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Review

# Traumatic Carotid Artery Dissection: A Different Entity without Specific Guidelines

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According to literature data, there are no distinct guidelines regarding the proper diagnostic and therapeutic management of traumatic carotid artery dissection (TCAD). Although most of cases evaluated in research studies refer to spontaneous carotid artery dissection, traumatic cases demand special considerations as far as diagnosis and treatment are concerned. Although both types of dissection share some common characteristics, a patient with TCAD usually presents with several concomitant injuries as well as a higher bleeding risk, thus complicating decision making in such patients. Therefore, aim of this review is to present available data regarding epidemiology, clinical presentation, diagnostics and treatment strategy in cases with TCAD in order to produce useful conclusions for everyday clinical practice.

Key Words: Carotid arteries, Nonpenetrating wounds, Anticoagulation, Endovascular

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## INTRODUCTION

Traumatic carotid artery dissection (TCAD) is a different clinical entity compared to spontaneous carotid dissection demanding special considerations regarding proper management.

Recent guidelines on carotid disease management do not make any differentiation among spontaneous and traumatic dissection [1]. According to these guidelines, antithrombotic or antiplatelet treatment is recommended in patients with neurological symptoms (Class IIa, Level B recommendation) and endovascular intervention is suggested only when neurological status of a patient deteriorates under conservative medical treatment (Class IIb, Level C recommendation) [1]. However, considering traumatic cases, special issues should be taken under consideration.

Therefore, this review aims to highlight the specific characteristics of epidemiology, clinical presentation, diagnostic investigation and proper treatment in patients with traumatic dissection of the carotid artery.

## EPIDEMIOLOGY

In general, blunt carotid injury is observed in 1%-2.6% of blunt trauma cases and in 2.7% of patients with severe multisystem trauma [2]. Moreover, blunt carotid injury has been associated with a high stroke rate (up to 60%) and mortality rate (19%-43%) [3]. Many of these cases are asymptomatic and they remain undetected until symptoms of cerebrovascular ischemia present. Recent data indicate that symptoms occur after a mean of 12.5 hours in survivors, and after a mean of 19.5 hours in non-survivors [3]. However, TCAD is very rare (estimated incidence 0.08%), and although it is associated with mild symptoms, it can sometimes be fatal [4]. Thus, this type of dissections is often overlooked life-threatening injuries. Even though most of the carotid artery dissections occur spontaneously, about 4% of the dissections are related to severe trauma.

Furthermore, spontaneous dissections are usually seen in older patients (over 50 years of age) although traumatic dissections mostly affect young patients around 40 years of age [5].

Regarding the mechanism of injury, TCAD may result from a direct blow to anterolateral aspect of the neck, or an extreme extension and rotation of the neck. Trauma mechanisms involved are variable, ranging from high speed motor vehicle accidents to trivial traumas in certain groups of patients (for example patients with hypertension or connective tissue diseases). Distraction/extension, distraction/flexion or lateral flexion forces of the cervical spine may result in traumatic TCADs as well [5]. Even a vasocompression between C-spine and mandibula during a hyperinclination trauma can lead to a dissection of the internal carotid artery. The forces implicated in such injuries may cause small lesions of the vessel wall, which could result in intimal tears, intramural hematomas or complete lumen displacement/obstruction [6].

Although there are certain vascular risk factors associated to spontaneous dissection such as coronary heart disease (33%), hypertension (57%), and hypercholesterolemia (29%), history of smoking (45%) and history of migraine (21%), in younger patients suffered from TCAD the aforementioned factors are usually not present [7]. Moreover, in cases of spontaneous dissections, no history of any kind of cervical trauma or stressful movement is reported. Intrinsic susceptibiliity has been observed in certain patients with monogenic connective tissue disease (Ehlers Danlos syndrome, Marfan syndrome, polycystic kidney disease, deficiency of alpha-1 antitrypsin and hereditary hemochromatosis). Almost 2% of dissections have been correlated with such conditions [7].

## CLINICAL PRESENTATION

Only 10% of cases present immediate symptoms although most clinical signs usually occur within the first 24 hours of the occurrence of the trauma. TCAD is suspected and diagnosed when neurological symptoms occur unexpectedly after a trauma of the neck or the head. The most frequent presentations of TCAD are stroke, Hörner syndrome due to pressure of a hematoma, and paralysis of a cranial nerve. TCAD evolves into stroke in 80% of cases within the first week of the trauma. The common cause of stroke is arterial thrombosis resulting in permanent neurological deficits, with a mortality rate approaching 40% [8]. Due to the traumatic mechanism, bleeding through the oral cavity, nostrils or ears could be detected as well. Time of ischemic signs onset is very variable too, diverging from immediate to several months delay [9].

Unlike spontaneous cases, these patients present with concomitant injuries of the neck or the skull, and frequently, their cognitive status is significantly altered. Almost one third of such patients could present with a cerebrovascular infarct that could not be justified otherwise. Therefore, several risk indices (Denver group criteria, Memphis or Kerwin criteria) have been developed in order to early screen such patients and proceed with proper treatment promptly [10]. Such indices share most of their included risk factors such as neurologic status incosistent with radiologic findings, severe soft tissue injury/hematoma of the neck, high grade facial fractures and high risk mechanism of injury (Fig. 1). Biffl et al. [11] have added and some other factors such as Glascow coma scale <6 and diffuse axonal injury. Hence, the number of risk factors present is strongly associated with increasing stroke and mortality risk as well. Such criteria are essential for early screening in order to identify patients in higher risk for blunt carotid injuries yielding a high sensitivity and specificity. Additionally, the cost of long-term rehabilitation care and human life after dissection-associated neurologic events is substantial [12]. Therefore, prompt identification of such patients is imperative in order to proceed with further imaging investigation and proper treatment [13].

## DIAGNOSTIC INVESTIGATION

Duplex ultrasonography has the advantage of being a non-invasive method although its performance is strongly affected by the experience of the operator. Furthermore, this modality shows a high disposability even in smaller

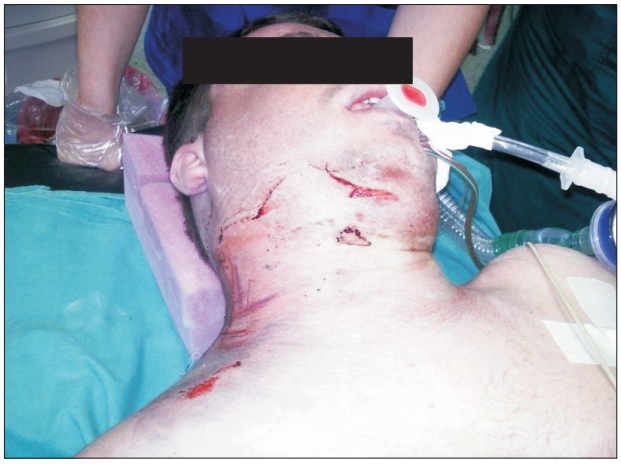

**Fig. 1.** Trauma patient with soft tissue injury of the neck and altered mental status due to traumatic carotid artery dissection. The patient was intubated due to low Glascow scale.

rural hospitals, making it the first choice for most practitioners as far as early screening is concerned. It is a common method to detect a possible vascular injury, although it offers a poor vision of the intracranial aspects of a TCAD and it also gives limited information about small intimal tears [6]. Finally, when extensive soft tissue injuries present, the sensitivity of diagnostic ultrasound is even lower in case of traumatic dissections compared to spontaneous cases.

Although, the golden standard for identifying a possible dissection is digital subtraction angiography (DSA) according to many authors [3], computed tomography angiography (CTA) would probably be more appropriate as initial screening modality in cases of severe trauma. DSA offers a very high diagnostic performance of 97% for vascular injuries and at the same time, it permits a potential intervention via endovascular techniques when indicated (Fig. 2) [11]. However, DSA is an invasive method with a complication rate of almost 1%, and it is not readily available in all institutions, especially in those without an endovascular suite. However, computed tomography (CT) scan combined with CTA would be more appropriate in identifying carotid dissections in patients with severe trauma as it would facilitate the detection of concomitant cerebral or vertebral injuries [14]. "String signs", indicating constrictions of the lumen, are reckoned to be indirect signs of artery dissection. Moreover, recent data indicate that traumatic dissections present higher (Segment III; level of first-second cervical vertebrae) compared to spontaneous dissections (Segment I; origin of vessel up to fifth cervical vertebrae) [15]. According to Harrigan et al. [16], CTA was selected in almost 60% of cases in a large series of more than 11,000 patients with blunt cervical trauma. Hence, a recent evaluation of CTA with 16-section CT scanners has demonstrated 97.7% sensitivity and 100% specificity compared with the criterion standard of conventional DSA [17]. Therefore, many authors suggest CTA in such patients followed by DSA for inconclusive cases or when endovascular repair is indicated.

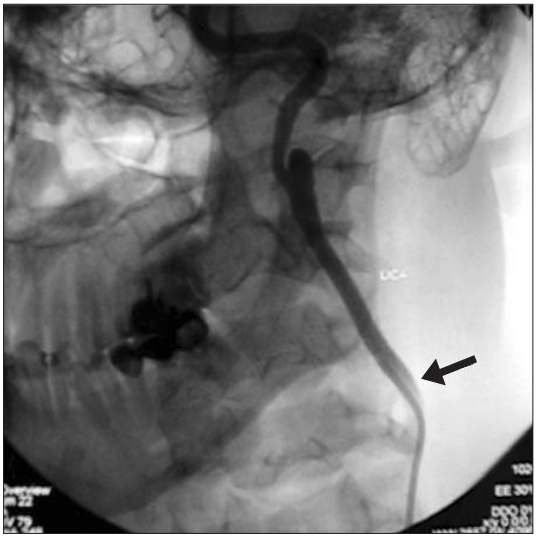

**Fig. 2.** Digital subtraction angiography illustrating a traumatic dissection of the common carotid artery (arrow) and an occlusion of the internal carotid artery.

Finally, magnetic resonance (MR) scanning combined with MR angiography (MRA) could be an alternative for early TCAD detection. Especially for blunt trauma, MRA determines dissections in up to 99% and provides additional information about concomitant injuries such as brain injuries or skull fractures [3,18]. Recent data show that MRA is equal to CTA in the diagnosis of carotid and vertebral artery dissection in general [18]. However, there are specific concerns for TCAD cases. First of all, MRA is not available in all institutions as an emergency modality, especially in smaller hospitals. Additionally, besides the long duration of the scan, the existance of metal objects or life supporting devices (fragments, implants, pacemakers etc.) within the trauma or the upper body of the patient would raise contraindications for undergoing such investigation.

## TREATMENT

In general, asymptomatic patients with low-grade dissections are typically treated conservatively with medical management and close imaging observation [19]. The majority of these cases achieve anatomic and symptomatic resolution, with low rates of recurrence over long-term follow-up [20]. Regarding proper medical treatment for spontaneous carotid artery dissection, anticoagulation or antiplatelets should be initiated promptly to prevent further thromboembolic events [1,5,6]. Data so far indicate that both type of agents show similar efficacy in reducing neurological sequalae without increasing the risk for stroke [19,21]. However, cases of traumatic causes demand a more interdisciplinary approach and show specific considerations concerning treatment.

When other concomitant injuries with a high risk for severe bleeding are present (for example major fractures of long bones or pelvis, solid organ injuries within the abdomen or the thorax, closed head injuries etc.), then the optimal type and time of treatment should be considered in an interdisciplinary approach (including consultation by a trauma surgeon, orthopedic surgeon, neurosurgeon, vascular surgeon, radiologist, neurologist). For most of cases, medical treatment should be initiated as soon as possible. When anticoagulants are initiated, the therapeutic heparinization should be regulated according to partial thromboplastin time levels (50-70 seconds), followed by per



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os treatment (international normalized ratio 2-3) for at least 3 months. However, practitioners should always take into consideration the patient's risks, comorbidities and overall injuries when deciding on the proper agent. According to some authors, anticoagulation is preferred to antiplatelet agents in cases of severe stenosis, arterial occlusion or pseudoaneurysm although antiplatelets are preferred when high risk for bleeding, inadequate collateral circulation or large infarcts are present [6]. Finally, concerning the role of thrombolysis in TCAD, data are limited in literature given the high bleeding risk in trauma patients. Even for spontaneous dissection, the results seem to be contradictory [22].

Regarding the role of endovascular treatment for TCAD, this minimally invasive method is becoming more popular lately, and it is described as a good therapeutic alternative even for traumatic cases [23], although there have been reports of high complication and occlusion rates with high grade injuries in the past [24]. In a recent meta-analysis on endovascular management of carotid dissections, the method has shown optimal technical success, low recurrent rates and satisfying one year follow-up. Hence, one third of the included patients were traumatic cases [25]. In this review, the authors suggest the following criteria for stenting: (i) patients with recurrent symptoms despite medical therapy, (ii) patients with hemodynamic hypoperfusion (involvement of multiple vessels or poor collateral vessels), (iii) patients with expanding or symptomatic pseudoaneurysm and (iv) contraindication to anticoagulation because of intracranial or systemic hemorrhage [25]. Furthermore, in a more recent study by Spanos et al. [26], overall 193 patients were treated endovascularly for TCAD yielding low periprocedural morbidity and mortality rates, namely 6% and 1.2% respectively, as well as low re-intervention rate during long-term follow-up.

Finally, endovascular repair with stenting also requires pre- and post-treatment antiplatelet therapy to avoid embolic complications or stent occlusion. In the aforementioned study, antiplatelet therapy (either single or dual) was initiated postoperatively, lasting from three months to inevitably with good longterm outcomes [26]. However, this often results in a frustrating clinical conundrum because many patients are referred for endovascular repair specifically due to contraindications to antithrombotic therapy such as severe intracranial injury, multisystem trauma, or penetrating injuries. Seth et al. [2] suggest avoiding the placement of stents in patients who have not or are not able to receive appropriate pre- and post-procedural antiplatelet therapy, unless faced with an immediately life-threatening injury.

## CONCLUSION

Patients with TCAD remain a subgroup of cases that require special considerations and management in an interdisciplinary setting. Although presenting not frequently and with a subtle clinical picture at the beginning, this entity is associated with major morbidity and mortality. Thus, guidelines for proper detection and treatment need to be adjusted in order to achieve optimal results.

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