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REVIEW ARTICLE

Review article: Non-penetrating neck artery dissection in young adults: Not to be missed!

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Abstract

Young adults who present to the ED with neck pain following non-penetrating, seemingly trivial trauma to the neck, are at risk of neck artery dissection and subsequent stroke. Sport-related neck injury is the chief cause. Physical examination may often be unremarkable, and although there may be reluctance to expose young patients to radiation, radiological imaging is central to making a diagnosis of arterial wall disruption. A comprehensive literature search was performed in relation to neck artery dissection, and the evidence was scrutinised. We discuss the typical mechanism of injury, symptoms, anatomical considerations and clinical aids in diagnosis of neck artery dissection. Although the incidence is low, neck artery dissection has a mortality of 7%. As such, it is important for front-line physicians to have a high suspicion of the diagnosis and a low threshold to organise radiological examinations, specifically computerised tomography. Early detection of neck artery dissection will trigger clinical protocols that call for multidisciplinary team management of this condition. In general, guideline-based recommendation for the management of neck artery dissection involving an intimal flap is by anti-platelet therapy while treatment of neck artery dissection that results in a pseudoaneurysm or thrombosis is managed by surgical intervention or endovascular techniques. Close follow up combined with antithrombotic treatment is recommended in these individuals, the goal being prevention of stroke.

Key words: *carotid arteries, carotid artery injuries, internal carotid artery dissection, neck injury, stroke.*

Key findings

- Neck artery dissection accounts for 15% of young strokes.
- In the setting of seemingly trival trauma, the diagnosis of neck artery dissection is challenging and requires a low threshold for radiological imaging.
- Anti-platelet therapy and early surgical intervention can prevent stroke.

Introduction

Stroke in young adults that results from ischaemia secondary to neck artery dissection following non-penetrating injury is uncommon. Neck artery dissection particularly in young adults requires 'top-of-the-mind' suspicion because a missed diagnosis is a missed opportunity to prevent stroke. The annual reported incidence of symptomatic spontaneous internal carotid artery and vertebral artery dissection was 2.5–3 per 100 000.[1] A recent study indicated that about 3% of young persons with neck artery dissection was probably missed.[2] In a large Finnish study, neck artery dissection accounted for 15% of young strokes.[3]

Literature review and methods

Data acquisition for this review was through a search of MEDLINE/PubMed and Embase between 2000 and 2022 using the MeSH terms cervical artery dissection, blunt trauma, neck, sport and stroke. Initially, we reviewed all abstracts written in the English language and translations of abstracts that were originally written in languages other than in English. Articles of selected abstracts, including reviews that incorporated at least 10 years of previous articles up to the time of publication, were downloaded for further reading. Further, those articles of importance and drawn from references within the index publications, were revisited. In all, 52 papers were read and reviewed. This narrative review focuses upon mainly sport-induced neck artery dissection, discusses anatomy of the neck arteries, the pathophysiology of dissection and reviews imaging modalities. There are no specific sport-related neck artery dissection management guidelines published to date. Because the pathophysiology and management of sport-related neck artery dissection seem to align with established guidelines of the management of neck artery dissection in general, we describe principles of management using current evidence-based guidelines as applied to sport-related neck artery injury.

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Demography

In the largest study of the aetiology of sport-related neck artery dissection, involving 190 published cases, approximately one fourth of injuries were in women, and at least 44 types of sport were associated with cervical artery dissection.[4] Schlemm *et al.* showed that the median age at presentation of neck artery dissection was 35 years with the youngest reported in a 4-year-old after playing on a trampoline.[4] Contrary to popular belief, the majority of dissections were reported in golfers (8%), followed by weight lifters and weight trainers (7%), basketball (6%), football (5%) and tennis (5%) players. Surprisingly, such contact sport as rugby football was reported to be associated with cervical artery dissection in only 2% in this series. This is because the mechanism of arterial injury is mainly related to shearing forces, not so much due to contact. Although spontaneous neck artery dissection causing stroke has been reported in patients with connective tissue disorders such as Ehlers-Danlos, Marfan syndrome and osteogenesis imperfecta, there was no known specific association between sport, cervical artery dissection in young individuals and an underlying connective tissue disorder. None of the young individuals with sport-related neck artery dissection in the study by Schlemm was found to have hypertension, dyslipidaemia or disorders of cardiac rhythm.[4]

Mechanism of injury

The postulated mechanism of injury is a shearing force upon the structures of the neck generated by neck rotation and hyper-extension, much like a whiplash.[5] Both carotid and vertebral arteries are susceptible to injury. In the case of the carotid artery, the internal carotid is almost always the site of damage. Giossi *et al.*[6] reported that those with cervical artery dissection tended to display tortuous carotid arteries compared with matched controls. Computational models of aortic dissection simulation showed that there is an association between intra-luminal arterial pressure, the degree of axial stretch of the vessel wall and initiation of an intimal tear.[7] This core theory of high intra-luminal pressure and axial stretch that is induced in sport injury of the neck may help explain intimal dissection in neck arteries that are tortuous, kinked and coiled.

Anatomical considerations

The internal carotid arteries are bilateral structures that arise from the bifurcation of the common carotid artery in the neck and traverse the neck within the carotid sheath to enter the base of skull through the carotid canal. In the neck, the artery courses an extra-cranial length of between 8.5 and 10 cm,[8] where it is susceptible to shearing forces. The vertebral arteries arise from the first part of the subclavian arteries bilaterally and travel along the side of the neck to enter foramina within the transverse processes of the cervical vertebrae commencing from the sixth cervical vertebra, which enter the skull to unite in a midline basilar artery that forms the circle of Willis.[9] The vertebral arteries, albeit protected within the osseous tunnel of the cervical vertebrae, are prone to twisting and shearing forces that cause intimal dissection along the course in the lateral part of the neck. Both, the internal carotid and vertebral arteries, were sites of dissection during rotation-hyperextension movement of the neck[4] – 49% of dissections were left sided, 40% right sided and 11% bilateral.[4] Furthermore, there seemed to be no difference in the frequency of injury for internal carotid compared with vertebral artery dissections.[4] The vast majority of internal carotid and vertebral artery dissections have been in the extra-cranial portion, and for the internal carotid artery, the point of dissection was typically reported at a point 2–3 cm above the bifurcation of the common carotid artery.[10]

Pathophysiology

Dissection of neck arteries following rotation-hyperextension of the neck commences with intimal damage as an initial step. In young persons, the commonest pattern of dissection is blood flow-induced elevation of a sub-intimal plane. Intimal injury is followed by platelet activation, aggregation and thrombosis. In case of expansion of the thrombus, which may now fill the lumen and lie within a false channel that is caused by intimal elevation, complete vessel occlusion or stenosis will result. In 10% of cases, a pseudo-aneurysm may form[11] which is defined as a locally contained haematoma with turbulent blood flow.[12] Also, neck artery thrombosis has the potential for distal embolism into the cerebral circulation, which risks permanent injury to those areas of the brain.

Classification of neck artery dissection

Based on the pathology of internal carotid artery dissection injury combined with accurate imaging, three classifications have been proposed; the Borgess classification,[13] another by Biffl *et al.*[14] and a third by Seth *et al.*[15] Although there is no direct comparison between these grades, the Biffl scale seems most evaluated and forms the benchmark for clinical guidelines in the Denver and expanded Denver criteria[16,17] in management of all blunt cervical arterial injury. The scale describes five grades of injury: grade I – irregularity of the intima or dissection with 25% luminal stenosis; grade II – arterial wall dissection with greater than 25% stenosis; grade III – pseudoaneurysm defined by enlargement $\times 1.5$ times the diameter of the normal vessel; grade IV – complete occlusion of the vessel lumen; grade V – transection of the internal carotid artery with extravasation of contrast. Grade V injuries of the internal carotid artery are rare, and because they occur in association with base of skull fractures that are an infrequent occurrence in sport injury, only grades I–IV become useful in management.

Symptoms/clinical features

The commonest presentation to an ED is with trivial symptoms of either headache, unilateral neck pain or dizziness.[18] For vertebral artery

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dissection, classical clinical features are occipito-cervical pain which may be associated with vertigo, dysarthria, visual deficit and diplopia – features of posterior circulation ischaemia. In the case of internal carotid artery dissection, presentation is with ipsilateral neck pain, headache, a partial Horner's syndrome – miosis and ptosis, without anhydrosis, because pseudomotor facial fibres traverse the wall of the external carotid artery and escape being injured.[1] In fact, the presence of any two of the above three clinical features is thought to be strongly suggestive of carotid artery dissection.[1] In a condition where less than one half of patients with neck artery dissection will have reported a typical history of neck injury,[19] heightened awareness of this injury coupled with an incisive history will alert the clinician to a rotational, hyper-extension movement of the neck, such as a golfer's drive. The spectrum is wide and may range from no symptoms to transient ischaemic attack with neurological deficit.[20] Almost one half of persons who sustain neck artery dissection will develop symptoms within minutes,[4] a quarter will become symptomatic from a few hours up to 1 day and the remainder later than 1 day after onset of injury. 75% of patients with neck artery dissection will develop symptoms within 24 h following the onset of injury.[4,21]

Imaging

Imaging is central to diagnosis because: (i) it confirms and pinpoints the site of injury; (ii) indicates the extent of injury; (iii) provides information about distal blood flow and the region of brain involved by ischaemia; and (iv) enables early detection because radiological signs emerge before the onset of clinical and neurological signs. Imaging by duplex ultrasound examination of the neck arteries for suspected patients coming through the ED would be a useful screening investigation that will determine the need for further imaging. Duplex ultrasound is widely available, non-invasive, and has the capacity to identify vessel thrombus and display real time flow characteristics.[22] Although duplex scanning is safe, the investigation is operator dependent and assessment of the neck by duplex ultrasound alone has been deemed insufficient[23] because of its inability to provide finer detail of an injured vessel wall and characteristics of brain blood flow compared with computed tomography angiography (CT-A) (Fig. 1) or magnetic resonance angiography (MR-A). Zhang *et al.* concluded that both CT-A and MR-A provided accurate display of vessel morphology and flow dynamics.[24] In young patients with a trivial history and non-specific examination, a decision to order radiation-associated imaging such as a CT-A is a challenging one. The Expanded Denver Criteria for CT-A (Table 1)[17] is a clinical aid tool used in the setting of traumatic neck injuries to determine the need for CT-A in the ED, and its use can be extended to guide decision-making for young patients with trivial presentations following sport injury. The current choice of CT-A over MR-A is based on universal availability of CT and the shorter examination times. Furthermore, MR-A is subject to the availability of an expert radiologist in the emergency setting and is more expensive compared with CT-A.[25] In a minority, CT-A alone may be either normal or display non-specific radiographic signs, which may be misleading and are likely to shroud

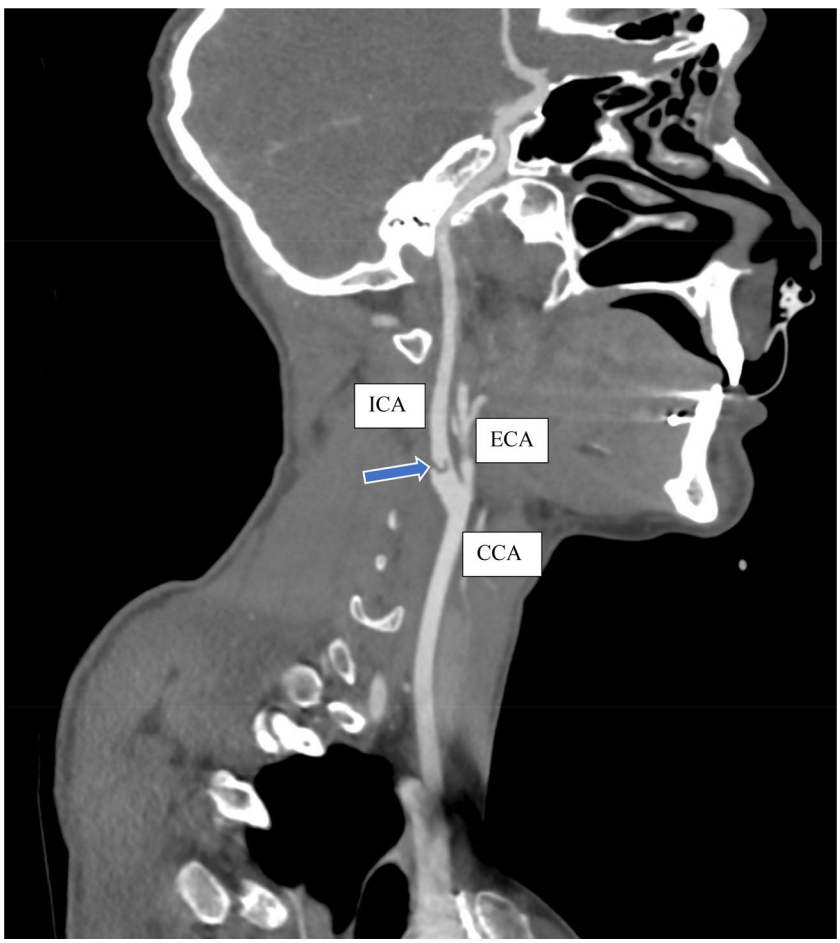

Figure 1. *Computed tomographic angiogram showing the dissected intimal flap (Biffl grade II) within the internal carotid artery (blue arrow), in a young woman athlete presenting with neck pain and transient dizziness following a weight training session. CCA, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery.*

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TABLE 1. *Expanded Denver criteria for computed tomography angiography† (adapted from Geddes* et al.,[17] *with permission, data included all patients with neck artery injury including motor vehicle accidents)*

Signs and symptoms
Arterial haemorrhage – neck, mouth or nose
Cervical bruit <50 years
Expanding cervical haematoma
Focal neurological deficit
Neuro examination incongruous with CT findings
Ischaemic stroke on CT/MR
Imaging
Base of skull involving carotid canal
Le Fort II or III fracture
Cervical spinal fracture – subluxation; extension to transverse foramen; C1–C3 fracture
Diffuse axonal injury; Glasgow Coma Scale <6
Mechanism
High-energy mechanism
Near-hanging with axonal injury

†Presence of one or more of the above is an indication for CTA.

other signs of arterial dissection.[19] In such case, high-resolution MR-A has the added sensitivity to delineate the structure of the vessel wall and the site of dissection.[24]

Management

Mortality associated with neck artery dissection has been reported to be 7%.[4] The goal of management in the ED should be early detection aided by a focused history in these young people who present with a trivial history. Physical examination is often unremarkable, and screening by duplex ultrasound examination of the neck is a way-pointer at this juncture[24] and should be used in all cases of suspicious sport-related neck injury. However, it is useful for the emergency physician to have a low threshold for further investigation by CT-A in the presence of one or more expanded Denver criteria.[17] The finding of a neck artery dissection should trigger immediate referral to the vascular surgery team to assist with ongoing management. The best available evidence suggests immediate use of antiplatelet therapy in these patients to prevent progression of pathology, acute ischaemic stroke and recurrent stroke.[26] Some may prefer anti-coagulant medication to prevent stroke but there is no evidence that prophylactic anti-coagulation is beneficial over anti-platelet therapy.[27]

There is an exponential rise in stroke risk with worsening Biffl grade (grade I – 3%; grade II – 11%; grade III – 33%; and grade IV – 44%). These risks need to be communicated to the patient in decision-making for treatment. Up to one half of internal carotid artery injuries will not show radiological improvement but remain stable.[28] For those with Biffl grade I and II internal carotid artery dissection who are treated with rest and medication, careful follow up is recommended because 5% have been reported to progress to a higher grade of injury.[28] Follow-up CT-A is generally recommended on day 7 and at 3 months after injury to establish that pathology had not progressed to pseudoaneurysm formation.[29] In 34% of patients, radiological surveillance will show an improvement with complete healing.[28] Return to sport must be supervised and graded mostly because these individuals will continue to remain on anti-platelet or anti-coagulant medication for up to 6 months as a measure of stroke prevention following injury.[30] Open (primary repair or bypass) and endovascular surgical intervention remain the treatment of choice for Biffl grade III and IV injuries. Surgical intervention is assuming a lesser role in modern day management of Biffl grade I and II neck artery dissection. To-date, there are no randomised controlled trials or case-controlled studies that have compared either surgical intervention of Biffl grade I and II neck artery dissection *versus* medical management and so, treatment of early-grade neck artery injury will likely be personalised and depend on multiple factors such as the patient's preference, available surgical expertise, taking into consideration that these injuries occur in young fit sportsmen and women who may aspire for early return to sporting activity.[31]

More recently, endovascular intervention has become a popular minimally invasive option for repair of dissection in neck arteries.[32] Endovascular intervention may take the form of mechanical thrombectomy, endovascular stenting – open and covered, and coil embolization of a pseudo-aneurysm.[33] An early study reported a technical success rate of 99% for stenting.[34] Nonetheless, endovascular treatment is reserved for repair of progressive enlargement of a pseudoaneurysm or in selected cases of ongoing neurological deficit.[35] Adverse effects of endovascular therapy include vessel rupture, generation of thromboemboli and stent thrombosis that has been reported in 45% of cases.[36] Hence the need for longer-term anti-platelet treatment.

Emerging technology

Optical coherence tomographic angiography (OCTA) is a technique that has been employed for visualisation of the retinal artery to help evaluation of aneurysmal retinopathy.[37] It is a non-invasive imaging method

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that enables depth resolution in blood vessels by using data from an angiogram coupled with data of the blood vessel structure to produce a three-dimensional cube image.[37] OCTA in neck artery dissection has shown promise in imaging aneurysms,[37] and OCTA has revealed intimal disruptions and micro-thrombi in the internal carotid artery that were not visualised using CT or MR.[38] Thus, it has the potential for earlier conversion of non-surgical to surgical management to prevent the risk of stroke in young patients, but there is also potential risk of over-treatment.

Conclusion

Neck artery dissection, which often follows unsuspecting sport injury, mostly affects young people in the prime of their lives. It is a challenging diagnosis and requires a high degree of suspicion on a background of a remote history with little or no symptoms. While duplex ultrasound is useful as a screening tool, the threshold for CT-A should be low and guided by the expanded Denver criteria. Confirmation of neck artery dissection should trigger an urgent referral to the vascular surgery team. Early intervention not only prevents stroke in these individuals but is cost effective compared with the treatment spectrum for the patient with established stroke. The mainstay of treatment in neck vessel dissection is careful monitoring, rest and medication that includes anti-platelet agents for up to 6 months. Because of the lack of high-level evidence, although medical management is recommended for early-grade neck artery dissection, surgical or endovascular repair may be individualised in a select few with early-grade injury based on the requirement to return early to sporting activity or personal preference. For Biffl grade III or greater neck artery injury, surgical or endovascular intervention is the optimum choice. Follow-up CT-A is recommended on day 7 and at 3 months in these patients.

Competing interests

None declared.

Data availability statement

Not applicable to this article as no datasets were generated or analysed during the current study.

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