

Traumatic Internal Carotid Artery Injuries: Do We Need a Screening Strategy? Literature Review, Case Report, and Forensic Evaluation



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Abstract: Internal carotid artery dissection (ICAD) represents the cause of ictus cerebri in about 20% of all cases of cerebral infarction among the young adult population. ICAD could involve 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 traumas. 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 highlight 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.

ARTICLE HISTORY

Received: January 07, 2021
Revised: April 05, 2021
Accepted: June 01, 2021

DOI:
10.2174/1570159X19666210712125929



Keywords: Internal carotid artery dissection, trauma, diagnostic screening, cervical trauma, neurological impairment, direct or indirect trauma.

1. INTRODUCTION

Internal carotid artery dissection (ICAD) occurs when the blood penetrates the arterial wall because of a defect in the internal elastic lamina. The collection of 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 approximately 20% of cases of cerebral infarction among the young adult population [1, 2]. ICAD can be spontaneous (SICAD) or traumatic (TICAD). SICAD occurs in the absence of a traumatic event and usually correlates with genetic syndromes, recent infections, or specific risk factors (*i.e.*, hypertension, migraine, and hypercholesterolemia). Conversely, TICAD follows a traumatic event. Both the extracranial and intracranial ICAs can be involved [3, 4]. Usually, a direct or indirect cervical injury is described, often correlating with motor vehicle accidents [5-8]. The need for diagnostic screening for TICAD in cases of head and/or cervical injury is controversial [9].

TICAD is often misdiagnosed or diagnosed when neurological symptoms have already occurred [9, 10]. As a consequence, significant and permanent neurological impairment can occur. For blunt carotid injuries, the morbidity rate is estimated to be as high as 80%, and the mortality rate is estimated to be as high as 40% [10-12]. Therefore, TICAD could have forensic consequences.

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

2. 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

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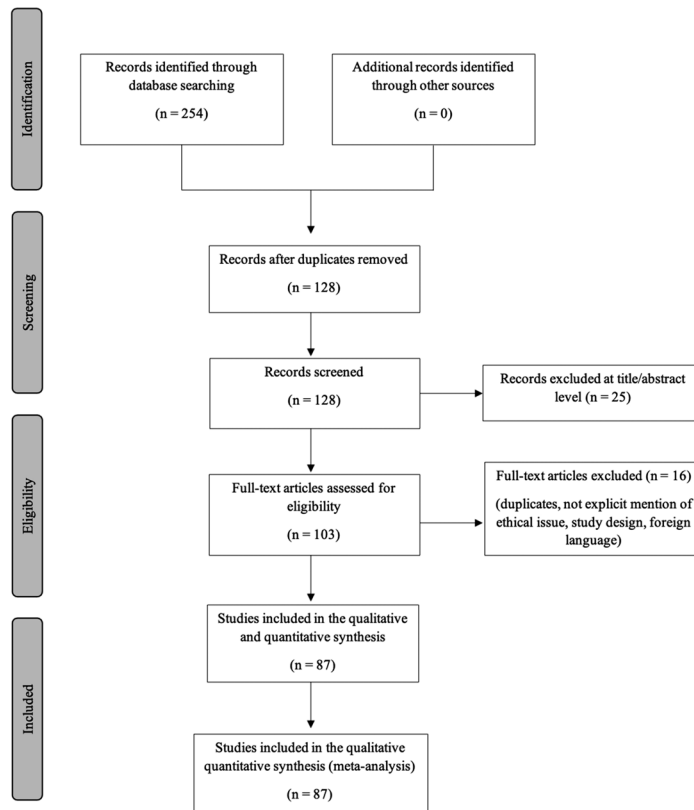


Fig. (1). An appraisal based on titles and abstracts as well as a hand search of reference lists was carried out. The resulting 254 references were screened to exclude duplicates, which left 128 articles for further consideration. These publications were carefully evaluated, taking into account the main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

“trauma” in the title, abstract, and keywords. The bibliographies of all located papers were examined and cross-referenced to identify relevant literature further. 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 Biffi type I, II, and III vascular injuries (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 exempted from institutional review board approval as it did not involve human subjects..

3. 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 abstracts. 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. Fig. (1) illustrates our search strategy.

Table 1 summarizes all the studies published from 1990 to the present. The studies published before 1990 were excluded from Table 1 but are briefly described below. In a few cases, complete data extraction was not possible. However, the eligible data are reported in Table 1.

As shown in Table 2, the trauma mechanisms causing TICAD were gathered from the published reports, when possible, and categorised in five classes (“Type of trauma”). The classes were also divided into 28 subclasses (“subtype of trauma”) to provide a more detailed analysis. Table 2 shows the results of this re-analysis; the proportion of each class and subclass of the total reported cases is also shown.

3.1. Brief Description of the Studies Published Before 1990

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 that extended to the middle cerebral artery. Subsequently, in 1944, Northcroft and Morgan described dissection of the left ICA that occurred by accidental hanging [17]. In 1967, Yamada *et al.* investigated 51 cases of carotid artery

Table 1. Summary of the literature regarding post-traumatic internal carotid artery dissection. Studies conducted before 1990 have been excluded.

References	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 <i>et al.</i> 1991	3	15ys	M	MVA	Hemiparesis	-	CT, DUS, angiography	ICAD	-
		22ys	F		CT, angiography				
		43ys	F		angiography		Bilateral ICAD		
Romner <i>et al.</i> 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 <i>et al.</i> 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 <i>et al.</i> 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 <i>et al.</i> 1995	2	27ys	2 M	Assault with a lead pipe	Loss of consciousness, hemiplegia, fixed dilated left pupil	< 1 day	Facial X-rays, head CT, angiography	ICA tapering	MCA and ACA infarct
		39ys			Hemiplegia and hemianopsia	1 day			
Lemmerling <i>et al.</i> 1996	1	50ys	1 M	Car accident	Dysarthria, difficult swallowing and hypoglossal nerve dysfunction	< some hours	Neck CT, MRA	ICAD	-
Laitt <i>et al.</i> 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 <i>et al.</i> 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 contralateral thrombosis (2), bilateral ICAD (1), false aneurysm (2), tight stenosis (1)	Cerebral infarct or hypodense cerebral lesions (6)
Pica <i>et al.</i> 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 <i>et al.</i> 1996	1	17mo	M	Soft palate injury	Seizures	48 hours	MRI, MRA	ICAD	Soft palate abrasion; cerebral infarct

(Table 1) contd....

References	Number of Cases	Age	Sex	Type of Trauma	Presenting Neurological Symptoms	Trauma - Symptoms Interval	Before Diagnosis Imaging	Type of ICA Lesions	Other Correlated Findings
Duke <i>et al.</i> 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 <i>et al.</i> 1997	1	20ys	1 F	MVA (no seat belt)	Carotidynia, unilateral oculosympathetic paresis, unilateral loss of limbs sensation, hemiparesis	3 days	Cervical spine X-rays (soon after the accident), arteriography	Right ICAD at C1 level with pseudoaneurysm	-
Vishteh <i>et al.</i> 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 <i>et al.</i> 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 <i>et al.</i> 1998	1	45ys	1 M	Vomiting	hemiplegia, one eye loss of vision, slurred speech, a 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 <i>et al.</i> 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 <i>et al.</i> 1998	4	35ys (range 31-39)	3 M, 1 F	Softball neck direct impact (2), car accidents (2)	None (only the softball cases are described)	-	Head CT, angiography	ICAD with intimal flap, aneurysm, maybe thrombosis	-
					Monolateral ptosis and miosis (only the softball cases are described)	3 days	DUS, MRA	ICAD	-
Simionato <i>et al.</i> 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 <i>et al.</i> 2000	1	43ys	1 F	Car accident (seat-belt 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

(Table 1) contd....

References	Number of Cases	Age	Sex	Type of Trauma	Presenting Neurological Symptoms	Trauma - Symptoms Interval	Before Diagnosis Imaging	Type of ICA Lesions	Other Correlated Findings
Duncan <i>et al.</i> 2000	1	39ys	1 M	Car accident (seat-belt 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 <i>et al.</i> 2000	1	27ys	1 F	Motorcycle accident	Progressive loss of consciousness	Several hours	Brain CT, angiography	Bilateral ICAD	VAD; cerebral infarct
Hughes <i>et al.</i> 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 persistent headache, transient visual disturbances such as unilateral scotoma and “granular” vision, transient complete blindness, unilateral ptosis and anisocoria	< some hours 1 day	- Head CT (normal at day 2), ophthalmoscopic examination (day 9), dilated funduscopic examination, MRI and MRA (day 11)	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
Malek <i>et al.</i> 2000	2	37ys 44ys	2 F	Strangulation MVA (whiplash injury)	Upper limbs weakness, leg numbness, and dysphasia Dysphasia, unilateral upper limb weakness and numbness	-	-	ICAD	-
Scavée <i>et al.</i> 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 <i>et al.</i> 2002	1	18ys	M	Gunshot	Not appreciable (sedated)	-	Head, face, and cervical spine CT, angiography	ICA pseudoaneurysm	Distal embolic angular artery occlusion
Duane <i>et al.</i> 2002	2	31ys 27ys	F F	Strangulation and stabbing attempt Gun shot	Seizure, tongue deviation -	8 days -	Neck CT, angiography Head x-ray, angiography	ICA pseudoaneurysm ICA AVF with pseudoaneurysm	peritonsillar abscess -
Blanco Pampin <i>et al.</i> 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

(Table 1) contd....

References	Number of Cases	Age	Sex	Type of Trauma	Presenting Neurological Symptoms	Trauma - Symptoms Interval	Before Diagnosis Imaging	Type of ICA Lesions	Other Correlated Findings
-	-	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 <i>et al.</i> 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 <i>et al.</i> 2004	1	37ys	M	Car accident	One upper limb weakness episodes	15 years	Cervical MRI, MRA	ICA pseudoaneurysm	-
Fanelli <i>et al.</i> 2004	1	17ys	1 M	Motorcycle accident	Hemiplegia and positive Babinski's sign	< some hours	Head CT	Bilateral ICAD	Right hemisphere cerebral infarct
Payton <i>et al.</i> 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 <i>et al.</i> 2005	1	52ys	1 M	Car accident	Altered consciousness, hemisyn-drome	< some hours	Craniocervicalthoracic CT	ICAD with tight stenosis and luminal thrombosis	Cerebral arteries' filling defects related to thromboembolic events
Clarot <i>et al.</i> 2005	2	38ys	1 M	Attempted strangulation	Altered consciousness, bilateral Babinski's sign, permanent eye elevation, bradycardia, and right hemiparesis	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
		42ys	1 F		Headache	2 days	DUS, brain CT	Bilateral CAD	-
Cohen <i>et al.</i> 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 up to 19 days	Brain CT, angiography	ICAD	-
Cothren <i>et al.</i> 2005	46	32±2 ys	65% M, 35% F	MVA, falls, skiing injuries	Not specified, 38 patients asymptomatic, 8 patients symptomatic	-	Angiography	Pseudoaneurysm	-
Joo <i>et al.</i> 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	

(Table 1) contd....

References	Number of Cases	Age	Sex	Type of Trauma	Presenting Neurological Symptoms	Trauma - Symptoms Interval	Before Diagnosis Imaging	Type of ICA Lesions	Other Correlated Findings		
de Borst <i>et al.</i> 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 <i>et al.</i> 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 <i>et al.</i> 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 ray, angiography			ICAD	Frontal scalp laceration, some cranial and C2 fractures, pneumocranium, subarachnoid hemorrhage
		48ys		Altered consciousness, visual acuity reduction, extraocular movements alteration, hemiparesis	7 days	Brain and facial CT, angiography	Multiple craniofacial fractures, haemorrhagic ACA and MCA infarct				
Jariwala <i>et al.</i> 2006	1	17ys	F	Car accident	Progressive consciousness alteration, hemiparesis and sensation loss	< some hours	Brain and neck spine CT ----- Brain CT, MRI, MRA	ICAD	MCA and partially PCA infarct		
Pierrot <i>et al.</i> 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 <i>et al.</i> 2007	1	7ys	1 M	Playing at a water park	Head and neck pain, vomiting, hemiparesis, Babinski's sign, hemifacial palsy with slurred speech and uvula deviation	< some hours	Brain CT, MRI, MRA, angiography	ICAD	Acute cerebral infarct		
Lo <i>et al.</i> 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 <i>et al.</i> 2007	1	28ys	1 M	Bungee jumping (no fall)	Right arm paraesthesia	< some hours	Neck US, brain MRI, MRA	ICAD with intramural haematoma	-		
Schulte <i>et al.</i> 2008	2	27 and 39ys	1 M, 1 F	Blunt neck trauma	TIA, headache, vertigo	-	DUS, CTA	CAD	-		
Fuse <i>et al.</i> 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,		

(Table 1) contd....

References	Number of Cases	Age	Sex	Type of Trauma	Presenting Neurological Symptoms	Trauma - Symptoms Interval	Before Diagnosis Imaging	Type of ICA Lesions	Other Correlated Findings
Flaherty and Flynn 2008	1	34ys	F	Hand assault (hit on the face)	Horner's syndrome	4 days	Brain CT, neck CTA	ICAD	-
Vadikolias <i>et al.</i> 2008	1	48ys	M	Intense jackhammer use	Hemiparesis	< some hours	Brain CT, DUS, TCD	ICAD	MCA infarct with haemorrhage
Moriarty <i>et al.</i> 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 <i>et al.</i> 2012	1	49ys	F	Strangulation attempt	Altered consciousness	-	Brain and neck CTA	Bilateral ICAD	Neck strangulation groove
Keilani <i>et al.</i> 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 <i>et al.</i> 2011	1	55ys	F	MVA	-	-	CTA, angiography with IVUS	ICAD	Several other lesions, no brain injury
Herrera <i>et al.</i> 2011	14	-	-	Gunshot and stab injuries	Bleeding, pulsatile mass, neck bruit, hematoma, stroke, dementia syndrome	-	-	Pseudoaneurysm, AVF, dissection, active bleeding	-
Fridley <i>et al.</i> 2011	1	40ys	M	Wakeboarding	Headache, hemiplegia	1 day	Head CT, MRI, MRA, angiography	ICAD	Unilateral basal ganglia and internal capsule infarct
Taşçılar <i>et al.</i> 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 <i>et al.</i> 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

(Table 1) contd....

References	Number of Cases	Age	Sex	Type of Trauma	Presenting Neurological Symptoms	Trauma - Symptoms Interval	Before Diagnosis Imaging	Type of ICA Lesions	Other Correlated Findings
		19ys	M	Motorbike accident	Altered consciousness, different blood pressure between the arms Still altered consciousness, spontaneous bilateral stretching of both arms and hemiparesis	< some hours 7 and 8 days	Aortic CTA Brain CT and CTA	-	Multiple cranial and skull base fractures, multiple intracerebral hematomas, ACA and MCA infarct
Cohen <i>et al.</i> 2012	23	44ys (range 17-66)	19 M, 4 F	Multiple trauma (11), penetrating neck injury (2), minor cervicocranial trauma (10)	Ischaemic stroke symptoms (14), TIA (3), Horner's syndrome (1), carotidynia (1)	2 hours up to 21 days	Head and neck CT, CTA (all)	ICAD	-
Makhlouf <i>et al.</i> 2013	1	60ys	1 F	Hand assault (hit on the head)	Headache Unilateral facial palsy and Horner's syndrome	< some hours 3 months	Cervical X-rays Brain MRI and MRA	ICAD with pseudoaneurysm	Unilateral corona radiata infarct
Prasad <i>et al.</i> 2013	1	22ys	F	MVA	Altered consciousness	< some hours	Head CT, angiography	ICAD with AVF	Multiple facial fractures, subarachnoid haemorrhage, cerebral oedema
Seth <i>et al.</i> 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 <i>et al.</i> 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 <i>et al.</i> 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 hypoaesthesia				
Kalantzis <i>et al.</i> 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 trauma	Transient loss of consciousness Headache, unilateral visual loss, hemiparesis, unilateral hyperreflexia and Babinski's sign	< some hours 48 hours	- Brain CT, MRI, angiography	ICAD with stenosis	Neck abrasion, carotid bruit; acute cerebral infarct

(Table 1) contd....

References	Number of Cases	Age	Sex	Type of Trauma	Presenting Neurological Symptoms	Trauma - Symptoms Interval	Before Diagnosis Imaging	Type of ICA Lesions	Other Correlated Findings
Crönlein <i>et al.</i> 2015	1	28ys	1 F	Car accident	Altered consciousness, head pain, anisocoria	< some hours	Total body CT ----- CTA, US	Bilateral ICAD	Unilateral central region cerebral infarct
Uhrenholt <i>et al.</i> 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 <i>et al.</i> 2016	39	41ys	22 M, 17 F	-	-	-	Head and neck CTA	ICA pseudoaneurysm (bilateral in 4 cases)	Cerebral infarct (7)
Griessenauer <i>et al.</i> 2016	2	21ys	1 M, 1 F	MVA	Altered consciousness	< some hours	Head CT, CTA	ICA aneurysm	Cranial and facial fractures, intracranial haemorrhage
Taoussi <i>et al.</i> 2017	1	29ys	F	Car accident	Dysphasia, upper limb hemiparesis,	< 12 hours	MRI	Bilateral ICAD	Multiple cerebral infarct
Cebeci <i>et al.</i> 2018	1	10ys	1 M	Trivial shoulder trauma	Headache, speech impairment, vomiting, and facial paralysis	6 hours	Head MRI and MRA	ICAD	.
Ariyada <i>et al.</i> 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
					Blairiness	1 month	CT angiography, MRA, DSA	Bilateral ICAD	VAD with thrombus
Gabriel <i>et al.</i> 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, unilateral facial droop	48 hours			
Petetta <i>et al.</i> 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 <i>et al.</i> 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	Total articles 77	-	-	-	-	-

Abbreviations: 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.

Table 2. Traumatic events causing internal carotid artery dissection in the literature. Only cases in which the traumatic event was reported are included. MVA indicates a 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, 112]
	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	

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 that the intimal injuries were produced by hyperextension and lateral flexion of the neck, which caused the artery wall to be

stretched. An additional two cases were described by Krajewski and Hertzner, while another series of six cases were reported by Zelenock *et al.* [21, 22]. In their work, they reported the causes to be motor vehicle accidents in three cases, falls from less than three metres in two cases, and 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 manifestations at least two weeks after 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]. Mokri *et al.* reported 18 cases of extracranial ICAD as a consequence of blunt head or neck trauma [25]. Again, motor vehicle accidents were the major cause. Watridge *et al.* described 24 cases of patients admitted to their medical centre after trauma [26]. The presenting symptoms varied (hemiparesis, aphasia, *etc.*) No patients presented with external signs of a direct neck injury, while two patients had cervical and thoracic spinal fractures. Prompt head CT scans were performed in all cases, but 17 of the 24 patients 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 CADs and six bilateral CADs. 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 deficits than spontaneous dissections.

3.2. Brief Description of the Studies Published from 1990 to the Present

From 1990 to the present, 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 common (94/227 cases, Table 2). For example, Reddy *et al.* reported the autopsy of a woman who developed ICAD as a consequence of a car accident [28]. The authors suggested that arterial injury was caused by 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 haematoma. In addition, angiography also showed tortuosity of the artery, which in the authors' opinion could predispose patients to dissection in case of a traumatic event. A series of six cases concerning ICADs from motor vehicle accidents highlighted the importance of initial patient evaluations and timely angiography examinations [30]. In fact, in four of those cases, the diagnosis of ICAD was made within 6 hours of hospital admission, while in the remaining cases, the patients were diagnosed within at least the third day of hospitalization. All patients showed normal ICA contours at the last follow-up angiography, even though three of them still had neurological deficits. Another case of ICAD subsequent to a motor vehicle accident was described by Matsuura *et al.* [31]. In this case, a woman was driving without a fastened seat belt. She developed neurological symptoms after three days of hospitalization, and angiography was performed, revealing a right ICAD with a pseudoaneurysm. Conversely, Babovic *et al.* reported the case of a woman who was driving her car with a fastened seat belt 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, they found that the woman had bilateral ICADs 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 angiography diagnosed bilateral ICADs with a thrombus in the right ICA. The authors suggested the aetiological role of airbag deployment. In addition, 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 Uhrholt *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 experience any other injury. Another interesting case was published by Fusonie *et al.* [36]. A young man experienced 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 a right ICA pseudoaneurysm and underwent covered stent exclusion; afterward, he did not experience any other episodes. In many other works, motor vehicle accidents, with or without direct head/neck trauma, were the cause of ICAD [8, 37-56]. In contrast, only three cases of post-traumatic internal carotid artery lesions related to bicycle accidents have been reported [7, 57, 58].

Some cases described horse riding accidents [46, 59, 60]. A fall from height was the cause of ICAD in 11/227 cases [8, 22, 24, 55, 61, 62].

Direct, blunt trauma to the neck is another possible mechanism of ICA lesions. 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 seven cases, ICAD was an incidental finding during cervical spine and/or brain magnetic resonance imaging (MRI) or angiography performed for other reasons. No evidence of cerebral infarct was seen on brain CT, and the patients did not present any neurological symptoms correlated to ICAD. Lo *et al.* collected 18 cases of post-traumatic ICA lesions (10 pseudoaneurysms) and suggested a correlation with craniofacial fractures [70]. Unfortunately, the authors did not specify the traumatic causes 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 athletics, both in cases with and without some kind of trauma [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 following wakeboarding [77]. Zhou *et al.* published the case of a young man who went bungee jumping and experienced neck pain after ten minutes [78]. Some hours later, he also experienced paraesthesia in one arm. A carotid artery ultrasound and then brain MRA revealed left CCA and ICA dissection with intramural haematoma. In another case, a man developed a headache during taekwondo training [79]. Days later, he developed progressive neurological deficits, such as aphasia, visual disturbances, hemi-

paresis, and sensory loss. A brain CT scan followed by MRI and MR angiography revealed 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, the TICADs occurred 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 in one eye, slurred speech, and a decrease in consciousness. An MRI showed an infarct in the anterior cerebral artery (ACA) and MCA territory, a left ICAD with occlusion, and a right ICA intimal flap with normal blood flow. Lee and Jensen also described a case of bilateral ICAD following minor trauma [81]. Their patient developed headache and visual disturbances days after riding a mountain bicycle despite not having any accidents 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 were also reported by Alimi *et al.* [83]. The authors described a case of ICA stenosis after cervical manipulation and identified neck hyperextension as the cause of the arterial lesion. In the study by 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 by a screening MRI [84]. Pezzini *et al.* reported a case of ICAD after playing the French horn. The patient also had 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 with penetrating neck 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 injuries [88].

With regard to the paediatric population, aside from the previously mentioned work by Morgan *et al.* [24], in the literature, there are at least 14 cases of children (< 16 years old) who developed TICAD, often in relation to minor trauma [58, 89-96]. In particular, the causative event was trauma to the soft palate/pharynx (fall while holding 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 specifically report the traumatic causes of 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].

3.3. Case Report

A 54-year-old man with no medical history was involved in a high-speed head-on collision against a lamppost while driving a truck. The truck's frame was highly damaged during the impact. The man experienced a sudden transient loss of consciousness soon after the accident. He was immediate-

ly transferred to the local emergency department, and the first evaluation revealed a blood pressure of 130/85 mmHg, heart rate of 85 bpm, oxygen saturation of 96%, right frontal skin abrasion, crush injuries of the right foot with an exposed fracture, and normal neurological, thoracic, and abdominal examination results. The patient was agitated, and therefore 20 mg of midazolam was administered. An X-ray examination of the right foot confirmed displaced fractures in the tibia and fibula. A whole-body CT scan without contrast was also performed, showing a displaced fracture of the right arc of the C1 vertebra with atlanto-occipital disarticulation; multiple left pulmonary contusions associated with pneumatocele; and a fracture in the D10 vertebral body. No ischaemic or haemorrhagic brain injuries were present. A cervical collar was prescribed, and the patient was admitted to the Orthopaedic Department of the same hospital to undergo surgery for the foot fracture. Two days after admission, he complained that he could not move his left upper limb, and paralysis was confirmed during the physical examination. Therefore, brain CT plus CT angiography was performed, revealing a right posterior cortico-subcortical temporoparietal insular ischaemic lesion with a median shift and a right ICAD with almost complete lumen obstruction and consequent decrease in the right middle cerebral artery blood flow (Figs. 2 and 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, divergent strabismus of the right eye, bilateral miosis reactive to light stimuli, and Cheyne-Stokes respiration, while the vital signs were as follows: blood pressure 130/70 mmHg, heart rate 70 bpm, oxygen saturation 97% (85% in apnoea phases), and body temperature 36,6°C. The patient received oxygen therapy; the vital signs were constantly monitored. During the following hours, he experienced two episodes of left hemibody fasciculations and breathing alterations and was treated with lorazepam. A day later, he was comatose, with bilateral mydriasis and stertorous breathing. A brain CT scan showed progression of the ischaemic lesion with a 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 irrigated. Nevertheless, his neurological status deteriorated further until 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 (Fig. 4).

The right common, internal, and external carotid arteries were sampled and then studied after formaldehyde fixation (Figs. 5 and 6).

Histological examination was performed, confirming the presence of cerebral oedema and right ICAD. Specifically, the ICA presented an intramural haematoma with intimal and media laceration, and a thrombus was confirmed to be in the lumen (Fig. 7).

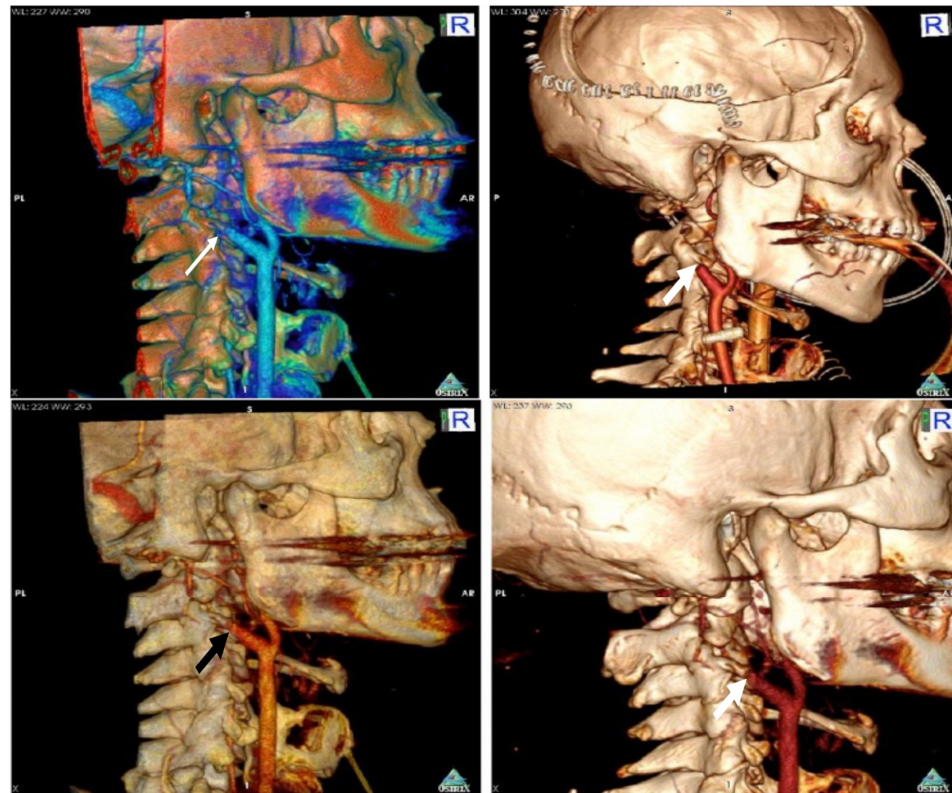


Fig. (2). TC angiography performed soon after neurologic manifestation showed a right ICAD with almost completed lumen obstruction and consequent right middle cerebral artery blood flow decrease. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

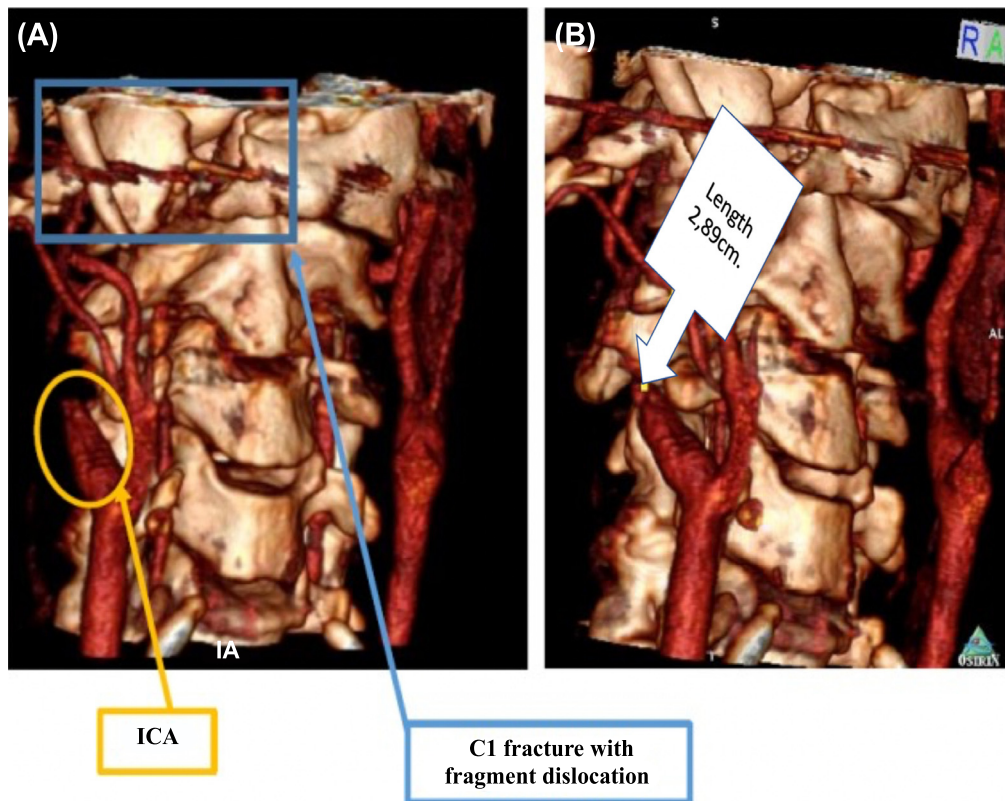


Fig. (3). CT 3D reconstruction details showing C1 dislocated fragment could not be the cause of the TICAD. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

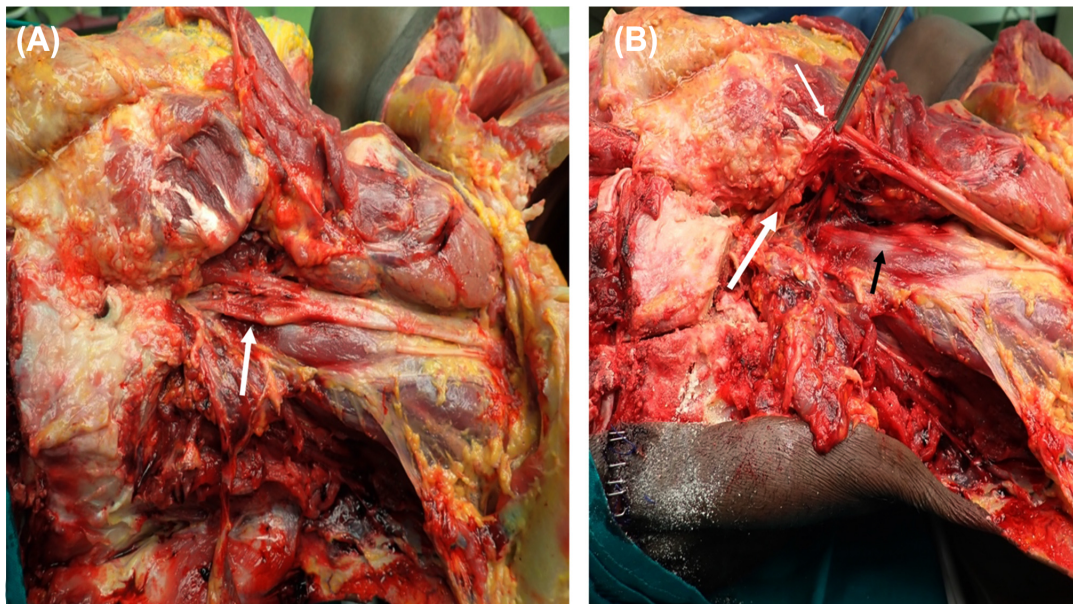


Fig. (4). Right common, internal, and external carotid arteries dissection. Right ICA showed a modest adventitial haemorrhagic infiltration a few centimetres upper than the carotid bifurcation. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

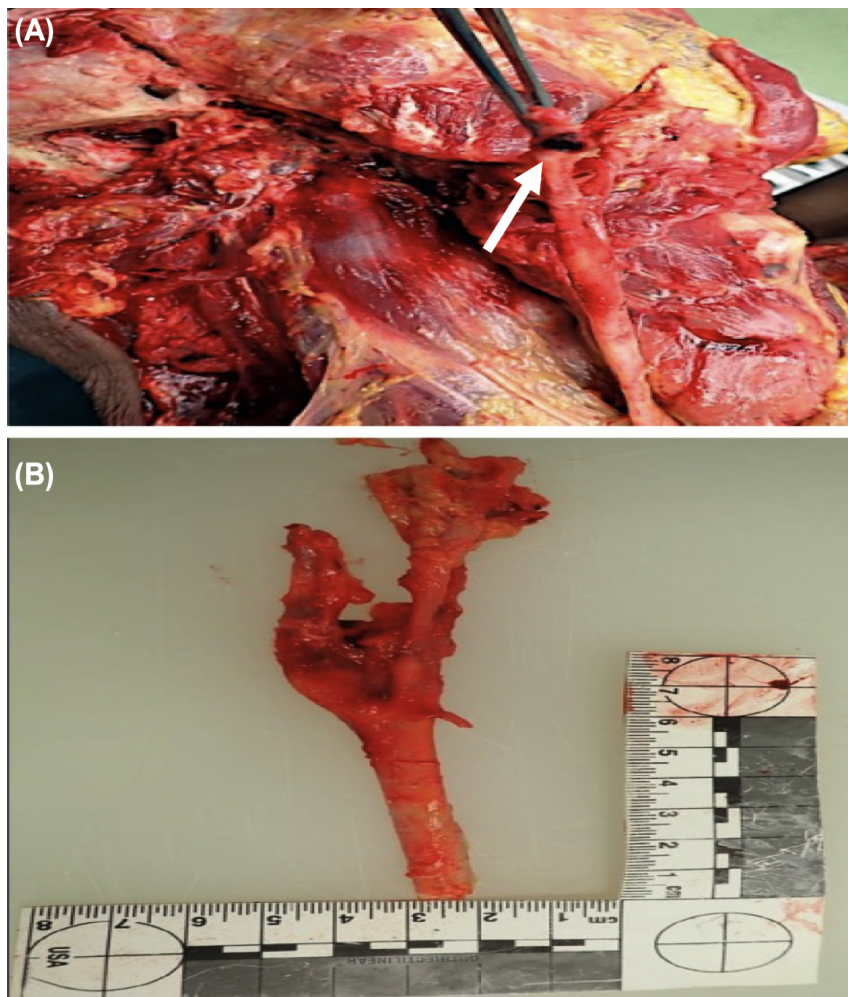


Fig. (5). Right common, internal, and external carotid arteries dissection and collection. ICA was sectioned at its petrous level. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

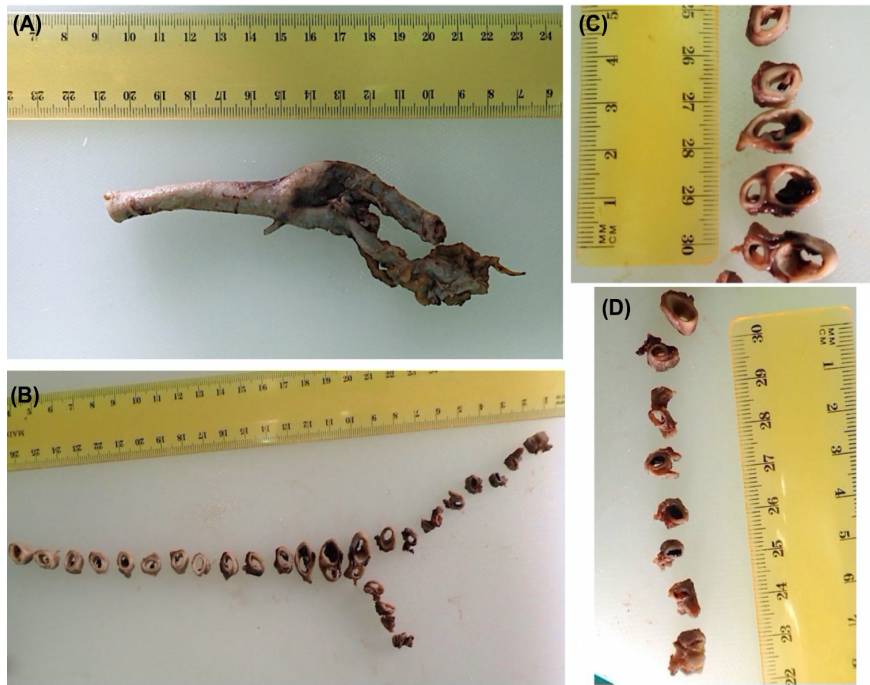


Fig. (6). Right common, internal, and external carotid arteries sample section and macroscopic. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

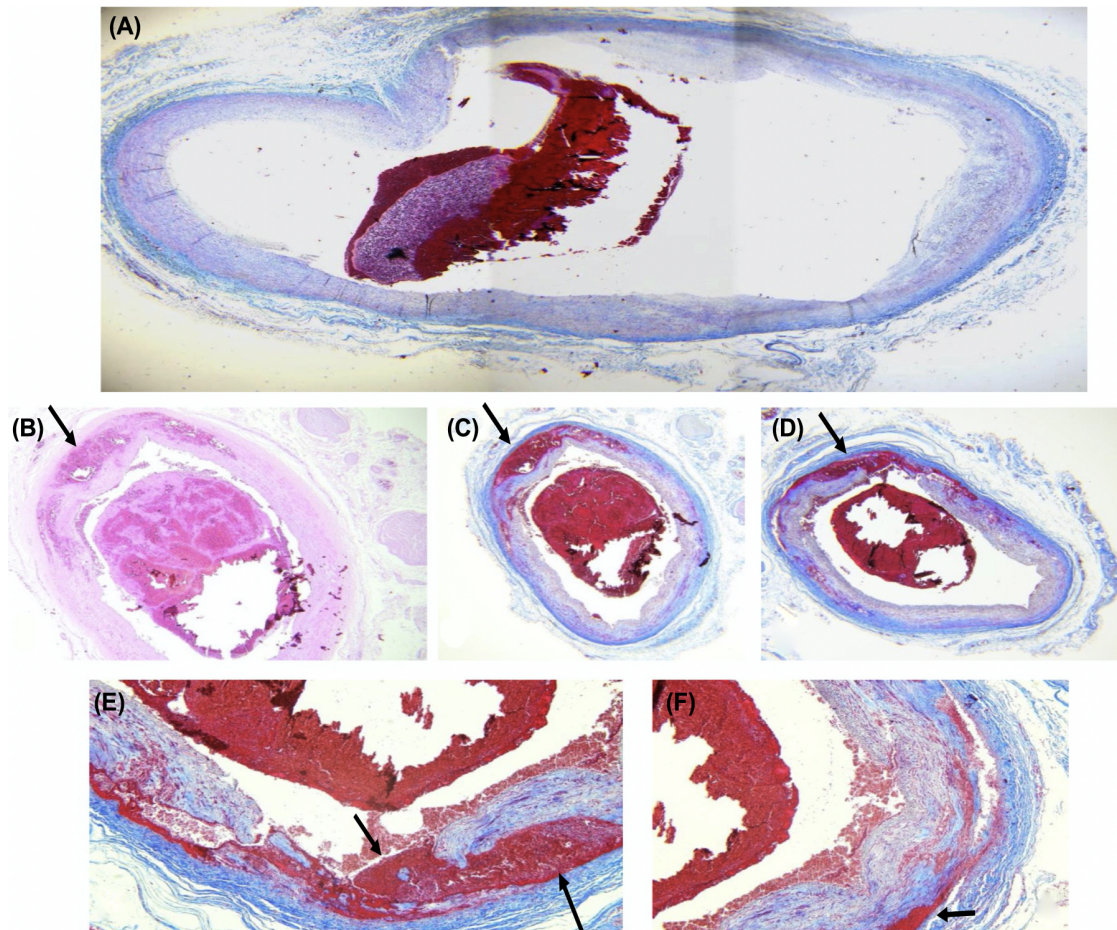


Fig. (7). Right ICAD histological examination revealed an intramural hematoma with intimal and media laceration and a thrombus into the lumen. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

4. DISCUSSION

We presented the case of a middle-aged man who was involved in a road traffic accident. He was transferred to the emergency department after a sudden transient loss of consciousness. No brain injuries were identified on the CT scan. Two days after hospitalization, while he was waiting for surgical treatment for a foot fracture, he developed left upper limb paralysis. Brain CT and CT angiography showed a large ischaemic lesion and right ICAD. Even though a decompressive hemicraniectomy was performed, he died after a few days. A forensic autopsy was required. This confirmed that right ICAD was the cause of brain injury.

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

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

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

Given the above, TICAD should be taken into consideration when a young adult or middle-aged patient presents with severe blunt trauma, although there are no specific guidelines regarding TICAD screening [9]. The risk factors for a blunt carotid injury that indicate examinations to exclude 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 craniofacial fracture [14].

In addition, understanding which kind of traumatic event is most associated with TICAD could help clinicians optimize their diagnostic process. In the literature, TICAD is mostly correlated with traffic accidents (41,4%), specifically to car accidents (at least 17,2%), and to direct or indirect head and cervical trauma (33,5%). Usually, TICAD is a consequence of a high-energy collision/blunt trauma, but in a few cases, TICADs due to trivial traumas have also been reported.

The mechanism of TICAD development has been mostly referred to as vigorous extension and flexion of the cervical spine and rotation of the skull. During such movements, the ICA is stretched, and the arterial wall may be damaged. Shear forces seem to be more intense where the ICA movement is averted by the surrounding anatomical structure, such as the skull base [57]. Nevertheless, TICAD could be found in both the extracranial and intracranial ICAs. When TICAD is extracranially located, neck duplex ultrasonography (DUS) could help to identify 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 precisely visualise the dissection [44]. An aggressive angiographic evaluation has also been proposed [108]. Brommeland *et al.* recommend applying the Denver screening criteria and then performing computed tomography angiography (CTA) in cases of blunt trauma [109]. Nevertheless, those indications have not yet been completely accepted by the scientific community, and there is no uniform screening strategy among physicians.

Regarding ICAD treatment, both medical and surgical management have been described. Endovascular and surgical approaches, while effective, pose a greater risk for severe complications than medical management. However, antithrombotic treatment has been shown to be especially effective in the management of carotid dissection during endovascular procedures. Indeed, a better outcome may be expected with the combination of intravenous thrombolysis and endovascular methods (stenting and thromboaspiration), as recent reports have suggested a better outcome after r-TPA treatment using stent-assisted intra-arterial thrombolysis [110]. While antithrombotic treatment has been shown to be effective in the management of carotid dissection, no treatment guidelines have favoured anticoagulation over antiplatelet agents. The majority of cerebrovascular dissections heal by themselves, and the risk of developing new ischaemic complications has been reported to be low; however, in the case of recurrent ischaemic symptoms, the outcomes can be devastating. This potential severity deems treatment necessary to prevent the development of permanent neurological deficits. Antithrombotic therapy with either antiplatelets or anticoagulants is the mainstay of treatment to prevent thromboembolic complications. Nevertheless, both antiplatelet and anticoagulation agents can be used in the management of intracranial carotid dissection with similar rates of new or recurrent ischaemic stroke, TIA, and hemorrhage [111].

With regard to the case presented in this paper, ICAD can be considered a consequence of motor vehicle accidents despite the absence of any signs suggesting a direct neck or head injury. In addition, from the neck CT images obtained during hospitalization and the autopsy findings, it was possible to exclude that the C1 fracture fragments were involved in ICAD development (Fig. 3). Nevertheless, the dissection was probably due to stretching or compression of the ICA as a consequence of 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 spe-

cific and internationally accepted guidelines leaves physicians alone when facing the matter of TICAD screening/diagnostic protocols. In our case, the reasons behind the diagnostic delay, other than the absence of specific guidelines, were the trauma-to-symptom interval (two days) and the presence of other injuries requiring timely surgery. Then, when the ICAD diagnosis was made, the brain was already gravely injured, so vascular repair surgery was not possible [112]. This case highlights the importance of screening guidelines to help physicians anticipate a TICAD diagnosis before symptoms develop and prevent permanent neurological impairment or attenuate poor prognoses.

CONCLUSION

TICAD is a rare condition largely described in correlation with traffic accidents. It mainly affects the young adult population, and it can cause 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 consensus among physicians. Therefore, medical liability claims correlated to TICAD are possible. The case reported in this paper is an emblematic example of a delayed TICAD diagnosis that resulted in patient death. This case highlights the need for screening guidelines to attenuate not only poor prognoses but also avoid medico-legal claims. Identifying which type of trauma is more likely to cause ICAD could be a good way to help increase suspicion for this infrequent condition, despite the absence of specific and internationally accepted guidelines. Through a literature review, we have confirmed that TICAD is mainly described as a consequence of traffic accidents. In such cases and, in general, when a direct or indirect neck injury is described, ICA stretching or compression needs to be suspected. Despite the absence of internationally accepted guidelines, a thorough and detailed trauma mechanism anamnesis is necessary to identify the cases in which MRI or angiography are indicated for early diagnosis of TICAD and to prevent devastating neurological outcomes.

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.

LIST OF 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.

CONSENT FOR PUBLICATION

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

STANDARDS OF REPORTING

PRISMA guidelines were followed for the study.

FUNDING

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

ACKNOWLEDGEMENTS

Declared none.

SUPPLEMENTARY MATERIAL

PRISMA checklist is available on the publisher's website along with the published article.

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