections, or to specific risk factors (i.e. hypertension, migraine, and hypercholesterolemia). Conversely, TICAD follows a traumatic event. Both extracranial and intracranial ICA could be involved. Usually, a direct or indirect cervical injury is described, and it often correlates to motor vehicle accidents [5-8]. The need for diagnostic screening for TICAD in cases of head and/or cervical injury is controversial [9]. The fact remains that TICAD is often misdiagnosed or diagnosed when neurological symptoms have already occurred [9,10]. As a consequence, it could lead to significant neurological permanent impairment. In blunt carotid injury, morbidity is estimated up to 80% and mortality up to 40% [10-12]. Therefore, it could have forensic consequences.

In this paper, a review of the literature concerning TICAD has been carried out to highlights its correlation with specific traumatic events. Besides, its clinical and medico-legal implications are investigated through the presentation of a case report.

## Methods

The present systematic review was carried out according to the Preferred Reporting Items for Systematic Review (PRISMA) standards [13]. A systematic literature search and a critical review of the collected studies were conducted. An electronic search of PubMed, Science Direct Scopus, Google Scholar, and Excerpta Medica Database (EMBASE) from database inception to November 2020 was performed. The search terms were “internal carotid artery”, dissection”, and “trauma” in the title, abstract, and keywords. The bibliographies of all located papers were examined and cross-referenced to further identify relevant literature. A methodological appraisal of each study was conducted according to the PRISMA standards, including an evaluation of bias. The data collection process included study selection and data extraction. Three researchers (RLR, PF, and MDP) independently examined the papers with titles or abstracts that appeared to be relevant and selected those that analysed traumatic internal carotid artery dissection with reference to type I, II, and III Biffl Vascular Injury grade (intimal flap, dissection, and pseudoaneurysm) [14]. Disagreements concerning eligibility among the researchers were resolved by consensus. Preprint articles were excluded. Only papers in English were included in the research. Data extraction was performed by two investigators

(AM, ACM) and verified by two other investigators (VF, ET). This study was exempt from institutional review board approval, as it did not involve human subjects.

**Results**

A review of the titles and abstracts as well as a manual search of the reference lists were carried out. The reference lists of all identified articles were reviewed to find missed literature. This search identified 254 articles, which were then screened based on their abstract. The resulting 128 reference lists were screened to exclude duplicates, which left 103 articles for further consideration. In addition, non-English papers were excluded, and the following inclusion criteria were used: (1) original research articles, (2) reviews and mini-reviews, and (3) case reports/series. These publications were carefully evaluated, taking into account the main aims of the review. This evaluation left 87 scientific papers comprising original research articles, case reports, and case series. Figure 1 illustrates our search strategy. Studies conducted before 1990 were excluded from the summarizing Table 1 but are briefly described below. All the remaining studies published from 1990 to nowadays are summed up in Table 1. In a few cases, complete data extraction was not possible. However, eligible data have been reported in Table 1.

The very first report of TICAD dates back to 1872 when Verneuil autopsied a person who died of head trauma [15, 16]. He found an intimal tear of the ICA and a thrombus in its lumen which was extended to the middle cerebral artery. Formerly in 1944, Northcroft and Morgan described the dissection of the left ICA occurred by accidental hanging [17]. In 1967, Yamada *et al.* investigated 51 cases of carotid artery occlusion due to blunt injury [18]. Then, a report of ICAD following a blunt head injury was published by Sullivan *et al*. [19]. In 1980, Stringer and Kelly reported six cases of traumatic extracranial ICAD [20]. They suggested the intimal injury was produced by hyperextension and lateral flexion of the neck, which cause the artery wall to be stretched. Other two cases were described by Krajewski and Hertzer, while another series of six cases was reported by Zelenock *et al*. [21, 22]. In their work, they reported motor-vehicle accidents in three cases, falls from less than three meters in two cases, and a direct neck blunt trauma (fistfight) in the last case. Six cases were described by Pozzati *et al.* in two different papers [7, 23]. Peculiarly, five patients had neurological manifestation after at least two weeks from the traumatic event (range two weeks – six months). In 1987, Morgan *et al.* described five other cases of post-traumatic ICA injury, two involving children [24]. Then, Mokri *et al*. reported 18 cases of extracranial ICAD as a consequence of blunt head or neck trauma [25]. Again, motor-vehicle accident was the major dynamic. Watridge *et al*. illustrated 24 cases of patients admitted to their medical center after trauma [26]. The presenting symptoms were various (hemiparesis, aphasia, etc.) and no one manifested external signs of a direct

neck injury while two patients had cervical and thoracic spinal fractures. A prompt head CT scan was performed in all cases but 17 up to 24 did not show any cerebral alterations within the first four hours, while 12 of those 17 later developed areas of cerebral infarction. Cerebral arteriography was then performed, revealing 18 monolateral CAD and six bilateral CAD. In 1990, Mokri reported a series of patients suffering from ICAD, 21 of which were traumatic [27]. At follow up, traumatic dissections appeared to be more likely to cause permanent neurological deficit compared to spontaneous dissections.

From 1990 to nowadays, several articles concerning TICAD and traumatic internal carotid artery injuries have been published. Regarding the type of trauma causing the injury, traffic accidents are the most reported (94/227 cases – Table 2). For example, Reddy *et al*. reported the autoptic case of a woman who developed ICAD as a consequence of a car accident [28]. The authors suggested the arterial injury was caused by a seatbelt trauma. In another article, the case of a woman who developed tongue deviation four months after a car accident was described [29]. Magnetic resonance angiography (MRA) revealed ICAD plus intramural hematoma. Besides, the angiography also showed tortuosity of the artery, which in the Authors' opinion could predispose to dissection in case of a traumatic event. A series of six cases concerning ICADs in motor vehicle accidents highlighted the importance of initial patient evaluation and timely angiography execution [30]. In fact, in four of those cases, the diagnosis of ICAD was made within 6 hours from hospital admission, while in the remaining ones it was however made within at least the third day of hospitalization. All patients showed normal ICA's contour at the last follow up angiography, even if three of them still had neurological deficits. Another ICAD subsequent to a motor vehicle accident was described by Matsuura *et al*. [31]. In this case, a woman was driving with no seat belt fastened. She developed neurological symptoms after three days of hospitalization and so and angiography was performed, revealing a right ICAD with a pseudoaneurysm. Vice versa, Babovic *et al.* reported the case of a woman who was driving her car with the seat belt fastened when she was involved in a high-speed collision [32]. The airbag deployed. She had several lesions, including facial bone fractures requiring surgical fixation. Some days after the surgery, on the tenth day after admission, she complained of unilateral progressive visual loss. Through imaging evaluation, they found out the woman had bilateral ICAD with bilateral thrombus formation, causing embolization and cerebral infarction. A similar case was also presented by Jariwala *et al.* [33]. Duncan *et al*. described the analogous case of a man who had a frontal collision with the seat belt fastened and airbag deployment [34]. A brain CT scan and an angiography diagnosed bilateral ICAD with a thrombus in the right ICA. Authors suggested an aetiological role of the airbag deployment. Besides, this case is peculiar because there was evidence of ICA fibromuscular dysplasia, which could be a predisposing pre-existing risk factor

for traumatic dissection. Another particular case of ICAD associated with a car accident was presented by Uhrenholt *et al*. [35]. A man was diagnosed with unilateral ICAD as a consequence of a whiplash injury due to sudden braking while driving a car. ICAD was directly traced back to whiplash trauma since the man did not undergo any other injury. Another interesting case was published by Fusonie *et al.* [36]. A young man manifested three episodes of transient unilateral upper limb weakness over a period of four months. He said he was involved in a car accident several years before. He was diagnosed with right ICA pseudoaneurysm and underwent covered stent exclusion, and then he did not manifest any other episodes. In many other works, motor vehicle accidents, with or without direct head/neck trauma, were the cause of ICAD development [8, 37-56]. On the other hand, only three cases of post-traumatic internal carotid artery lesions referred to bicycle accidents have been reported [7, 57, 58].

Some cases regarded horse riding accidents [46, 59, 60]. A fall from height was the cause of ICAD in 11/227 cases [8, 22, 24, 55, 61, 60].

Direct neck blunt trauma is another possible mechanism of ICA's lesion. For example, eight cases of ICAD as a consequence of hanging and/or strangling have been described [48, 63-67]. There are cases of ICAD following assault, with or without some kind of unsharpened weapon, in which a blunt head or neck injury was probably the cause of the arterial lesion [6, 12, 22, 25, 46, 68]. Hughes *et al*. collected seven cases of ICAD after blunt head trauma [69]. Peculiarly, in all the seven cases ICAD was an incidental finding at cervical spine and/or brain magnetic resonance imaging (MRI) or angiography performed for other reasons. No evidence of cerebral infarct was seen at brain CT, and the patients did not manifest any neurological symptoms correlated to the ICAD. Lo *et al.* collected 18 cases of post-traumatic ICA lesion (10 pseudoaneurysms) suggesting a correlation with craniofacial fractures [70]. Unfortunately, they did not specify the traumatic dynamics of all the cases. Other papers concerning blunt head and/or neck trauma are described in Table 1 [71]. Some Authors described cases of TICAD related to sports practice, both in case of some kind of trauma or not [25, 62, 72-79]. For instance, in Mokri *et al.*'s work, there are cases correlated to football, water-skiing, and skydiving [25]. Fridley *et al.* described a case of TICAD followed wakeboarding [77]. Zhou *et al.* published the case of a young man who went bungee jumping and manifested neck pain after ten minutes [78]. Some hours later, he also experienced paraesthesia to one arm. A carotid arteries ultrasound and then a brain MRA revealed left CCA and ICA dissection with intramural hematoma. In another case, a man developed a headache during a taekwondo training [79]. The days after, he manifested progressive neurologic deficits, such as aphasia, visual disturbances, hemiparesis, and sensory loss. A brain CT scan followed by an MRI and MR angiography revealed a unilateral ICAD with middle cerebral artery (MCA) infarction.

Alimi *et al*. focused on bilateral TICAD, collecting a series of eight cases [61]. Most of them occurred after car accidents, both with or without the seat belt fastened, while in two cases it happened after a moped accident and after a stairway fall. Another case of bilateral ICAD was described by Kumar *et al*. [80]. The Authors correlated the dissection to a minor trauma that occurred while the patient was vomiting. Some hours later, he developed hemiplegia, loss of vision from one eye, slurred speech, and a decrease of consciousness. A MRI showed an infarct in the anterior cerebral artery (ACA) and MCA's territory, a left ICAD with occlusion, and a right ICA's intimal flap with normal blood flow. Lee and Jensen also described a case of bilateral ICAD following a minor trauma [81]. Their patient manifested headache and visual disturbances days after a mountain bicycle ride without any accident or falling off the bike. Vadikolias *et al.* presented the case of a man who developed ICAD after intense jackhammer use (several hours) in a horizontal position [82].

Dissections from trivial injury are also reported by Alimi *et al.* [83]. The Authors described a case of ICA stenosis after cervical manipulation and identified the neck hyperextension as the cause of the arterial lesion. In Fuse *et al.,* an indirect neck injury consequent to dropping a heavy load was the cause of ICAD, which was diagnosed three months after the trauma because of a screening MRI [84]. Pezzini *et al*. reported a case of ICAD after French horn playing. The patient had also two risk factors for spontaneous dissection (hyperhomocysteinemia and aberrant connective tissue morphology), so the Authors considered the case as SICAD. They also questioned the real correlation between trivial traumas and TICAD [85].

TICAD has also been described in correlation to neck penetrating injuries, such as gunshots and stab wounds [66, 86-88]. In particular, Herrera *et al.* collected 14 cases of ICA injuries due to gunshot or stab lesions [88].

With regards to paediatric population, aside from the previously mentioned work of Morgan *et al*. [24], in the literature, there are at least 14 cases of children (< 16-year-old) who developed TICAD, often in relation to minor trauma [58, 89-96]. In particular, the causing event was a trauma to the soft palate/pharynx (a fall carrying something in the mouth or falling with the mouth open against a hard object) in 5/227 cases [93-96].

In some studies, the Authors did not focus on or report specifically the traumatic dynamic causing ICAD [86, 88, 97-103]. For example, Vishteh *et al.,* Herrera *et al.,* and Cohen *et al.* published retrospective studies evaluating only patients who underwent revascularization procedures [86, 88, 103]. Useful data of those papers are reported in Table 1.

**Case report**

A 54-year-old man with no medical history was involved in a head-on highspeed collision against a lamppost while driving a truck. The truck's cockpit was highly damaged during the impact. The man

manifested a transient sudden loss of consciousness soon after the accident. He was immediately transferred to the local Emergency Department and the first evaluation revealed: blood pressure 130/85 mmHg, heart rate 85 bpm, oxygen saturation 96%, right frontal skin abrasion, right foot crush injuries with exposed fracture, normal neurological, thoracic, and abdominal examination. The patient was agitated and therefore 20 mg of midazolam were administered. An X-ray examination of the right foot confirmed the tibia and fibula's fragmented displaced fracture. A whole-body CT scan without contrast was also performed, showing: a fragmented displaced fracture of the right arc of the C1 vertebra with atlanto-occipital disarticulation; multiple left pulmonary contusions associated with pneumatocele; D10 vertebra's body fracture. No ischaemic nor haemorrhagic brain injuries were present. A cervical collar was prescribed, and the patient was admitted to the Orthopaedic Department of the same hospital with the program of surgery for the feet fracture. Two days after admission, he complained that he could not move the left upper limb and the paralysis was confirmed at the physical examination. Therefore, a brain CT scan plus CT angiography was performed, revealing a right posterior cortico-subcortical temporo-parietal insular ischaemic lesion with median shift and a right ICAD with almost completed lumen obstruction and consequent right middle cerebral artery blood flow decrease (Figures 2-3).

A revascularization procedure was not indicated. The patient received 18% mannitol and was transferred to the Stroke Unit. Here, the physical examination showed drowsiness, left hemiplegia, right-sided head deviation, right eye divergent strabismus, bilateral miosis reactive to light stimuli, Cheyne-Stokes respiration, while the vital parameters were the following: blood pressure 130/70 mmHg, heart rate 70 bpm, oxygen saturation 97% (85% in apnoea phases), body temperature 36,6° C. The patient received oxygen-therapy; vital parameters were constantly monitored. During the following hours, he presented two episodes of left hemibody fasciculations and breathing alterations, treated with Lorazepam. The day after, he was comatose, with bilateral mydriasis, and stertorous breathing. A brain CT scan showed ischaemic lesion progression with mass effect, left median shift, and left uncal herniation. He underwent a decompressive hemicraniectomy. During the surgery, a partial temporal lobectomy was also performed since the cerebral parenchyma was not irrorated. Nevertheless, his neurological status deteriorated further, till he was declared brain-dead.

A forensic autopsy was then performed and revealed, aside from cranial surgery sequelae and obvious brain damage, modest adventitial haemorrhagic infiltration of the right ICA a few centimetres distal to the right carotid bifurcation (Figure 4). The right common, internal, and external carotid arteries were sampled and then studied after formaldehyde fixation (Figures 5-6). Histological examination was performed, confirming the presence of cerebral oedema and right ICAD. Specifically, ICA

presented an intramural hematoma with intimal and media laceration, a thrombus was confirmed to be into the lumen (Figure 7).

**Discussion**

ICAD represents the cause of ictus cerebri in 2% of cases but it explains about 20% of all the cases of cerebral infarction among the young adult population [1, 2]. It has been estimated that carotid injuries could complicate the 0,32% of cases of general blunt trauma and the percentage seems to be higher in cases of severe multiple trauma [104, 105]. Specifically, TICAD seems to complicate about 0,21% of all trauma [69]. TICAD could have devastating consequences, from permanent neurological impairment to death [106]. Besides, follow-up studies demonstrated that dissections do not always heal spontaneously and so the risk of complications could persist [14, 107]. Thus, even if it is a rare condition, a prompt diagnosis is essential.

Usually, TICAD is diagnosed when neurological symptoms have already occurred [9]. Clinical presentation is variegated but mostly it is represented by headache, altered consciousness, Horner's syndrome, and focal neurological symptoms such as hemiparesis/hemiparalysis (Table 1). Concerning the timing of clinical presentation, the trauma-symptoms interval is various, from a few minutes up to months. In a peculiar case, clinical manifestation occurred several years after the traumatic event [36]. Nevertheless, in most of the cases, the trauma-symptoms interval does not exceed a week.

In such traumatic cases, there are often concomitant injuries, which can hide or mitigate TICAD neurological manifestations. Besides, other life-threatening injuries could require immediate treatment and/or surgery, delaying a proper neurological examination (i.e. abdominal organ laceration).

Given the above, TICAD should be taken into consideration when a young adult or middle-aged patient presents after severe blunt trauma, although there are no specific guidelines regarding TICAD screening [9]. The risk factors for a blunt carotid injury that should suggest excluding TICAD are cervical hyperextension or hyperflexion, a direct head/neck blunt injury, seat-belt sign, a GCS score </= 6, diffuse axonal brain injury, any kind of cervical spine or cranio-facial fracture [14].

Besides, understanding which kind of traumatic event is most associated with TICAD could help clinicians to direct their diagnostic process. In the literature, TICAD is mostly correlated to motor vehicle accidents (94/227), specifically to car accidents (at least 39/94), and to direct or indirect head and cervical trauma (76/227). In Table n. 2 all the types of event which have been correlated to TICAD are summed up. Usually, TICAD is a consequence of a high-energy collision/blunt trauma, but in few cases, trivial traumas have also been reported.

The mechanism of TICAD development has been mostly referred to as a vigorous extension and flexion of the cervical spine and rotation of the skull. During such movements, ICA is stretched, and the arterial wall may be damage. Shear forces seem to be more intense where ICA movement are averted by surrounding anatomical structure, such as the skull base [57]. Nevertheless, TICAD could be found in both extracranial and intracranial ICA. When TICAD is extracranially located, neck duplex ultrasonography (DUS) could help to identify the arterial wall injury. Therefore, DUS could be suggested as a non-invasive screening tool, but it has low sensitivity and its use is limited to extracranial arteries [50]. Gouny *et al*. emphasized the importance of MRI, which can display precisely the dissection [44]. An aggressive angiographic evaluation has been also proposed [108]. Brommeland *et al.* recommend applying the Denver screening criteria and then performing a computer tomography angiography (CTA) in case of blunt trauma [109]. Nevertheless, those indications have not been completely accepted by the scientific community yet, and there is no uniformity about screening strategies among physicians.

With regards to the case presented in this paper, ICAD can be considered a consequence of the motor vehicle accident despite the absence of any sign suggesting a direct neck or head injury. Besides, from the neck CT images obtained during the hospitalization and the autopsy findings, it was possible to exclude that C1 fracture fragments were involved in ICAD's development (Figure n. 3). Nevertheless, the dissection was probably due to ICA stretch or compression as a consequence of the sudden deceleration. As already said, many Authors suggest that hyperextension and rotation, or direct compression may be the cause of TICAD [12, 22, 57, 90].

From a medico-legal point of view, another issue is the possibility of a medical liability claim. The absence of specific and internationally accepted guidelines leaves physicians facing alone the matter of TICAD screening/diagnostic protocol. In our case, the reasons behind the diagnostic delay, other than the absence of specific guidelines, were the trauma - symptoms interval (two days), and the presence of other injuries requiring timely surgery. Then, when ICAD diagnosis was made, the brain was already gravely injured and so there was no possibility of vascular repairing surgery [110]. This case highlights the importance of screening guidelines to guide physicians to anticipate TICAD diagnosis before symptoms develop, in order to prevent permanent neurological impairment or to attenuate poor prognosis.

## Conclusion

TICAD is a rare condition largely described in correlation to motor vehicle accidents. It affects mainly the young adult population and it could determine permanent neurological defects or even death. TICAD is usually diagnosed when neurological symptoms and cerebral damage have already occurred. The need for screening in cases of head/neck injury is debated, and even if some Authors

have suggested diagnostic criteria, there is no uniformity among physicians. Therefore, there is the possibility of a medical liability claim correlated to TICAD. Identifying which type of trauma is more likely to cause ICAD could be a valid help to suspect this infrequent condition, despite the absence of specific and internationally accepted guidelines.

**Abbreviations**

ACA anterior cerebral artery; CT, computer tomography; CTA, computer tomography angiography; DUS, duplex ultrasonography; GCS, Glasgow Coma Scale; ICA, internal carotid artery; ICAD, internal carotid artery dissection; MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.

## Declarations

### Ethics approval and consent to participate

Not applicable.

### Consent for publication

Informed consent was granted by the Judicial Authority governing specific information included herein.

### Availability of data and materials

There are no associated datasets for this manuscript. Related queries can be directed to the corresponding author.

### Competing interests

The authors declare that they have no competing interests.

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### Authors' contributions

A.F. analysed and interpreted the patient data; A.M., performed the histological examination; E.T., V.F. were involved in writing—review, editing, and supervision; M.D.P. and R.L.R. contributed in writing the manuscript; A.C.M. and A.D.M. performed the literature search. All authors read and approved the final manuscript.

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**Figure legend**

**Figure 1 -** An appraisal based on titles and abstracts as well as a hand search of reference lists was carried out. The resulting 254 references were screened to exclude duplicates, which left 128 articles for further consideration. These publications were carefully evaluated taking into account the main aims of the review. This evaluation left 87 scientific papers comprising original research articles, case reports, and case series.

**Figure 2 -** TC angiography performed soon after neurologic manifestation showed a right ICAD with almost completed lumen obstruction and consequent right middle cerebral artery blood flow decrease.

**Figure 3 -** CT 3D reconstruction details showing C1 dislocated fragment could not be the cause of the TICAD.

**Figure 4 -** Right common, internal, and external carotid arteries dissection. Right ICA showed a modest adventitial haemorrhagic infiltration a few centimetres upper than the carotid bifurcation.

**Figure 5 -** Right common, internal, and external carotid arteries dissection and collection. ICA was sectioned at its petrous level.

**Figure 6 -** Right common, internal, and external carotid arteries sample section and macroscopic examination.

**Figure 7 -** Right ICAD histological examination revealed an intramural hematoma with intimal and media laceration, and a thrombus into the lumen.

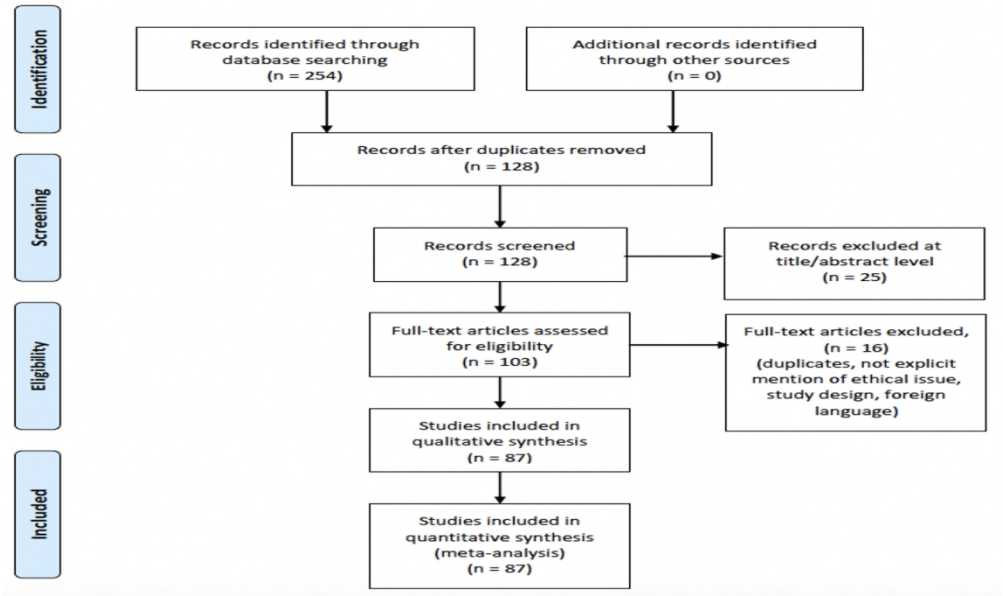


Figure 1 - An appraisal based on titles and abstracts as well as a hand search of reference lists was carried out. The resulting 254 references were screened to exclude duplicates, which left 128 articles for further consideration. These publications were carefully evaluated taking into account the main aims of the review. This evaluation left 87 scientific papers comprising original research articles, case reports, and case series.

450x268mm (100 x 100 DPI)

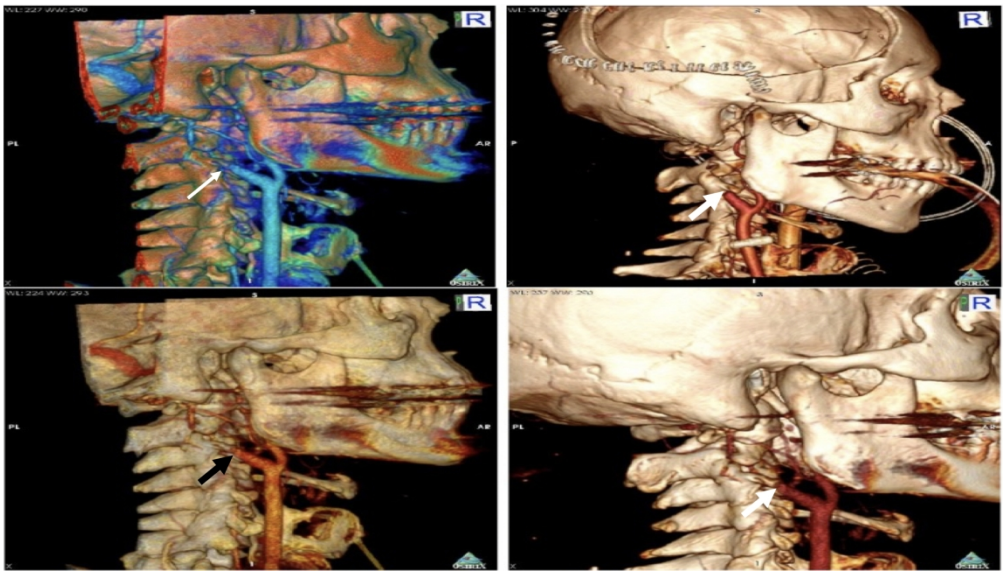

Figure 2 - TC angiography performed soon after neurologic manifestation showed a right ICAD with almost completed lumen obstruction and consequent right middle cerebral artery blood flow decrease.

464x263mm (144 x 144 DPI)

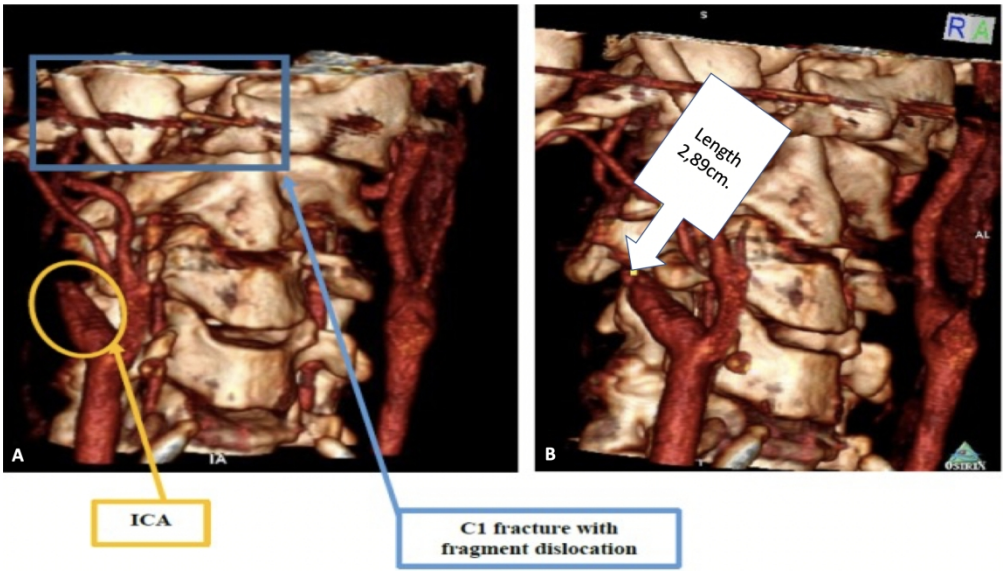


Figure 3 - CT 3D reconstruction details showing C1 dislocated fragment could not be the cause of the TICAD.

460x261mm (144 x 144 DPI)

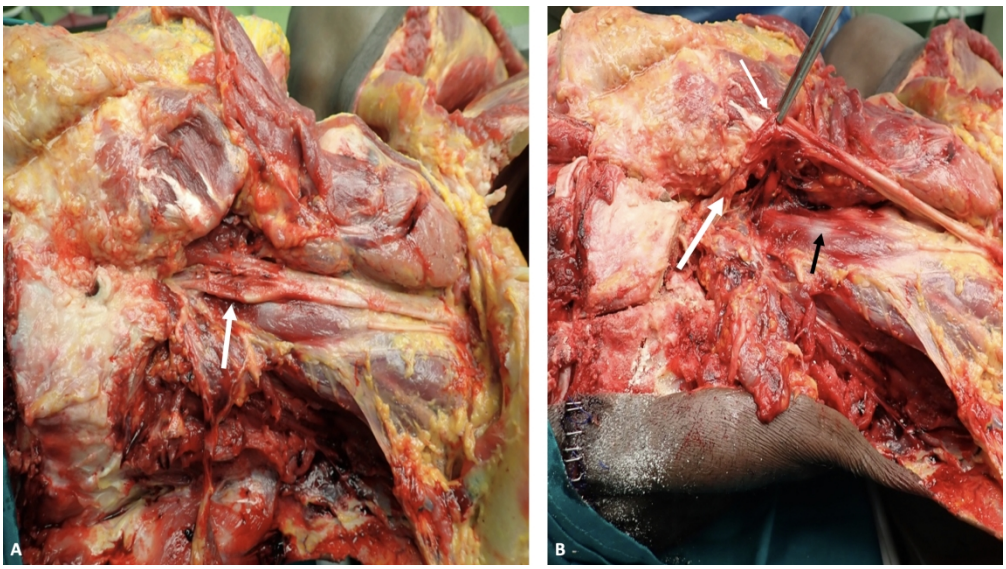


Figure 4 - Right common, internal, and external carotid arteries dissection. Right ICA showed a modest adventitial haemorrhagic infiltration a few centimetres upper than the carotid bifurcation.

479x268mm (144 x 144 DPI)


Figure 5 - Right common, internal, and external carotid arteries dissection and collection. ICA was sectioned at its petrous level.

205x240mm (144 x 144 DPI)


Figure 6 - Right common, internal, and external carotid arteries sample section and macroscopic examination.

470x264mm (144 x 144 DPI)

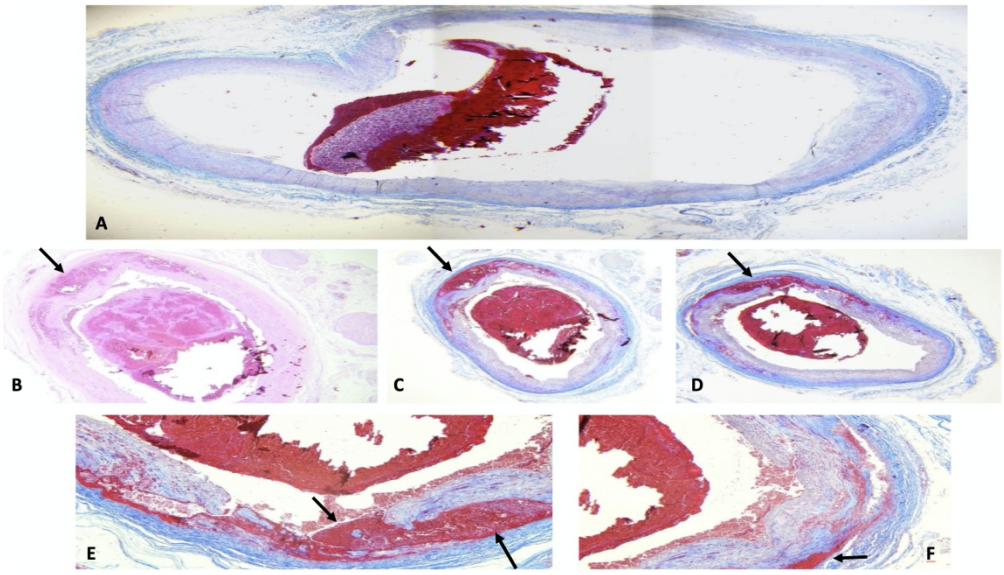


Figure 7 - Right ICAD histological examination revealed an intramural hematoma with intimal and media laceration, and a thrombus into the lumen.

465x263mm (144 x 144 DPI)

Table 1. Summary of the literature regarding post-traumatic internal carotid artery dissection. Studies conducted before 1990 have been excluded. ACA indicates anterior cerebral artery; AVF, arteriovenous fistula; CA, cerebral artery; CAD, carotid artery dissection; CCAD, common carotid artery dissection; CT, computer tomography; CTA, computer tomography angiography; DSA, digital subtraction angiography; DUS, duplex ultrasonography; ECAD, external carotid artery dissection; GCS, Glasgow Coma Scale; ICA, internal carotid artery; ICAD, internal carotid artery dissection; IVUS, intravascular ultrasound; MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; MVA, motor vehicle accident; PMCT, post-mortem computer tomography; TIA, transient ischaemic attack; TCD, transcranial doppler sonography; VAD, vertebral artery dissection.

| Reference | Number of cases | Age | Sex | Type of trauma | Presenting neurological symptoms | Trauma - symptoms interval | Before diagnosis imaging | Type of ICA lesions | Other correlated findings |
|---|---|---|---|---|---|---|---|---|---|
| Martin *et al*. 1991 | 3 | 15ys | M | MVA | Hemiparesis | - | CT, DUS, angiography | ICAD | - |
| | | 22ys | F | | - | | CT, angiography | | |
| | | 43ys | F | | | | angiography | Bilateral ICAD | |
| Romner *et al* 1994 | 2 | 26ys | M | Wrestling | Altered consciousness, dysphasia, hemiparesis | < 1 day | CT, TCD, angiography, SPECT | ICAD | - |
| | | 23ys | F | Fall from staircase | | Some minutes | | | MCA infarct |
| Achtereekte *et al*. 1994 | 1 | 48ys | 1 M | Bicycle accident (blunt head trauma) | Transient loss of consciousness, aphasia, concentration disturbances, short-term memory loss | < some hours | Skull CT, TCD, DUS, angiography | ICAD with saccular aneurysm | Hematoma and bruise of the frontal area; Right MCA blood flow decrease |
| Fletcher *et al*. 1995 | 1 | 31ys | 1 M | Jockey fall (jaw and neck injury) | Loss of consciousness (soon recovered), Horner's syndrome | < some hours | Neck X-rays, head CT | Left ICAD with complete occlusion | Left MCA infarct, left vertebral artery occlusion |
| | | | | | Horner's syndrome, major convulsive seizure, aphasia, hemiplegia | 4 days | Head CT, angiography | | |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Sanzone *et al.* 1995 | 2 | 27ys<br>39ys | 2 M | Assault with a lead pipe | Loss of consciousness, hemiplegia, fixed dilated left pupil<br>Hemiplegia and hemianopsia | < 1 day<br>1 day | Facial X-rays, head CT, angiography | ICA tapering | MCA and ACA infarct |
| Lemmerling *et al.* 1996 | 1 | 50ys | 1 M | Car accident | Dysarthria, difficult swallowing and hypoglossal nerve dysfunction | < some hours | Neck CT, MRA | ICAD | - |
| Laitt *et al.* 1996 | 8 | 35.9ys (range 21-52) | 5 M, 3 F | MVA (6), assault (1), horse fall (1) | Hemiparesis or hemiplegia (8), dysphasia or aphasia (4) | 4 hours up to 75 days | Brain CT, angiography, MRI and MRA (1) | ICAD (7), ICA pseudoaneurysm (1) | Cerebral infarct (7) |
| Alimi *et al.* 1996 | 7 | 35.7ys (range 21-59) | 3 M, 4 F | MVA (6), cervical manipulation (1) | Hemiparesis (2), hemiplegia (2), aphasia (2), Horner's syndrome (1), oculomotor disturbances (1), recurrent TIAs (1) | < some hours | CT (7), doppler arteriography (5), arteriography (2) | Unilateral ICAD (3) with controlateral thrombosis (2), bilateral ICAD (1), false aneurysm (2), tight stenosis (1) | Cerebral infarct or hypodense cerebral lesions (6) |
| Pica *et al.* 1996 | 1 | 31ys | F | Car accident (restrained passenger) | Right retroorbital headache, right-sided tongue deviation, dysarthria | 4 months | Head and neck CT, lumbar puncture, MRI and MRA | Right ICAD with intramural hematoma | ICA tortuosity |
| Sidhu *et al.* 1996 | 1 | 17mo | M | Soft palate injury | Seizures | 48 hours | MRI, MRA | ICAD | Soft palate abrasion; cerebral infarct |
| Duke *et al.* 1997 | 6 | 29.5ys (range 19-40) | 3 M, 3 F | MVA | Horner's syndrome (1), hemiparesis (2), no symptoms (4) | < 2 hours up to 5 days | Head CT, angiography | ICAD (3), ICA intimal flap (1), ICA pseudoaneurysm (3) | Cerebral infarcts (2) |
| Matsuura *et al.* 1997 | 1 | 20ys | 1 F | MVA (no seat belt) | Carotidynia, unilateral oculosympathetic paresis, unilateral loss of limbs | 3 days | Cervical spine X-rays (soon after the accident), arteriography | Right ICAD at C1 level with pseudoaneysysm | - |

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| | | | | | sensation, hemiparesis | | | | |
| Vishteh et al. 1998 | 13 | 30,6ys (range 12-71) | 9 M, 4 F | Blunt trauma (11), gunshot (1), stab (1) | Hemiparesis (11), cranial nerve deficits (2), aphasia (2), Hornes's syndrome (2), focal seizure (1) | within some hours or later after hospital discharge | Brain CT (11), brain MRI (3), angiography (all) | ICAD (9 cervical, 3 distal cervical and petrous, 3 cavernous, and 1 petrous segments), plus occlusion (7), dissecting aneurysm (6), and rupture with carotid-cavernous fistula formation (2) | Cerebral contusion (5), elevated intracranial pressure (4), basal cranial fractures (5), vertebral fracture (2) |
| Alimi *et al.* 1998 | 8 | 35,2ys (range 17-54) | 3 M, 5 F | MVA (6 in car, 4 of which with seatbelt fastened; 1 moped), stairway fall (1) | Neurological deficit (3) plus aphasia (1), unconsciousness (6), hemiplegia (2) | < some hours up to 13 days | Brain CT, DUS (4), angiography | Bilateral ICAD (8), with or without stenosis, dilatation, or thrombosis | Unilateral cerebral infarction (5), bilateral cerebral infarction (3), plus haemorrhagic cerebral contusion (2) |
| Kumar *et al.* 1998 | 1 | 45ys | 1 M | Vomiting | hemiplegia, one eye loss of vision, slurred speech, decrease of consciousness | About 18 hours | Head CT, DUS, brain MRI | Bilateral ICAD, one side with occlusion, other side only intimal flap | ACA and MCA's infarct |
| Gouny *et al.* 1998 | 1 | 39ys | 1 M | Motorcycle accident | Unilateral anisocoria and mydriasis, hemiplegia | < some hours | Brain CT, cervical echography, MRI | Bilateral ICAD with bilateral thrombosis | - |
| Schievink *et al.* 1998 | 4 | 35ys (range 31-39) | 3 M, 1 F | Softball neck direct impact (2), | None (only the softball cases are described) | - | Head CT, angiography | ICAD with intimal flap, aneurysm, maybe thrombosis | - |

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| | | | | car accidents (2) | Monolateral ptosis and miosis (only the softball cases are described) | 3 days | DUS, MRA | ICAD | - |
| Simionato *et al.* 1999 | 1 | 39ys | 1 M | Car accident (craniofacial trauma) | Hemiparesis | < some hours | Head CT and MRI, MRA, digital subtraction angiography | ICAD with aneurysm and obstruction | Fronto-insular and deep hemisphere infarct |
| Babovic *et al.* 2000 | 1 | 43ys | 1 F | Car accident (seatbelt fastened, airbag deployment) | Unilateral progressive visual loss | 10 days | Orbits CT, fundus oculi examination, head CT, head MRI and MRA | Bilateral ICAD with bilateral thrombus | Closed head injury and facial fractures; frontal lobe infarct |
| Duncan *et al.* 2000 | 1 | 39ys | 1 M | Car accident (seatbelt fastened, airbag deployment) | Hemiplegia | < some hours | Brain CT, angiography | Bilateral ICAD with thrombus in the right ICA | fibromuscular ICA dysplasia; parietal lobe infarct with later haemorrhage |
| Busch *et al.* 2000 | 1 | 27ys | 1 F | Motorcycle accident | Progressive loss of consciousness | Several hours | Brain CT, angiography | Bilateral ICAD | VAD; cerebral infarct |
| Hughes *et al.* 2000 | 7 | - | - | Severe blunt head trauma | None (incidental finding) | - | Cervical spine/brain MRI (6), angiography (1) | ICAD | - |
| Lee and Jensen 2000 | 1 | 43ys | 1 F | Bicycle ride (no fall or accident) | Acute headache | < some hours | - | Bilateral extracranial ICAD with bilateral hematomas and pseudoaneurysms and stenosis | bilateral poor disc and cup margins, small inferotemporal cotton-wool spot in the left eye |
| | | | | | persistent headache, transient visual disturbances such as unilateral | 1 day | Head CT (normal at day 2), ophthalmosco | | |

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| | | | | | scotoma and "granular" vision, transient complete blindness, unilateral ptosis and anisocoria | | pic examination (day 9), dilated funduscopic examination, MRI and MRA (day 11) | | |
| Malek *et al.* 2000 | 2 | 37ys | 2 F | Strangulation | Upper limbs weakness, leg numbness, and dysphasia | - | - | ICAD | - |
| | | 44ys | | MVA (whiplash injury) | Dysphasia, unilateral upper limb weakness and numbness | | | | |
| Scavée et al. 2001 | 1 | 53ys | M | MVA | Neck pain and dizziness | 6 weeks | CT, angiography, MRI | ICA pseudoaneurysm with intramural thrombus | - |
| Windfuhr 2001 | 1 | 5ys | F | Pharynx penetrating injury | Oral bleeding and anemia | 9 days | Angiography | ICAD with aneurysm | 3 mm pharyngeal lesion |
| McNeil *et al.* 2002 | 1 | 18ys | M | Gunshot | Not appreciable (sedated) | - | Head, face, and cervical spine CT, angiography | ICA pseudoaneurysm | Distal embolic angular artery occlusion |
| Duane *et al.* 2002 | 2 | 31ys | F | Strangulation and stabbing attempt | Seizure, tongue deviation | 8 days | Neck CT, angiography | ICA pseudoaneurysm | peritonsillar abscess |
| | | 27ys | F | Gun shot | - | - | Head x-ray, angiography | ICA AVF with pseudoaneurysm | - |
| Blanco Pampin *et al.* 2002 | 2 | 19ys | M | Car accident | Confusion, speech difficulties, unilateral facial nerve paralysis, and unilateral hemiplegia | 48 hours | Head CT, DUS, angiography | ICAD with thrombosis | Neck bruise and cerebral infarct |

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| | | 33ys | F | Hanging attempt | Loss of consciousness and unilateral hemiplegia | 6 hours | Head CT | ICAD | Neck bruise and cerebral infarct with C2 odontoid fracture |
| Men *et al.* 2003 | 1 | 48ys | M | MVA | - | Few weeks | Angiography | ICAD with AVF | - |
| Pary and Rodnitzky 2003 | 1 | 43ys | M | Taekwondo training | Headache, transient visual disturbances, unilateral hemisensory loss and hemiparesis, Wernicke's aphasia | < some hours | Head CT, brain MRI, MRA | ICAD with hematoma | MCA infarct |
| Fusonie *et al.* 2004 | 1 | 37ys | M | Car accident | One upper limb weakness episodes | 15 years | Cervical MRI, MRA | ICA pseudoaneurysm | - |
| Fanelli *et al.* 2004 | 1 | 17ys | 1 M | Motorcycle accident | Hemiplegia and positive Babinski's sign | < some hours | Head CT | Bilateral ICAD | Right hemisphere cerebral infarct |
| Payton *et al.* 2004 | 1 | 11ys | 1 M | Playing accident (hitting head or neck to a padded wall) | Dysarthria, lethargy, ocular deviation, hemiplegia | < some hours | Multiple X-rays, head and cervical spine CT, head MRI and MRA | Bilateral ICAD | - |
| Fateri *et al.* 2005 | 1 | 52ys | 1 M | Car accident | Altered consciousness, hemisyndrome | < some hours | Craniocervicalthoracic CT | ICAD with tight stenosis and luminal thrombosis | Cerebral arteries' filling defects related to thromboembolic events |
| Clarot *et al.* 2005 | 2 | 38ys | 1 M | Attempted strangulation | Altered consciousness, bilateral Babinski's sign, permanent eye elevation, bradycardia, | Hospital admission (not known the time from the trauma) | Brain CT, DUS | Bilateral CAD with bilateral thrombus and right ICAD and ECAD | Neck ecchymosis and abrasions; cerebral infarct and subarachnoid haemorrhage |

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| | | | | | and right hemiparesis | | | | |
| | | 42ys | 1 F | | Headache | 2 days | DUS, brain CT | Bilateral CAD | - |
| Cohen *et al.* 2005 | 10 | 42.7ys (range 17-62) | 8 M, 2 F | Multiple (6) or cranio-cervical trauma (4), with penetrating injury (2) | Signs of ischaemic stroke, TIA, carotidynia, Horner's syndrome | 4 hours uo to 19 days | Brain CT, angiography | ICAD | - |
| Cothren *et al.* 2005 | 46 | 32±2 ys | 65% M, 35% F | MVA, falls, skiing injuries | Not specified, 38 patients asymptomatic, 8 patients symptomatic | - | Angiography | Pseudoaneurysm | - |
| Joo *et al.* 2005 | 4 | 28.5ys (range 19-38) | 4 M | Blunt trauma (3) | Limb weakness (2), none (1) | - | CT, MRI, arteriography | Extracranial ICA pseudoaneurysm | - |
| | | | | Stab wound (1) | Limb weakness, pulsatile swelling and bruit | | | Extracranial ICA pseudoaneurysm with ICA-internal jugular vein AVF | |
| de Borst *et al.* 2006 | 1 | 13ys | 1 F | Bicycle-motor vehicle accident | Hemiplegia with unilateral facial palsy, ipsilateral hemianopia | < some hours and few days after | Brain CT, brain MRI, and MRA | Bilateral ICAD | Unilateral ACA infarct |
| Chokyu *et al.* 2006 | 1 | 61ys | 1 F | Accidental strangulation | Hemiparesis, unilateral facial palsy | 1 day (soon after the trauma she also had tetraparesis due to spinal cord injury) | Brain CT, cervical MRI, MRA | Bilateral CCAD | Unilateral cerebral infarct |
| Yang *et al.* 2006 | 3 | 22ys | 3 M | Fall (1) | Altered consciousness, hemiparesis | 2 days | Brain CT, neck CTA, DUS | ICA thrombus and caliber narrowing | Neck abrasion and bruit |
| | | 47ys | | MVA (2) | Altered consciousness | < some hours | Brain CT, cervical X | ICAD | Frontal scalp laceration, some cranial and C2 |

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| | | | | | | | ray, angiography | | fractures, pneumocranium, subarachnoid hemorrhage, |
| | | 48ys | | | Altered consciousness, visual acuity reduction, extraocular movements alteration, hemiparesis | 7 days | Brain and facial CT, angiography | | Multiple craniofacial fractures, haemorragic ACA and MCA infarct, |
| Jariwala *et al.* 2006 | 1 | 17ys | F | Car accident | Progressive consciousness alteration, hemiparesis and sensation loss | < some hours | Brain and neck spine CT | ICAD | MCA and partially PCA infarct |
| | | | | | | | Brain CT, MRI, MRA | | |
| Pierrot *et al.* 2006 | 2 | 4,5ys | 2 F | Soft palate injury (with oral bleeding) | Altered consciousness, hemiplegia, central facial palsy, aphasia | 24 hours | Brain CT and MRI | ICAD with parietal thrombus | Insular cortex infarct |
| | | 3,5ys | | | - | < some hours | | | - |
| Lin *et al*. 2007 | 1 | 7ys | 1 M | Playing at a water park | Head and neck pain, vomiting, hemiparesis, Babinski's sign, hemi facial palsy with slurred speech and uvula deviation | < some hours | Brain CT, MRI, MRA, angiography | ICAD | Acute cerebral infarct |
| Lo *et al.* 2007 | 10 | 29.7ys (range 16-57) | 7 M, 3 F | MVA (2), unspecified (8) | Altered consciousness (2), unspecified (8) | - | Brain CT, CTA, | ICA pseudoaneurysm | Craniofacial fractures |
| Zhou *et al.* 2007 | 1 | 28ys | 1 M | Bungee jumping (no fall) | Right arm paraesthesia | < some hours | Neck US, brain MRI, MRA | ICAD with intramural haematoma | - |
| Schulte *et al*. 2008 | 2 | 27 and 39ys | 1 M, 1 F | Blunt neck trauma | TIA, headache, vertigo | - | DUS, CTA | CAD | - |

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| Fuse *et al*. 2008 | 1 | 42ys | M | Neck injury dropping a heavy load | - | - | Head and neck MRI, angiography, single photon emission CT | ICAD | Tracheal fracture, recurrent transient bilateral nerve paralysis; cerebral infarct, |
| Flaherty and Flynn 2008 | 1 | 34ys | F | Hand assault (hit on the face) | Horner's syndrome | 4 days | Brain CT, neck CTA | ICAD | - |
| Vadikolias *et al.* 2008 | 1 | 48ys | M | Intense jackhammer use | Hemiparesis | < some hours | Brain CT, DUS, TCD | ICAD | MCA infarct with haemorrage |
| Moriarty *et al.* 2009 | 1 | 10mo | F | Soft palate injury | Altered consciousness and progressive hemiplegia (no oral bleeding) | 1 day | Brain CT, brain and neck MRI, neck and intracranial MRA | ICAD with thrombus | MCA infarct with haemorrhage, MCA thrombus |
| Molacek *et al.* 2012 | 1 | 49ys | F | Strangulation attempt | Altered consciousness | - | Brain and neck CTA | Bilateral ICAD | Neck strangulation groove |
| Keilani *et al.* 2010 | 1 | 52ys | F | Horse riding fall and multiple injuries | Altered consciousness | 1 day (at admission she had several other lesions which required surgery) | Brain MRI, angiography | ICAD with pseudoaneurysm | Multiple cerebral infarcts |
| Stager *et al.* 2011 | 1 | 55ys | F | MVA | - | - | CTA, angiography with IVUS | ICAD | Several other lesions, no brain injury |
| Herrera *et al.* 2011 | 14 | - | - | Gunshot and stab injuries | Bleeding, pulsatile mass, neck bruit, hematoma, stroke, dementia syndrome | - | - | Pseudoaneurysm, AVF, dissection, active bleeding | - |
| Fridley *et al.* 2011 | 1 | 40ys | M | Wakeboarding | Headache, hemiplegia | 1 day | Head CT, MRI, MRA, angiography | ICAD | Unilateral basal ganglia and |

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|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | internal capsule infarct |
| Taşcılar *et al.* 2011 | 1 | 31ys | M | Football (neck struck by the ball) | Altered consciousness, hemifacial paresis, hemiplegia, aphasia, positive Babinski's sign | < 6 hours | CT, DUS, MRA | ICAD | MCA infarct |
| van Wessem *et al.* 2011 | 5 | 20ys | 2 F | Car accident | Altered consciousness | < some hours | Head and cervical CT, DUS, CTA | ICAD | C0 condyle fracture, MCA infarct |
| | | 49ys | | | Altered consciousness, legs paralysis and sensory loss | | Brain CT, CTA and angiography | | Multiple spine fractures, right temporal lobe hematoma |
| | | 19ys | 1 M | | Altered consciousness | | Brain and cervical CT and CTA | | Multiple facial, C0 condyle, skull base fractures, MCA infarct |
| | | 53ys | M | Truck accident | Sudden decrease of consciousness, hemiparesis, unilateral Babinski's sign | < some hours | Brain and neck CT | | Bilateral C0 fracture, MCA infarct |
| | | 19ys | M | Motorbike accident | Altered consciousness, different blood pressure between the arms | < some hours | Aortic CTA | | Multiple cranial and skull base fractures, multiple intracerebral hematomas, ACA and MCA infarct |
| | | | | | Still altered consciousness, bilateral spontaneous stretching of both arms and hemiparesis | 7 and 8 days | Brain CT and CTA | | |
| Cohen *et al.* 2012 | 23 | 44ys (range 17-66) | 19 M, 4 F | Multiple trauma (11), | Ischaemic stroke symptoms (14), TIA (3), Horner's | 2 hours up to 21 days | Head and neck CT, CTA (all) | ICAD | - |

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| | | | | penetrating neck injury (2), minor cervico-cranial trauma (10) | syndrome (1), carotidynia (1) | | | | |
| Makhlouf *et al.* 2013 | 1 | 60ys | 1 F | Hand assault (hit on the head) | Headache | < some hours | Cervical X-rays | ICAD with pseudoaneurysm | Unilateral corona radiata infarct |
| | | | | | Unilateral facial palsy and Horner's syndrome | 3 months | Brain MRI and MRA | | |
| Prasad *et al.* 2013 | 1 | 22ys | F | MVA | Altered consciousness | < some hours | Head CT, angiography | ICAD with AVF | Multiple facial fractures, subarachnoid haemorrhage, cerebral oedema |
| Seth *et al.* 2013 | 47 | 34ys (range 17-71) | 32 M, 15 F | Blunt (47) and penetrating (6) injuries | - | - | CT or conventional angiography | Unilateral (41) and bilateral (6) ICAD with or without pseudoaneurysm | - |
| Hostettler *et al.* 2013 | 1 | 47ys | 1 M | Softball blunt injury | Neck and head pain, amaurosis fugax, Horner's syndrome | 1 week | Brain CT, DUS, MRA | ICAD with mural thrombus | - |
| Orman *et al.* 2013 | 5 | 3.6ys | F | Fall | Hemiplegia, aphasia | - | CT, MRI and/or CTA/MRA | ICAD | Cerebral infarct (3) |
| | | 7.6ys | M | Head trauma | - | | | | |
| | | 3.1ys | M | MVA | Focal seizure | | | | |
| | | 1.9ys | F | Head trauma | Altered mental state | | | | |
| | | 1ys | M | Fall | Unilateral hypoesthesia | | | | |
| Kalantzis *et al.* 2014 | 1 | 39ys | 1 M | Snowboarding fall | Horner's syndrome, periocular and neck pain | 2 days | Head and neck CT, MRI, MRA | ICAD | - |
| Correa and Martinez 2014 | 1 | 41ys | 1 M | Blunt head and neck | Transient loss of consciousness | < some hours | - | ICAD with stenosis | Neck abrasion, carotid bruit; |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | trauma | Headache, unilateral visual loss, hemiparesis, unilateral hyperreflexia and Babinski's sign | 48 hours | Brain CT, MRI, angiography | | acute cerebral infarct |
| Crönlein *et al.* 2015 | 1 | 28ys | 1 F | Car accident | Altered consciousness, head pain, anisocoria | < some hours | Total body CT<br>CTA, US | Bilateral ICAD | Unilateral central region cerebral infarct |
| Uhrenholt *et al.* 2015 | 1 | 42ys | 1 M | Sudden braking | Neck pain, headache, cramps, gradually altered consciousness | < some hours | Brain CT, (PMCT) | ICAD with pseudoaneurysm and mural thrombus | Subarachnoid haemorrhage |
| Morton *et al.* 2016 | 39 | 41ys | 22 M, 17 F | - | - | - | Head and neck CTA | ICA pseudoaneurysm (bilateral in 4 cases) | Cerebral infarct (7) |
| Griessenauer *et al.* 2016 | 2 | 21ys | 1 M, 1 F | MVA | Altered consciousness | < some hours | Head CT, CTA | ICA aneurysm | Cranial and facial fractures, intracranial haemorrhage |
| Taoussi *et al.* 2017 | 1 | 29ys | F | Car accident | Dysphasia, upper limb hemiparesis, | < 12 hours | MRI | Bilateral ICAD | Multiple cerebral infarct |
| Cebeci *et al.* 2018 | 1 | 10ys | 1 M | Trivial shoulder trauma | Headache, speech impairment, vomiting, and facial paralysis | 6 hours | Head MRI and MRA | ICAD | . |
| Ariyada *et al.* 2019 | 1 | 23ys | 1 M | Pedestrian run over | Altered consciousness (recovered in some hours) | < some hours | Whole body CT | - | Thin subdural hematoma, odontoid process, pelvis, and limbs' fracture |
| | | | | | Bleariness | 1 month | CT angiography, MRA, DSA | Bilateral ICAD | VAD with thrombus |
| Gabriel *et al.* 2019 | 1 | 37ys | 1 F | CrossFit training | Headache, dizziness, neck pain, unilateral amaurosis fugax | 1 hour | Cervical and chest X-ray, DUS, brain CT, MRI, angiography | Bilateral ICAD | Unilateral corona radiata infarct |
| | | | | | Hemiplegia, dyslalia, aphasia, dysphagia, | 48 hours | | | |

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| | | | | | unilateral facial droop | | | | |
| Petetta *et al.* 2019 | 1 | 44ys | M | Motorcycle accident | Altered consciousness, traumatic shock | < some hours | Whole body CT | - | Several other lesions, no brain injury |
| | | | | | Altered consciousness | 5 days | Brain CT, MRI, CTA | Bilateral ICAD with intraluminal thrombus | Multiple cerebral infarct |
| Wang *et al.* 2020 | 6 | 52.67 ys (range 43-62) | 5 M, 1 F | Car accident (2), motorcycle accident (2), fall from height (1), blunt head injury (1) | Paralysis (2), altered consciousness (2), headache (1), neck pain (1) | 4-45 hours | CT, CTA, DUS, DSA, MRI, TCD in various combinations | ICAD | Cerebral infarct (6) |
| Total articles 77 | Total subjects 334 | Mean age 18.9ys | 200 M 113 F | | | | | | |

Table 2. Traumatic event causing internal carotid artery dissection in the literature. Only cases in which the traumatic event was reported are included. MVA indicates motor vehicle accident.

| Type of trauma | Subtype of trauma | Number of cases | Tot. | References |
|---|---|---|---|---|
| **Traffic accidents** | Generic MVA | 36 | **94** | [7, 8, 20, 23, 26, 30, 45, 46, 48, 49, 51-53, 56, 70, 83, 92] |
| | Car accidents | 39 | | [20, 22, 25, 28, 29, 31- 38, 40, 43, 47, 54, 55, 61, 63] |
| | Truck accident | 1 | | [38] |
| | Motorcycle and moped accidents | 13 | | [7, 22, 25, 38, 39, 42, 44, 50, 55, 61, 70] |
| | Bicycle accidents | 3 | | [7, 57, 58] |
| | Pedestrian accidents | 2 | | [41, 46] |
| **Head or neck blunt injuries** | Not specified/indirect | 48 | **76** | [19, 24, 25, 55, 69, 71, 86, 92, 98, 99, 101, 103] |
| | Fistfight/ assault with or without blunt weapon | 7 | | [6, 12, 22, 25, 46, 68] |
| | Hanging or strangulation | 8 | | [17, 48, 63-67] |
| | Soft palate/pharynx injury | 5 | | [93-96] |
| | Trivial or minor traumas | 8 | | [80-84, 89-91] |
| **Penetrating injuries** | Not specified | 22 | **27** | [88, 99, 100, 103] |
| | Gunshot | 3 | | [68, 86, 87] |
| | Stab wound | 2 | | [86, 101] |
| **Sport (with or without specific blunt trauma)** | Horse-riding fall | 3 | **19** | [46, 59, 60] |
| | Football | 4 | | [24, 25, 76] |
| | Snowboarding | 1 | | [75] |
| | Water-skiing/ wakeboarding | 2 | | [25, 77] |
| | Skydiving | 1 | | [25] |
| | Basketball | 1 | | [7] |
| | Softball | 3 | | [73, 74] |
| | Taekwondo | 1 | | [79] |
| | CrossFit | 1 | | [72] |
| | Bungee jumping | 1 | | [78] |
| | Wrestling | 1 | | [62] |
| **Falls** | Not specific height | 6 | **11** | [8, 24, 55, 61, 62] |
| | < 3 meters | 4 | | [22, 92] |
| | > 3 meters | 1 | | [20] |
| Total | | | **227** | |