ISSN 2307-8960 (online)

# World Journal of *Clinical Cases*

*World J Clin Cases* 2020 October 26; 8(20): 4688-5069

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

Contents

Semimonthly Volume 8 Number 20 October 26, 2020

## MINIREVIEWS



## ORIGINAL ARTICLE

### Retrospective Cohort Study



### Retrospective Study

*World Journal of Clinical Cases*

**Semimonthly Volume 8 Number 20 October 26, 2020**

## Contents

*World Journal of Clinical Cases*

**Semimonthly Volume 8 Number 20 October 26, 2020**

## Contents

*World Journal of Clinical Cases*

**Semimonthly Volume 8 Number 20 October 26, 2020**

## Contents

*World Journal of Clinical Cases*

**Semimonthly Volume 8 Number 20 October 26, 2020**

# Contents

## ABOUT COVER

Peer-reviewer of *World Journal of Clinical Cases*, Dr. Aleem Ahmed Khan is a Distinguished Scientist and Head of The Central Laboratory for Stem Cell Research and Translational Medicine, Centre for Liver Research and Diagnostics, Deccan College of Medical Sciences, Kanchanbagh, Hyderabad (India). Dr. Aleem completed his Doctorate from Osmania University, Hyderabad in 1998 and has since performed pioneering work in the treatment of acute liver failure and decompensated cirrhosis using hepatic stem cell transplantation. During his extensive research career he supervised 10 PhD students and published > 150 research articles, 7 book chapters, and 2 patents. His ongoing research involves developing innovative technologies for organ regeneration and management of advanced cancers. (L-Editor: Filipodia)

## AIMS AND SCOPE

The primary aim of *World Journal of Clinical Cases* (*WJCC*, *World J Clin Cases*) is to provide scholars and readers from various fields of clinical medicine with a platform to publish high-quality clinical research articles and communicate their research findings online.

*WJCC* mainly publishes articles reporting research results and findings obtained in the field of clinical medicine and covering a wide range of topics, including case control studies, retrospective cohort studies, retrospective studies, clinical trials studies, observational studies, prospective studies, randomized controlled trials, randomized clinical trials, systematic reviews, meta-analysis, and case reports.

## INDEXING/ABSTRACTING

The *WJCC* is now indexed in Science Citation Index Expanded (also known as SciSearch®), Journal Citation Reports/Science Edition, PubMed, and PubMed Central. The 2020 Edition of Journal Citation Reports® cites the 2019 impact factor (IF) for *WJCC* as 1.013; IF without journal self cites: 0.991; Ranking: 120 among 165 journals in medicine, general and internal; and Quartile category: Q3.

## RESPONSIBLE EDITORS FOR THIS ISSUE



**NAME OF JOURNAL**
*World Journal of Clinical Cases*

**ISSN**
ISSN 2307-8960 (online)

**LAUNCH DATE**
April 16, 2013

**FREQUENCY**
Semimonthly

**EDITORS-IN-CHIEF**
Dennis A Bloomfield, Sandro Vento, Bao-Gan Peng

**EDITORIAL BOARD MEMBERS**
https://www.wjgnet.com/2307-8960/editorialboard.htm

**PUBLICATION DATE**
October 26, 2020



**INSTRUCTIONS TO AUTHORS**
https://www.wjgnet.com/bpg/gerinfo/204

**GUIDELINES FOR ETHICS DOCUMENTS**
https://www.wjgnet.com/bpg/GerInfo/287

**GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH**
https://www.wjgnet.com/bpg/gerinfo/240

**PUBLICATION ETHICS**
https://www.wjgnet.com/bpg/GerInfo/288

**PUBLICATION MISCONDUCT**
https://www.wjgnet.com/bpg/gerinfo/208

**ARTICLE PROCESSING CHARGE**
https://www.wjgnet.com/bpg/gerinfo/242

**STEPS FOR SUBMITTING MANUSCRIPTS**
https://www.wjgnet.com/bpg/GerInfo/239

**ONLINE SUBMISSION**
https://www.f6publishing.com

World Journal of Clinical Cases

Submit a Manuscript: https://www.f6publishing.com

*World J Clin Cases* 2020 October 26; 8(20): 4773-4784

DOI: 10.12998/wjcc.v8.i20.4773

ISSN 2307-8960 (online)

ORIGINAL ARTICLE

Retrospective Study

# Cerebral infarct secondary to traumatic internal carotid artery dissection

Guang-Ming Wang, Hang Xue, Zhen-Jie Guo, Jin-Lu Yu

**ORCID number:** Guang-Ming Wang 0000-0002-3736-8904; Hang Xue 0000-0002-4935-098X; Zhen-Jie Guo 0000-0003-4904-9852; Jin-Lu Yu 0000-0003-2329-7946.

**Author contributions:** Yu JL contributed to case conceptualization; Xue H and Wang GM contributed to data curation; Guo ZJ contributed to manuscript drafting; Wang GM and Yu JL contributed to manuscript review and editing.

**Institutional review board statement:** The study protocol was approved by the Institutional Review Board of the First Hospital of Jilin University.

**Informed consent statement:** Informed consent for publication of these cases was obtained from the patients or their relatives.

**Conflict-of-interest statement:** All authors have nothing to disclose.

**Data sharing statement:** No additional data are available.



**Guang-Ming Wang, Hang Xue, Zhen-Jie Guo, Jin-Lu Yu,** Department of Neurosurgery, The First Hospital of Jilin University, Changchun 130021, Jilin Province, China

**Corresponding author:** Jin-Lu Yu, MD, MSc, PhD, Doctor, Professor, Department of Neurosurgery, The First Hospital of Jilin University, No. 71 Xinmin Avenue, Changchun 130021, Jilin Province, China. jlyu@jlu.edu.cn

## Abstract

*BACKGROUND*

Traumatic internal carotid artery dissection (TICAD) is rare and can result in severe neurological disability and even death. No consensus regarding its diagnostic screening and management has been established.

*AIM*

To investigate the clinical presentation, imaging features, diagnostic workup, and treatment of TICAD.

*METHODS*

In this retrospective case series, emergency admissions for TICAD due to closed head injury were analyzed. The demographic, clinical, and radiographic data were retrieved from patient charts and the picture archiving and communication system.

*RESULTS*

Six patients (five males and one female, age range of 43-62 years, mean age of 52.67 years) presented with TICAD. Traffic accidents (4/6) were the most frequent cause of TICAD. The clinical presentation was always related to brain hypoperfusion. Imaging examination revealed dissection of the affected artery and corresponding brain infarction. All the patients were definitively diagnosed with TICAD. One patient was treated conservatively, one patient underwent anticoagulant therapy, two patients were given both antiplatelet and anticoagulant drugs, and two patients underwent decompressive craniectomy. One patient fully recovered, while three patients were disabled at follow-up. Two patients died of refractory brain infarction.

*CONCLUSION*

TICAD can cause catastrophic outcomes and even refractory brain hernia. Early and efficient diagnosis of TICAD is essential for initiating appropriate treatment.

**Manuscript source:** Unsolicited manuscript

**Received:** April 23, 2020
**Peer-review started:** April 23, 2020
**First decision:** April 29, 2020
**Revised:** August 16, 2020
**Accepted:** September 3, 2020
**Article in press:** September 3, 2020
**Published online:** October 26, 2020

**P-Reviewer:** Figueiredo EG, Kawabata H
**S-Editor:** Zhang L
**L-Editor:** Filipodia
**P-Editor:** Liu JH

The treatment of TICAD is challenging and variable and is based on clinician discretion on a case-by-case basis.

**Key Words:** Internal carotid artery dissection; Brain infarction; Treatment; Prognosis



**Core Tip:** Traumatic internal carotid artery dissection is a rare and acute condition. Early diagnosis and intervention can improve the prognosis and quality of life of patients. Case presentations and a literature review may provide insight into the pathology, clinical manifestations, imaging features, diagnosis, and treatment.

**Citation:** Wang GM, Xue H, Guo ZJ, Yu JL. Cerebral infarct secondary to traumatic internal carotid artery dissection. *World J Clin Cases* 2020; 8(20): 4773-4784
**URL:** https://www.wjgnet.com/2307-8960/full/v8/i20/4773.htm
**DOI:** https://dx.doi.org/10.12998/wjcc.v8.i20.4773

## INTRODUCTION

Traumatic internal carotid artery dissection (TICAD) at an extracranial or intracranial location is rare but can result in severe neurological disability or death and is often secondary to motor vehicle accidents, sports accidents, amusement park activities, and cervical chiropractic manipulation[1-5]. The morbidity rate associated with TICAD ranges from 23%-28%, and 48%-58% of survivors have significant neurological complications[6,7].

However, only 6% of blunt TICAD cases are diagnosed at the time of initial presentation and medical evaluation[8]. Therefore, screening of asymptomatic TICAD patients after traumatic accidents is recommended as timely recognition and appropriate treatment are vital to avoid fatal brain infarction[9,10].

Currently, the diagnosis and treatment of TICAD are incompletely understood. As TICAD is uncommon, studies of this entity are limited to case reports, and few studies have depicted the clinical and radiologic course of TICAD. In the present study, we describe six patients who were diagnosed with TICAD and provide a literature review of TICAD.

## MATERIALS AND METHODS

### *Data collection*

All patients included in the study sustained different types of traumatic injury and were confirmed to have TICAD at the First Hospital of Jilin University between July 2013 and April 2018. The study protocol was approved by the Institutional Review Board of the First Hospital of Jilin University. Informed consent for publication of these cases was obtained from the patients or their relatives.

Data were retrospectively collected from the patients' medical files, treatment reports, and follow-up notes. The Glasgow Coma Scale was used to evaluate the patients' neurologic status[11]. The patients' age, sex, symptoms, and onset time after injury were recorded, and their imaging data were obtained.

Follow-up was performed based on a clinical consultation or telephone interview. The Glasgow Outcome Scale (GOS) was used to assess prognosis, where GOS 1 indicated death, 2 indicated a persistent vegetative state, 3 indicated severe disability, 4 indicated moderate disability, and 5 indicated a good recovery[12].

### *Literature collection*

Eligible English-language articles (case reports, case series, and studies investigating TICAD) were identified by searching PubMed publications (the last search date was April 2020). The search algorithm used the terms "Traumatic internal carotid artery dissection" as the key word. The reference lists of the identified articles were also

manually searched for additional studies. The resulting flowchart is depicted in Figure 1.

The inclusion criteria were as follows: (1) The full text was available; (2) Clinical data were complete; and (3) All the cases in the articles were TICAD cases. Studies without sufficient descriptions of TICAD were excluded. After a review of the obtained literature, the current status of TICAD was discussed in terms of clinical features, radiological features, treatment, and prognosis.

## RESULTS

### *Patient demographics*

Six patients with closed head injuries were enrolled in the current study. Of these 6 patients (5 males and 1 female, age range of 43-62 years, mean age of 52.67 years), four were involved in traffic accidents, including car accidents ($n$ = 2) and motorcycle accidents ($n$ = 2), one patient fell from a height, and one patient suffered an amusement park injury.

### *Initial syndrome*

The six patients had no symptoms immediately after the traumatic incidents; the interval from injury to symptom onset was 4-45 h, and the mean interval was 15 h. The initial symptoms included paralysis ($n$ = 2), disturbance of consciousness (drowsiness or confusion) ($n$ = 2), headache ($n$ = 1), and neck pain ($n$ = 1). Four patients had a Glasgow Coma Scale score of 15, and two patients had scores of 14 and 13.

### *Imaging examination*

Computed tomography (CT), CT angiography (CTA), duplex ultrasonography (DU), digital subtraction angiography (DSA), magnetic resonance imaging (MRI), and transcranial doppler (TCD) were selectively performed in these six patients, which revealed TICAD, cerebral infarction, and insufficient intracranial blood flow. CT and CTA were performed in three cases. CT, CTA, and MRI were performed in one case. CT, CTA, MRI, DU, and DSA were performed in one case. CT, CTA, MRI, DU, and TCD were performed in one case.

In all six patients, CT demonstrated varying degrees of cerebral infarction. A hyperdense middle cerebral artery (MCA) sign on CT was visible in one CT scan. CTA, DU, DSA, and MRI revealed an intimal flap ($n$ = 2), tapering stenosis ($n$ = 5), and the appearance of an intramural hematoma ($n$ = 2).

In case 1, CTA and DSA clearly showed a rat tail sign at the beginning of the right internal carotid artery (ICA) (Figure 2B-F), and DU revealed that the true ICA lumen opened and shunted in synchrony with the vessel's pulse (*i.e.* the "open and close sign") (Figure 2E). MRI demonstrated a continuous crescentic high signal in the right ICA and an elongated, round high signal in the right MCA (Figure 2C and D), indicating the probable diagnosis of subacute dissection extending from the origin of the right ICA to the intracranial artery. Regarding case 2, a hyperdense MCA sign may be a predictor of rapidly progressive malignant lethal cerebral infarction. A later CT scan detected cerebral infarction (Figure 3B) and brain contusion (Figure 3A). Concomitant polytraumatic injuries (such as mandibular fracture) were also observed (Figure 3D). In cases 1 and 3, the left ICA was completely occluded (Figures 2B-F and Figure 4C), but the patients did not experience catastrophic cerebral infarction. CTA (Figures 2B and Figure 4B) and TCD (Figure 4E) indicated the presence of blood supply in the left MCA. The carotid compression test revealed the openness of the anterior communicating artery (Figure 4G), and in case 3, a cervical vertebra fracture was also revealed (Figure 4C). In case 5, MRI showed right ICA dissection manifested as a partial flow-void lumen surrounded by high-intensity signals due to an intramural hematoma (Figure 5D). In case 6, the patient exhibited deteriorating levels of consciousness, and decompressive craniectomy was performed to relieve the refractory high intracranial pressure (Figure 6).

### *Associated polytrauma*

Five of the six patients sustained polytraumatic injuries, including clavicular fracture ( $n$ = 1), brain contusion and mandibular fracture ($n$ = 1), skull base fracture and C6 fracture ($n$ = 1), and skull base fracture ($n$ = 2).

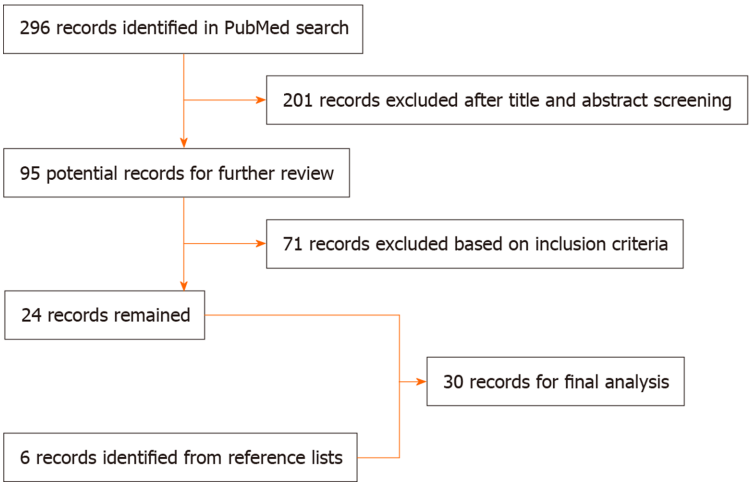


**Figure 1 Flowchart showing the selection of related references.**

### *Final diagnosis*

The final diagnosis in the six cases was TICAD.

### *Treatment*

One patient underwent anticoagulant therapy. Two patients were given both antiplatelet and anticoagulant drugs. Decompressive craniectomy was performed as a last-resort procedure to manage efficiently refractory brain infarction in two patients. One patient underwent conservative treatment (without antiplatelet or/and anticoagulant drugs or surgery).

### *Outcomes and follow-up*

During hospitalization, two patients died of refractory brain infarction. Outpatient follow-up (range: 1-4 years) was performed in the four surviving patients. Neurological function examinations were carried out using the GOS. The results were as follows: One patient fully recovered and had a score of 5, and three patients were disabled (one patient had a score of 4, and two patients had a score of 3). The clinical data are summarized in Table 1.

## DISCUSSION

TICADs are markedly different from spontaneous dissections or dissections occurring in everyday life (cough, nose blowing, rapid head turning, or rapid neck extension)[13-15]. TICAD frequently occurs when high-speed traumatic cerebrovascular injuries are sustained, such as those sustained in motor vehicle crashes, car crashes, assaults, falls, or amusement park activities or when hanging[16-18]. The overall mortality rate associated with TICAD ranges from approximately 20%-40%[19,20]. In our case series, the mortality rate was 33.3% (2/6), and these two patients died of refractory brain infarction.

In most TICAD cases, the pathological background is disruption of the intima, which causes luminal narrowing or even occlusion due to collapse of the false lumen against the true lumen and interferes with the blood flow in the primary lumen[13,21]. If dissection occurs between the media and adventitia, complete or partial transection can also occur, leading to flow-related pseudoaneurysm pouch formation[22,23].

Patients with TICAD present with a variety of symptoms and signs ranging from mild symptoms, such as headache, neck pain, or cranial nerve palsy, to more serious symptoms, such as stroke and even high intracranial pressure[1]. Usually, a latent period exists between the time of injury and symptom onset, which can complicate the diagnosis[24]. The average time for stroke to develop is 12-75 h post-trauma[3,5,25]. In our six cases, the asymptomatic period ranged from 4-45 h (average 15 h), which is similar to a previous report.

One of the main goals in the management of this condition is to establish an accurate diagnosis before stroke occurs[26]. Many studies have reported a correlation between TICAD and traumatic injury near the neck, especially with cervical and facial

**Table 1 Characteristics of the study cohort**

| No. | Sex/age | Cause of trauma | Asymptomatic period in h | Initial syndrome | GCS | Associated polytrauma | Imaging | Treatment | Follow-up in yr | GOS |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M/51 | Car crash | 4 | Confusion | 13 | Skull base fracture | CT, CTA, MRI, DU and DSA | Anticoagulant | 3 | 5 |
| 2 | M/52 | Motorcycle crash | 6 | Drowsiness | 14 | Brain contusion and mandibular fracture | CT and CTA | Conservative treatment | NA | 1 |
| 3 | M/62 | Car crash | 45 | Neck pain | 15 | Skull base fracture and C6 fracture | CT, CTA, MRI, DU and TCD | Anticoagulant and antiplatelet | 4 | 4 |
| 4 | M/43 | Motorcycle crash | 10 | Paralysis | 15 | Clavicular fracture | CT and CTA | DC | NA | 1 |
| 5 | F/61 | Amusement park accident | 12 | Paralysis | 15 | None | CT, CTA and MRI | Anticoagulant and antiplatelet | 3 | 3 |
| 6 | M/47 | Fall from height | 13 | Headache | 15 | Skull base fracture | CT and CTA | DC | 1 | 3 |

CT: Computed tomography; CTA: Computed tomography angiography; DC: Decompressive craniectomy; DSA: Digital subtraction angiography; DU: Duplex ultrasonography; F: Female; GCS: Glasgow Coma Scale; GOS: Glasgow Outcome Scale; M: Male; MRI: Magnetic resonance imaging; NA: Not applicable; TCD: Transcranial Doppler.

fractures[2,27,28]. The main risk factors for TICAD after severe traumatic brain injury are fracture involving the carotid canal, cervical spine injury, and thoracic trauma[16,29-31]. A recent study found that 42% of patients with polytrauma including TICAD had cervical spine fractures[16,32]. In our case series, 5/6 (83.33%) patients had multiple injuries near the ICA.

The diagnosis of TICAD is based mainly on neuroimaging findings. When TICAD occurs, the purpose of imaging examinations is to detect the cerebral parenchyma and the corresponding supply artery. The cerebral parenchyma can easily be visualized when performing CT or MRI[33]. TICAD may be detected by conventional DSA, DU, CTA, MRI, and magnetic resonance angiography[14,34,35]. In these imaging examinations, the characteristics of TICAD may include an intimal flap, a double lumen, tapering stenosis, tandem occlusions, the appearance of an intramural hematoma, and sometimes a small pseudoaneurysm[36]. TCD can also sometimes reveal a low blood flow velocity in the affected artery[4]. Early CT scans often do not show significant abnormalities, but high-density images of the MCA may occasionally be viewed if thrombosis of the MCA occurs[37].

TICAD must be treated as cerebral infarction can occur several hours after traumatic injury, when most patients are outside the narrow therapeutic window for timely therapy. However, considering the complexity and difficulty of the diagnosis of TICAD, in certain circumstances, the major goals of treatment are to prevent thromboembolic and hemodynamic injury[25,38]. Thus, when TICAD is suspected, antiplatelet and anticoagulant agents are required. However, medical management may be contraindicated in some cases, such as in case 2 in our study in which the CT scan revealed brain contusion. Furthermore, to avoid an increased risk of intracranial hemorrhage, we did not use any medical therapy. As recently described, stenting of the carotid arteries by an endovascular approach may provide immediate revascularization and can improve perfusion and limit embolus formation in patients presenting with embolic or hemodynamic symptoms[38]. However, in our study, the definitive diagnosis was determined too late for our six patients to be treated with vascular recanalization.

The prognosis of patients depends mainly on stenosis of the lumen and thrombosis of the dissected artery. Notably, in our six cases, we found that the degree of opening and compensation of the collateral circulation, such as the anterior and posterior communicating arteries, is very important for the blood supply to the brain on the injured side. Therefore, we hypothesize in some cases that the clinical presentation and prognosis of TICAD may be determined by the opening of the collateral circulation.

We are aware of some limitations in our study (*e.g.*, the small sample size and lack of follow-up imaging), but the various imaging techniques used revealed this traumatic arterial injury. A larger prospective randomized multicenter clinical study is

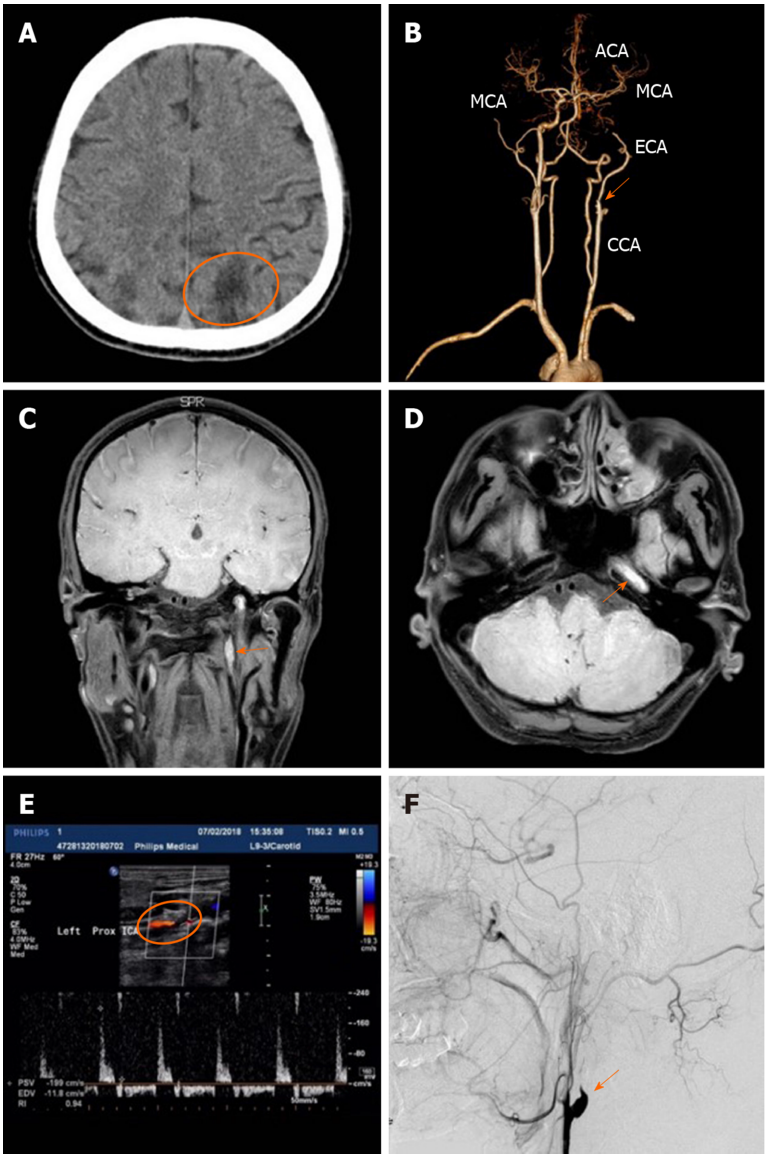


**Figure 2 Imaging of case 1.** A: Computed tomography revealed left parietal infarction (ellipse); B: Computed tomography angiography examination revealed tapering occlusion of the Internal carotid artery (ICA) suggestive of ICA dissection (arrow); C and D: Magnetic resonance imaging (T1W-VISTA sequence) demonstrated an intimal intramural hematoma extending to the intracranial section (arrows); E: Duplex ultrasonography examination revealed an intimal flap and significant narrowing of the lumen secondary to the equal-echoic mural hematoma (ellipse); F: Carotid artery digital subtraction angiography showed tapering occlusion of the left ICA just distal to the carotid bifurcation (Rat tail sign) (arrow). ACA: Anterior cerebral artery; CCA: Common carotid artery; ECA: External carotid artery; MCA: Middle cerebral artery.

required to increase the statistical power.

## CONCLUSION

TICAD is referred to as arterial dissection caused by an ICA intimal tear after trauma, and thromboembolism and hypoperfusion constitute the main pathology. TICAD can result in catastrophic outcomes. When the occurrence of dissection is suspected, detailed examinations should be carried out. Early and efficient diagnosis of TICAD is essential for initiating appropriate treatment. The treatment of TICAD is challenging and is always based on clinician discretion on a case-by-case basis.

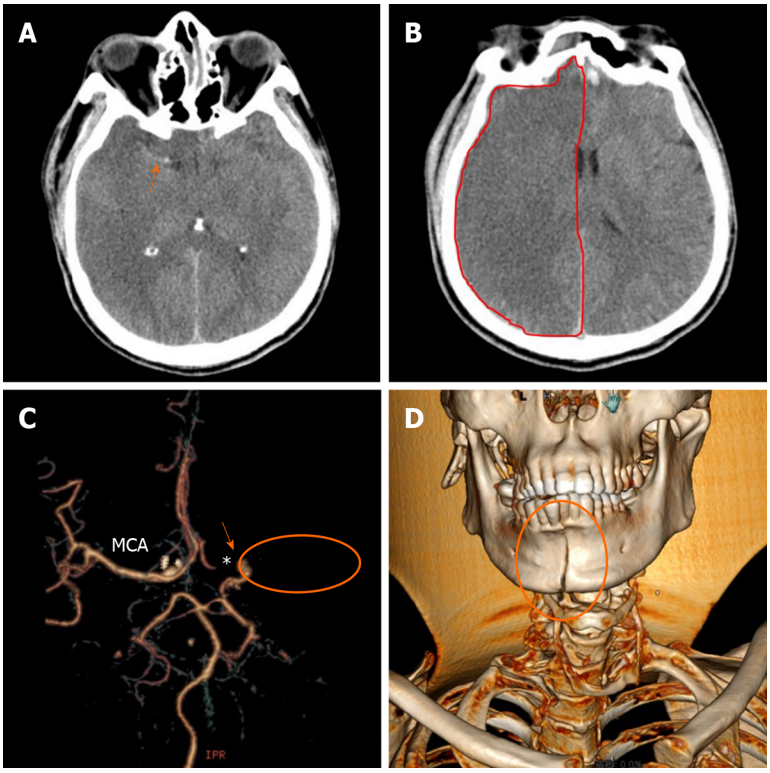


**Figure 3 Imaging of case 2.** A: Computed tomography (CT) showed a hyperdense right middle cerebral artery sign (arrow); B: CT revealed a large hypodense lesion with a mass effect in the frontotemporoparietal region (in the red line region) and a brain contusion in the frontal lobe (arrow); C: CT angiography examination revealed the anterior cerebral artery origin (asterisk) and middle cerebral artery (ellipse) occlusions suggestive of internal carotid artery dissection (arrow); D: A 3D-CT scan showed a mandibular fracture (ellipse). MCA: Middle cerebral artery.

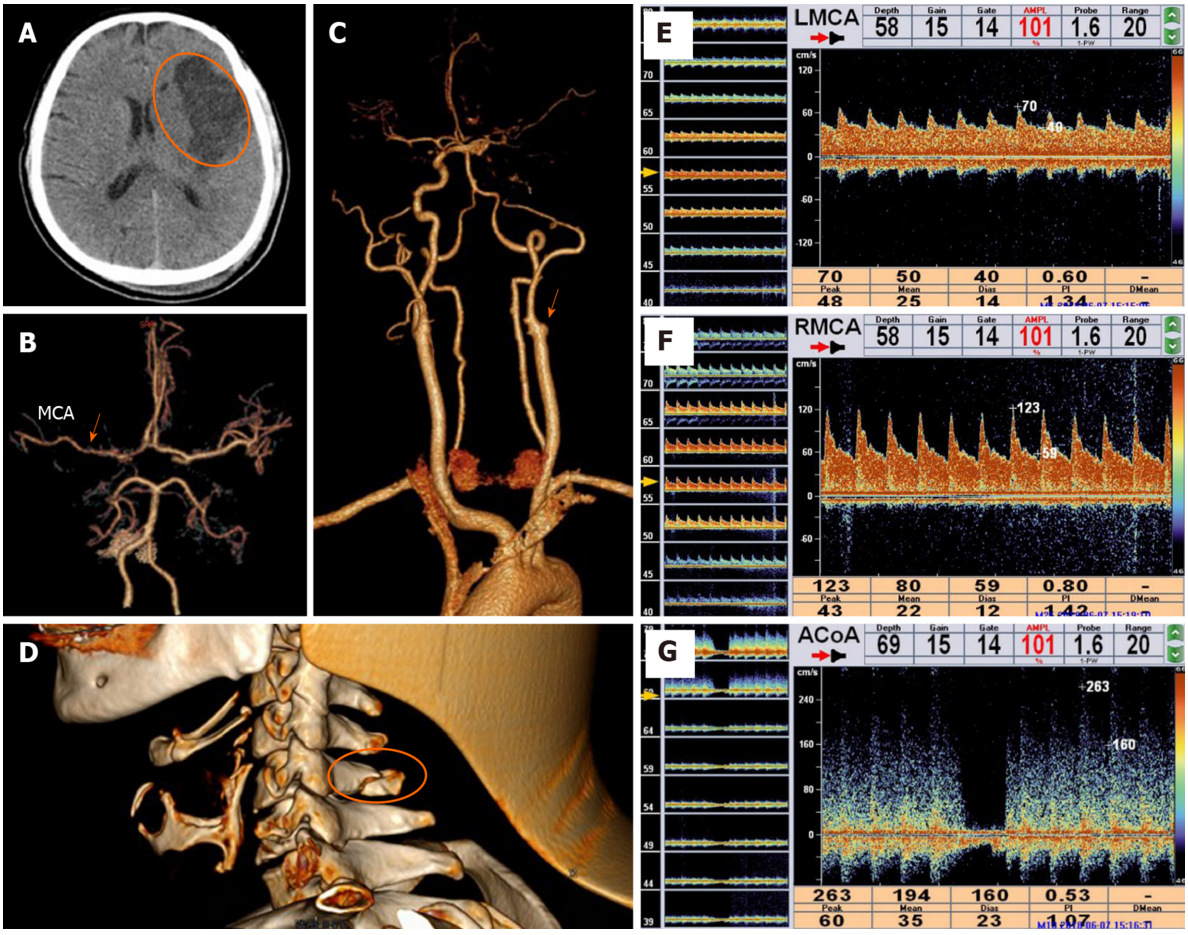


**Figure 4 Imaging of case 3.** A: Computed tomography (CT) revealed left frontotemporal infarction (ellipse); B: Head CT angiography revealed a thin middle cerebral artery (arrow); C: Cervical CT angiography revealed left internal carotid artery occlusion (arrow); D: A 3D-CT scan showed a cervical spinous process fracture (ellipse); E and F: Transcranial doppler showed asymmetry between the blood flow velocities of the two middle cerebral artery caused by left traumatic internal carotid artery dissection; G: When the right internal carotid artery was subjected to a neck compression test, the velocity of the anterior communicating artery rapidly decreased to zero and recovered when the compression was released. In E-G: FVd is the diastolic blood flow velocity, FVm is the mean blood flow velocity, FVs is the systolic blood flow velocity, and PI is the pulsatility index. MCA: Middle cerebral artery.

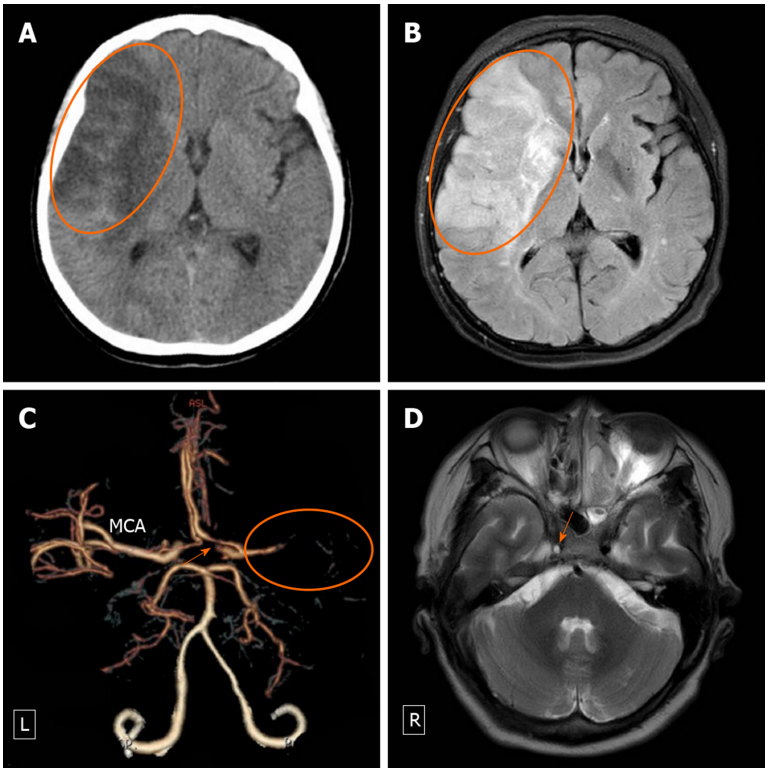


**Figure 5 Imaging of case 5.** A and B: Computed tomography (A) and (B) magnetic resonance imaging scans showed cerebral infarction in the territory of the right middle cerebral artery (ellipses); C: Computed tomography angiography revealed tapering occlusion of the right middle cerebral artery (ellipse), and the internal carotid artery in the cavernous sinus segment became thin and irregular (arrow); D: Magnetic resonance imaging showed right internal carotid artery dissection depicted as a partial flow-void lumen surrounded by high-intensity signals of an intramural hematoma (arrow). MCA: Middle cerebral artery.

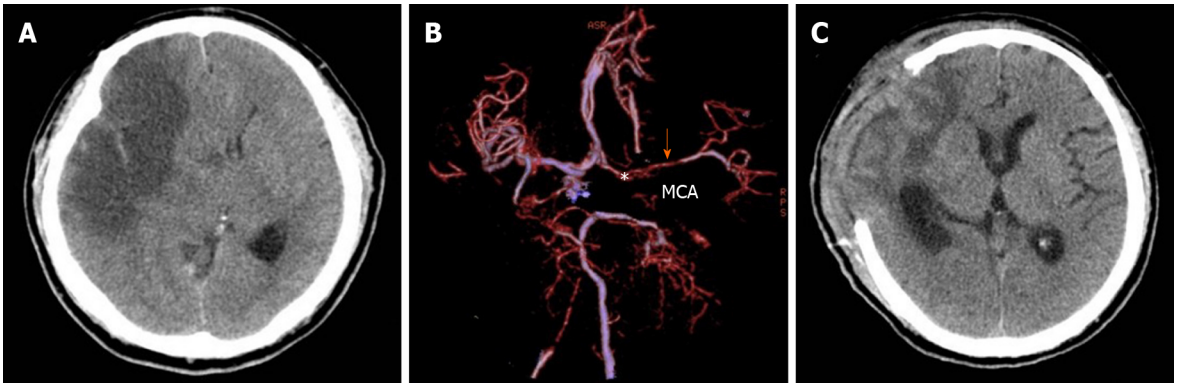


**Figure 6 Imaging of case 6.** A: Computed tomography revealed right frontotemporal infarction (ellipse) with a mass effect on the lateral ventricle and a midline shift; B: Computed tomography angiography revealed right internal carotid artery occlusion (asterisk) and a thin middle cerebral artery (arrow); C: Computed tomography revealed the decompression craniectomy with expansion of the infarcted brain parenchyma. MCA: Middle cerebral artery.

## ARTICLE HIGHLIGHTS

### *Research background*

The extracranial internal carotid artery refers to the anatomic location that reaches from the common carotid artery proximally to the skull base distally and is at considerable risk for injury. No consensus regarding its diagnostic screening and management has been established. The present study compared the outcomes of six different patients who suffered traumatic internal carotid artery dissection (TICAD).

### *Research motivation*

Despite a high incidence, reports of TICAD are limited to case reports or small case series. Currently, the frequency, cause, imaging changes, and influence on mortality of TICAD are not well defined. We therefore decided to conduct a retrospective study of

TICAD at a tertiary medical center. The risk factors of infarction, pathophysiology, clinical and radiological features, diagnosis, treatment, and prognosis were analyzed and delineated for TICAD.

### Research objectives

We performed a retrospective analysis and literature review of patients who were diagnosed as TICAD.

### Research methods

In this retrospective case series, emergency admissions for TICAD due to closed head injury were analyzed. The demographic, clinical, and radiographic data were retrieved from patient charts and the picture archiving and communication system, and a literature review of TICAD was also performed.

### Research results

Six patients presented with TICAD. Traffic accidents (4/6) were the most frequent cause of TICAD. The clinical presentation was always related to brain hypoperfusion. Imaging examination revealed dissection of the affected artery and corresponding brain infarction. All the patients were definitively diagnosed with TICAD. One patient was treated conservatively, one patient underwent anticoagulant therapy, two patients were given both antiplatelet and anticoagulant drugs, and two patients underwent decompressive craniectomy. One patient fully recovered, while three patients were disabled at follow-up. Two patients died of refractory brain infarction.

### Research conclusions

We found that TICAD should be identified in patients presenting after blunt trauma, including classical dissection, pseudoaneurysm, and stenosis/occlusion.

### Research perspectives

Early diagnosis and intervention can improve the prognosis and quality of life of patients who suffered TICAD. Based on the results of this study, future research should include prospective randomized control trial with a larger patient population so that we can better understand the diagnosis and treatment of TICAD.

## REFERENCES


1 **Bouzat P**, Francony G, Brun J, Lavagne P, Picard J, Broux C, Declety P, Jacquot C, Albaladejo P, Payen JF. Detecting traumatic internal carotid artery dissection using transcranial Doppler in head-injured patients. *Intensive Care Med* 2010; **36**: 1514-1520 [PMID: 20490780 DOI: 10.1007/s00134-010-1919-0]

2 **Tveita IA**, Madsen MRS, Nielsen EW. Dissection of the internal carotid artery and stroke after mandibular fractures: a case report and review of the literature. *J Med Case Rep* 2017; **11**: 148 [PMID: 28576125 DOI: 10.1186/s13256-017-1316-1]

3 **Kowalski RG**, Haarbauer-Krupa JK, Bell JM, Corrigan JD, Hammond FM, Torbey MT, Hofmann MC, Dams-O'Connor K, Miller AC, Whiteneck GG. Acute Ischemic Stroke After Moderate to Severe Traumatic Brain Injury: Incidence and Impact on Outcome. *Stroke* 2017; **48**: 1802-1809 [PMID: 28611087 DOI: 10.1161/STROKEAHA.117.017327]

4 **Zhou W**, Huynh TT, Kougias P, El Sayed HF, Lin PH. Traumatic carotid artery dissection caused by bungee jumping. *J Vasc Surg* 2007; **46**: 1044-1046 [PMID: 17980290 DOI: 10.1016/j.jvs.2007.06.026]

5 **Benedict WJ**, Prabhu V, Viola M, Biller J. Carotid artery pseudoaneurysm resulting from an injury to the neck by a fouled baseball. *J Neurol Sci* 2007; **256**: 94-99 [PMID: 17368675 DOI: 10.1016/j.jns.2007.02.016]

6 **Bajkó Z**, Maier S, Moţăţăianu A, Bălaşa R, Vasiu S, Stoian A, Andone S. Stroke Secondary to Traumatic Carotid Artery Injury - A Case Report. *J Crit Care Med (Targu Mures)* 2018; **4**: 23-28 [PMID: 29967897 DOI: 10.1515/jccm-2018-0003]

7 **Miller PR**, Fabian TC, Croce MA, Cagiannos C, Williams JS, Vang M, Qaisi WG, Felker RE, Timmons SD. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. *Ann Surg* 2002; **236**: 386-93; discussion 393 [PMID: 12192325 DOI: 10.1097/00000658-200209000-00015]

8 **Krajewski LP**, Hertzer NR. Blunt carotid artery trauma: report of two cases and review of the literature. *Ann Surg* 1980; **191**: 341-346 [PMID: 7362300 DOI: 10.1097/00000658-198003000-00014]

9 **Löhrer L**, Vieth V, Nassenstein I, Hartensuer R, Niederstadt T, Raschke MJ, Vordemvenne T. Blunt cerebrovascular injuries in acute trauma care: a screening protocol. *Eur Spine J* 2012; **21**: 837-843 [PMID: 21898164 DOI: 10.1007/s00586-011-2009-x]

10 **Furtner M**, Werner P, Felber S, Schmidauer C. Bilateral carotid artery dissection caused by springboard diving. *Clin J Sport Med* 2006; **16**: 76-78 [PMID: 16377982 DOI: 10.1097/01.jsm.0000173267.84112.a0]

11 **Teasdale G**, Maas A, Lecky F, Manley G, Stocchetti N, Murray G. The Glasgow Coma Scale at 40 years: standing the test of time. *Lancet Neurol* 2014; **13**: 844-854 [PMID: 25030516 DOI: 10.1016/S1474-4422(14)70120-6]

12 **McMillan T**, Wilson L, Ponsford J, Levin H, Teasdale G, Bond M. The Glasgow Outcome Scale - 40 years

of application and refinement. *Nat Rev Neurol* 2016; **12**: 477-485 [PMID: 27418377 DOI: 10.1038/nrneurol.2016.89]

13 **Foreman PM**, Harrigan MR. Blunt Traumatic Extracranial Cerebrovascular Injury and Ischemic Stroke. *Cerebrovasc Dis Extra* 2017; **7**: 72-83 [PMID: 28399527 DOI: 10.1159/000455391]

14 **Ben Hassen W**, Machet A, Edjlali-Goujon M, Legrand L, Ladoux A, Mellerio C, Bodiguel E, Gobin-Metteil MP, Trystram D, Rodriguez-Regent C, Mas JL, Plat M, Oppenheim C, Meder JF, Naggara O. Imaging of cervical artery dissection. *Diagn Interv Imaging* 2014; **95**: 1151-1161 [PMID: 25632417 DOI: 10.1016/j.diii.2014.10.003]

15 **Shindo T**, Ito M, Matsumoto J, Miki K, Fujihara F, Terasaka S, Isu T, Isobe M. A Case of Juvenile Stroke due to Carotid Artery Dissection from an Elongated Styloid Process-Revisiting Conservative Management. *J Stroke Cerebrovasc Dis* 2019; **28**: 104307 [PMID: 31383620 DOI: 10.1016/j.jstrokecerebrovasdis.2019.104307]

16 **Sporns PB**, Niederstadt T, Heindel W, Raschke MJ, Hartensuer R, Dittrich R, Hanning U. Imaging of Spontaneous and Traumatic Cervical Artery Dissection : Comparison of Typical CT Angiographic Features. *Clin Neuroradiol* 2019; **29**: 269-275 [PMID: 29374294 DOI: 10.1007/s00062-018-0666-4]

17 **Dandamudi VS**, Thaler DE, Malek AM. Cerebral embolus following chiropractic manipulation in a patient with a calcified carotid artery. *J Neuroimaging* 2013; **23**: 429-430 [PMID: 22818061 DOI: 10.1111/j.1552-6569.2012.00706.x]

18 **Vadikolias K**, Heliopoulos J, Serdari A, Vadikolia CM, Piperidou C. Flapping of the dissected intima in a case of traumatic carotid artery dissection in a jackhammer worker. *J Clin Ultrasound* 2009; **37**: 221-222 [PMID: 19208421 DOI: 10.1002/jcu.20556]

19 **Engelter ST**, Lyrer PA, Kirsch EC, Steck AJ. Long-term follow-up after extracranial internal carotid artery dissection. *Eur Neurol* 2000; **44**: 199-204 [PMID: 11096217 DOI: 10.1159/000008236]

20 **Giossi A**, Ritelli M, Costa P, Morotti A, Poli L, Del Zotto E, Volonghi I, Chiarelli N, Gamba M, Bovi P, Tomelleri G, Carletti M, Checcarelli N, Meneghetti G, Morra M, Chinaglia M, De Giuli V, Colombi M, Padovani A, Pezzini A. Connective tissue anomalies in patients with spontaneous cervical artery dissection. *Neurology* 2014; **83**: 2032-2037 [PMID: 25355826 DOI: 10.1212/WNL.0000000000001030]

21 **Ramchand P**, Mullen MT, Bress A, Hurst R, Kasner SE, Cucchiara BL, Messé SR. Recanalization after Extracranial Dissection: Effect of Antiplatelet Compared with Anticoagulant Therapy. *J Stroke Cerebrovasc Dis* 2018; **27**: 438-444 [PMID: 29100856 DOI: 10.1016/j.jstrokecerebrovasdis.2017.09.065]

22 **Delgado F**, Bravo-Rodríguez FA, Bautista MD, Chirosa MA, Molina T, Martos JM, Canis M. Carotid pseudoaneurysms secondary to dissection: endovascular management with bare stent-graft. *Cerebrovasc Dis* 2005; **19**: 136-138 [PMID: 15637436 DOI: 10.1159/000083243]

23 **Griessenauer CJ**, Foreman PM, Deveikis JP, Harrigan MR. Optical coherence tomography of traumatic aneurysms of the internal carotid artery: report of 2 cases. *J Neurosurg* 2016; **124**: 305-309 [PMID: 26252460 DOI: 10.3171/2015.1.JNS142840]

24 **Robertson JJ**, Koyfman A. Extracranial Cervical Artery Dissections. *Emerg Med Clin North Am* 2017; **35**: 727-741 [PMID: 28987426 DOI: 10.1016/j.emc.2017.06.006]

25 **Cohen JE**, Ben-Hur T, Rajz G, Umansky F, Gomori JM. Endovascular stent-assisted angioplasty in the management of traumatic internal carotid artery dissections. *Stroke* 2005; **36**: e45-e47 [PMID: 15746458 DOI: 10.1161/01.STR.0000158910.08024.7f]

26 **Cohen JE**, Gomori JM, Itshayek E, Spektor S, Shoshan Y, Rosenthal G, Moscovici S. Single-center experience on endovascular reconstruction of traumatic internal carotid artery dissections. *J Trauma Acute Care Surg* 2012; **72**: 216-221 [PMID: 22310130 DOI: 10.1097/TA.0b013e31823f630a]

27 **Davis JW**, Holbrook TL, Hoyt DB, Mackersie RC, Field TO Jr, Shackford SR. Blunt carotid artery dissection: incidence, associated injuries, screening, and treatment. *J Trauma* 1990; **30**: 1514-1517 [PMID: 2258964 DOI: 10.1097/00005373-199012000-00013]

28 **Misra S**, Haas CA, August M, Eberlin KR. Delayed Carotid Dissection Following Lower Lip Revascularization in the Setting of Hyoid Fracture--A Case Report and Review of the Literature. *J Oral Maxillofac Surg* 2016; **74**: 123-128 [PMID: 26435401 DOI: 10.1016/j.joms.2015.09.013]

29 **Jacobson LE**, Ziemba-Davis M, Herrera AJ. The limitations of using risk factors to screen for blunt cerebrovascular injuries: the harder you look, the more you find. *World J Emerg Surg* 2015; **10**: 46 [PMID: 26413148 DOI: 10.1186/s13017-015-0040-7]

30 **Berne JD**, Cook A, Rowe SA, Norwood SH. A multivariate logistic regression analysis of risk factors for blunt cerebrovascular injury. *J Vasc Surg* 2010; **51**: 57-64 [PMID: 19954917 DOI: 10.1016/j.jvs.2009.08.071]

31 **Esnault P**, Cardinale M, Boret H, D'Aranda E, Montcriol A, Bordes J, Prunet B, Joubert C, Dagain A, Goutorbe P, Kaiser E, Meaudre E. Blunt cerebrovascular injuries in severe traumatic brain injury: incidence, risk factors, and evolution. *J Neurosurg* 2017; **127**: 16-22 [PMID: 27471889 DOI: 10.3171/2016.4.JNS152600]

32 **Delgado Almandoz JE**, Schaefer PW, Kelly HR, Lev MH, Gonzalez RG, Romero JM. Multidetector CT angiography in the evaluation of acute blunt head and neck trauma: a proposed acute craniocervical trauma scoring system. *Radiology* 2010; **254**: 236-244 [PMID: 20019135 DOI: 10.1148/radiol.09090693]

33 **Debette S**, Leys D. Cervical-artery dissections: predisposing factors, diagnosis, and outcome. *Lancet Neurol* 2009; **8**: 668-678 [PMID: 19539238 DOI: 10.1016/S1474-4422(09)70084-5]

34 **Vertinsky AT**, Schwartz NE, Fischbein NJ, Rosenberg J, Albers GW, Zaharchuk G. Comparison of multidetector CT angiography and MR imaging of cervical artery dissection. *AJNR Am J Neuroradiol* 2008; **29**: 1753-1760 [PMID: 18635617 DOI: 10.3174/ajnr.A1189]

35 **Crönlein M**, Sandmann GH, Beirer M, Wunderlich S, Biberthaler P, Huber-Wagner S. Traumatic bilateral carotid artery dissection following severe blunt trauma: a case report on the difficulties in diagnosis and therapy of an often overlooked life-threatening injury. *Eur J Med Res* 2015; **20**: 62 [PMID: 26199074 DOI: 10.1186/s40001-015-0153-1]

36 **Bajkó Z**, Bălaşa R, Moţăţăianu A, Bărcuţean L, Stoian A, Stirbu N, Maier S. Malignant Middle Cerebral

Artery Infarction Secondary to Traumatic Bilateral Internal Carotid Artery Dissection. A Case Report. *J Crit Care Med (Targu Mures)* 2016; **2**: 135-141 [PMID: 29967852 DOI: 10.1515/jccm-2016-0021]

37 **Kurre W**, Bansemir K, Aguilar Pérez M, Martinez Moreno R, Schmid E, Bäzner H, Henkes H. Endovascular treatment of acute internal carotid artery dissections: technical considerations, clinical and angiographic outcome. *Neuroradiology* 2016; **58**: 1167-1179 [PMID: 27796447 DOI: 10.1007/s00234-016-1757-z]

38 **Daou B**, Hammer C, Mouchtouris N, Starke RM, Koduri S, Yang S, Jabbour P, Rosenwasser R, Tjoumakaris S. Anticoagulation vs Antiplatelet Treatment in Patients with Carotid and Vertebral Artery Dissection: A Study of 370 Patients and Literature Review. *Neurosurgery* 2017; **80**: 368-379 [PMID: 28362967 DOI: 10.1093/neuros/nyw086]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
**Telephone:** +1-925-3991568
**E-mail:** bpgoffice@wjgnet.com
**Help Desk:** https://www.f6publishing.com/helpdesk
https://www.wjgnet.com