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REVIEW

Blunt Cerebrovascular Injuries
A review of the literature

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الإصابات الكَليلَة للأوعية الدموية في الرأس والرقبة
مراجعة الأدبيات

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الملخص: لقد نالت الإصابات الكَليلَة للأوعية الدموية في الرأس والرقبة اهتمام العديد من الباحثين عالمياً، ويتضح ذلك من الكمية الهائلة للأدبيات المتوافرة في محركات البحث المعروفة. إن الاهتمام الذي حظيت به هذه الإصابات قد يُعزى إلى خطورة النتائج المرتبطة بها من ناحية معدلات الوفيات والمراضة التي تصيب الذين يتعرضون لها، خاصة مع إمكانية تشخيصها وتجنب مضاعفاتها باستخدام أجهزة الأشعة المتطورة. إن الخطوة الأهم في عملية تجنب عواقب هذه الإصابات هو تشخيصها المبكر بواسطة فحوصات يتم إجراؤها اعتماداً على آلية الإصابة الأكثر ارتباطاً بالإصابات الكليلة للأوعية الدموية في الرأس والرقبة. نعرض هنا مراجعة شاملة فيما يتعلق بآلية الإصابة والتصوير الشعاعي والعلاج في حالات الإصابات الكليلة، وتم تحديد المقالات من خلال البحث في الشبكة العالمية الطبية وسجل كوكرين المركزي للتجارب ذات الشواهد باستخدام الكلمات الرئيسية الآتية: كليلة، وعائية، السباتي، الفقري، الصدمة، السكتة الدماغية، تدابير علاجية، وقسطرة جراحية.

مفتاح الكلمات: وعائي، سباتي، فقري، إصابة، سكتة دماغية، علاج، قسطرة جراحية.

ABSTRACT: Blunt cerebrovascular injuries (BCVI) have been a topic of interest to many researchers worldwide as evidenced by the vast amount of available literature. The interest in these rare injuries is probably due to the significant possibility of mortality and morbidity amongst patients who sustain them, when the employment of radiological screening methods could prevent such an outcome. Recognition of these injuries is the most important step towards prevention of adverse outcomes. We present a comprehensive review of the literature regarding the mechanism of injury, imaging, management, and complications of BCVI. Articles were identified through a search of MEDLINE and the Cochrane Central Register of Controlled Trials using the keywords Blunt; Vascular; Carotid; Vertebral; Trauma; Stroke; Management, and Endovascular. The search was limited to humans and articles in English.

Keywords: Vascular; Carotid; Vertebral; Trauma; Stroke; Management; Endovascular.

Trauma caused by motor vehicle collisions (MVCs) is currently the third most common cause of death globally, with 90% of these deaths occurring in developing countries including Oman. In fact, Oman has one of the highest rates of deaths from MVCs in the world, with a mortality rate of 20–30 per 100,000.[1]

Some injuries may be evident at the time of admission, while another class of injuries may remain occult for some time and then become evident when it may be too late for any intervention.

Blunt cerebrovascular injuries (BCVIs) are one of the occult injuries caused by blunt trauma. They are considered to be relatively uncommon; however, they carry a high rate of mortality and morbidity if they remain undetected.

BCVIs may or may not initially present with symptoms or signs that warrant suspicion of such injuries and therefore the provision of adequate diagnostic investigations and management is crucial. The realisation of this fact led to increased awareness of the amplitude of such injuries and the need to establish adequate screening criteria and cost-effective screening modalities.

If symptoms do present, however, they are usually attributable to focal neurological deficits

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Table 1: Blunt cerebrovascular injuries grading scale

Injury grade	Angiographic findings
I	Luminal irregularity or a dissection/ intramural haematoma with <25% luminal narrowing
II	Dissection or intramural haematoma of 25% of the lumen
III	Pseudoaneurysm
IV	Vessel occlusion
V	Vessel transection

Source: Cothren, et al.[10]

caused by ischaemia of the carotid or vertebral artery territories, or are due to a traumatic carotid-cavernous fistula manifesting as orbital pain, proptosis, hyperaemia, cerebral swelling, or seizure.[2]

Incidence of BCVIs

The general incidence of BCVIs amongst all trauma admissions has been reported in the literature as <1%. The incidence of blunt carotid artery injuries (BCIs) reported in the literature ranges from 0.08–0.27% while the incidence of blunt vertebral artery injuries (BVIs) ranges from 0.20% to 0.77%.[3,4] Although these patients are a small fraction of all trauma patients, they carry risks of stroke and mortality that are as high as 58% and 59% respectively.[5,6,7]

Mechanism of Injury

The three basic mechanisms of injury to the cerebrovascular vessels are: 1) Severe hyperextension and rotation; 2) Direct blow to the vessel, and 3) Vessel laceration by adjacent bone fractures.[2] Injuries to the extracranial carotid artery are most commonly due to hyperextension of the vessel over the lateral articular processes of C 1–3 at the base of the skull. Direct blows to the vessels in seat-belt injuries and hanging attempts are also possible causes. Injuries to the intracranial segment of the carotid artery are mostly due to basal skull fractures.[2] Vertebral artery injuries occur most commonly in fractures involving the course of the vessel through the transverse foramina of C 2–6.[8]

Pathogenesis

With any of the mechanisms of injury mentioned above, the pathological changes in the vessels are that of intimal disruption, dissection, pseudoaneurysm formation, carotid-cavernous fistula, thrombosis and complete transection of the artery.[2,3] The resultant ischaemia caused by these injuries is thought to be due to the following: 1) Dissection of the artery causing haemodynamic instability, and 2) Intimal disruption exposing subendothelial collagen fibres and promoting platelet aggregation, subsequent thrombosis and thromboembolism.[3]

Is Screening Necessary?

Given the possible catastrophic implications of these injuries, the very high stroke rate in this population, and the increased awareness amongst health care professionals, the following question has been raised among investigators: "Is it necessary to implement screening protocols for patients at high risk of these injuries?"

The argument proposed by some investigators, who disagree with the implementation of screening protocols, is that screening this population of patients is not very cost-effective, nor does it really affect the outcome. In addition, after exposing the patient to aggressive screening and the risks of invasive digital subtraction angiography, a decision on treatment with anticoagulation therapy, interventional angiography or surgery is not always feasible. This would be because of the other morbidities from which patients of blunt trauma usually suffer, such as head injuries.[9]

However, other investigators attempted to prove that aggressive screening and early intervention is indeed justified and cost-effective. In a prospective analysis, between January 1996 and June 2004, Cothren *et al.*, screened 727 patients with blunt trauma who had injuries highly suspicious of an underlying BCVI, (according to comprehensive screening criteria) and underwent a 4-vessel cerebrovascular angiogram. Of these patients, 244 were identified as having BCVIs. Antithrombotic therapy was immediately started in the 187 asymptomatic patients who had no contraindications. Using the estimated stroke risk stratification according to the degree of carotid artery injury found on angiography [Table 1],[10]

Table 2: Screening criteria for blunt cerebrovascular injuries

Denver Criteria	Memphis Criteria
Signs/symptoms	
Arterial haemorrhage or expanding haematoma	Cervical spine fracture
Cervical bruit	Neurological exam not explained by brain imaging
Focal neurological deficit	Horner's syndrome
Neurological exam inconsistent with head computed tomography (CT) findings	Le Forte II or III fracture pattern
Stroke on follow-up head CT	Basilar skull fracture with involvement of the carotid canal
	Neck soft tissue injury (seatbelt sign or hanging or haematoma)
Risk factors	
Le Forte II or III fracture pattern	
Cervical spine fracture	
Basilar skull fracture with involvement of the carotid canal	
Diffuse axonal injury with Glasgow Coma Scale <6	
Near hanging with anoxic brain injury	

Source: Arthurs, et al.[2]

antithrombotic therapy was able to avert ischaemic neurological events in 32 patients. The authors concluded that the ability of screening protocols to detect these patients who are at risk of stroke, and subsequently preventing it by antithrombotic therapy, decreases the long-term morbidity associated with strokes.[10]

Screening Criteria

So far, two well-established screening criteria for BCVIs exist, the modified Denver criteria and the Memphis criteria [Table 2].[2] The Denver criteria was the first screening protocol to be established at the Denver Health Medical Centre and was later modified by its authors. Using this screening method, the percentage of patients who underwent screening during the time span of their study is 4.8%, and BCVIs were identified in 18% of these patients, with an overall incidence of 0.86% of BCVIs among all blunt trauma patients identified in their institution.[6] Using the Memphis criteria, 3.5% of patients were screened with BCVIs identified in 29% of them. The overall incidence of BCVIs in this study was found to be 1.03%.[11]

Modalities of Screening

Screening criteria are based on recognising types of injury mechanisms that have been shown to be highly associated with BCVIs. The advent of multi-slice computed tomography (CT) scanners has enabled eligible patients to undergo screening with a sensitivity and specificity that is comparable to angiography, the gold standard screening tool. The treatment of most injuries is anticoagulation and in some instances, wherever indicated, endovascular therapy might be the best choice.

DIGITAL SUBTRACTION ANGIOGRAPHY (DSA)

Digital subtraction angiography (DSA) is considered to be the gold-standard diagnostic modality to detect BCVIs[12,13] yet it carries a risk of serious complications given that it is an invasive modality. The risks attributable to DSA are similar to those of stroke and haemorrhage. The latter limitations and the fact that DSA is expensive and technically demanding limit its effectiveness as a screening modality for BCVIs.[1,14]

MULTIDETECTOR COMPUTED TOMOGRAPHIC ANGIOGRAPHY (MDCTA)

Multidetector computed tomographic angiography (MDCTA) has emerged as a very convenient and effective screening tool for BCVI,[15–19] especially after the advent of multidetector computed tomography (MDCT), with some studies reporting a sensitivity and specificity approaching that of DSA. In multitrauma patients, MDCT is routinely utilised to screen for injuries of the head, neck, spine, chest, abdomen and pelvic injuries. A MDCTA protocol to screen for BCVIs in high-risk patients is conveniently performed at the same time. It is also less expensive and non-invasive compared to DSA.[14]

In the literature, there only two prospective studies comparing single-slice computed tomographic angiography (CTA) and magnetic resonance

angiography (MRA) to DSA which were done by the Denver and Memphis groups. In their studies, CTA had a sensitivity of 47–68% and a specificity of 67–99%. CTA missed 55% of Grade I, 14% of Grade II, and 13% of Grade III injuries.[11,12] The authors concluded in this study that CTA should only be used to diagnose BCVIs when DSA is not available. However, it is important to note that the CTA scanners used at that time were single slice CTAs, which along with other limitations such as timing contrast injection, image acquisition protocols, post-image processing delays (reformatting process), and inexperience with interpretation, might have accounted for the disappointing results in these two studies.[14]

In 2003, 16-slice MDCTs were widely available across the USA and so trauma surgeons found resistance to using DSA.[2]

In a study by Berne *et al.* in 2004, 486 patients, identified to be at risk of BCVIs, underwent CTA. Patients who had a negative study received no further cerebrovascular imaging and were monitored for cerebral ischaemic complications. Patients who had a positive CTA underwent DSA to confirm the findings. The results showed that CTA had a sensitivity of 100% and a specificity of 94.0% and none of the patients who had a negative CTA subsequently developed any ischaemic neurological events.[20] These results were questioned because the patients who had a negative CTA were not confirmed to be true negatives by DSA. In the same year, Bub *et al.* published a retrospective analysis that included 32 patients who were suspected to have BCVIs and underwent both MDCTA and DSA. Results showed that 17 BCVIs were identified in 15 of the 32 patients. MDCTA done by three different radiologists had a sensitivity of 83–92% and specificity of 88–98% for detecting BCIs. However, the sensitivity for vertebral artery injuries ranged from 40–60%, and the specificity ranged from 90–97%.[21] In 2006, three studies, published by three different institutes, produced very similar results with regard to the use of 16-slice MDCT to scan for BCVIs in high risk patients. All three groups found MDCT to be as accurate as DSA in detecting BCVIs,[15,18,19] with the exception of one patient in the study done by Eastman *et al.*, where a Grade 1 injury was missed by MDCT and was detected by DSA.[19] In all three studies, none of the patients who was initially found to have a negative CTA later developed complications attributable to an undetected BCVI.

Although many of the studies discussed above do prove that the accuracy of CTA approaches that of DSA, the lack of prospective data comparing 16-slice CTA to DSA and the small number of patients included in each study limit the reliability of the results and the validity of the conclusion that CTA is equivalent to DSA. It is recommended that patients who are at high risk for BCVIs should be considered for DSA regardless of their CTA findings, until a large, multicentre prospective trial proves the case.[2,14]

MAGNETIC RESONANCE ANGIOGRAPHY (MRA)

The advantage of MRA over MDCT is the fact that it does not carry the risk of ionising radiation or the use of contrast, but in a multi-trauma setting MRA is not really a feasible or practical option. The time needed for the patient to be taken to the MR suite, for the test to be performed and the number of personnel required to perform the study are all limitations to its accessibility in a multi-trauma setting. However, in two prospective analyses by the Denver and Memphis groups, which compared CTA and MRA to DSA in patients suspected to have BCVIs, MRA had a sensitivity of 50–75% and specificity of 67–100%.[11,12]

SONOGRAPHY

Duplex ultrasound is a portable and inexpensive tool that is well established in monitoring and diagnosing non-traumatic cerebrovascular pathology. However, the fact that it is operator dependant, has a limited ability to visualise cerebral vasculature at the base of the skull and also to visualise minimal injuries and dissections that are not associated with disrupted flow, has limited its role in detecting BCVI.[2,14] In the few studies performed to evaluate the accuracy of duplex ultrasound to detect BCVIs, results were disappointing, showing that duplex has a low sensitivity and specificity compared to DSA and that it missed injuries in patients who later on developed ischaemic complications attributable to these injuries.[22,23]

Management

The early detection and treatment of BCVIs has been shown to reduce the morbidity and mortality related to these injuries. Both symptomatic and asymptomatic patients with BCVI should be managed and closely monitored for any neurological deterioration. The optimal treatment of patients with BCVI is not yet well established. The appropriate management of BCVI depends on the specific injury and its anatomic site.[3,7,24–28] Controversies do exist in the management of dissection, thrombosis and pseudoaneurysm formations. Older studies prefer surgical over non-surgical management of dissection and thrombosis.[29] However, most of these lesions extend beyond the skull base, and are therefore not amenable to open surgical repair. In addition, most neurologic sequelae of these injuries are related to acute thrombosis, thrombus propagation and distal embolisation, making surgical reconstruction irrelevant. There is therefore growing support for nonsurgical management of dissections and thromboses.[13,29–32] Prospective trials comparing surgery with anticoagulation are not feasible.

ANTICOAGULATION THERAPY

Although its efficacy has not been proven, anticoagulation therapy has been considered the treatment of choice in patients with BCVI, especially for those with high located intimal flaps, extensive dissections and small inaccessible pseudoaneurysms. Anticoagulation is documented to prevent cerebral embolisation and to avoid permanent occlusions of injured vessels.[3,7,25] Li [29] and Parikh[33] have suggested improvement of outcomes with anticoagulation alone.

Initial systemic heparin therapy is safe and should be considered if no contraindications are present, or if the anticipated benefit outweighs the risk of bleeding in high risk patients. This should be followed by oral anticoagulation therapy with warfarin (Coumadin) for three to six months.[3,7,25,28] Reported complications of anticoagulation include intracranial haemorrhage, gastrointestinal bleeding, retroperitoneal haemorrhage, bleeding at the site of blunt solid organ injury and re-bleeding from surgical wounds. These complications are reported to occur in 25–54% of trauma patients.[6]

Most authors agree on the need for follow-up for patients treated initially with anticoagulation for their BCVI in order to detect any subsequent development of pseudoaneurysms requiring surgical or endovascular interventions.[24–34]

ANTIPLATELETS THERAPY

Aspirin (and in few other reports, clopidogrel) has been used as an alternative to heparin in the treatment of patients with BCVI. It has been shown to have similar efficacy to heparin in preventing neurological events. In fact, aspirin has been proven to have a better safety profile with less risk of bleeding compared to heparin, especially in the population of trauma patients.[11,26–28]A combination therapy of anticoagulation and antiplatelets has been practised, but any advantage has not been proven; it definitely requires further prospective trials to elicit any added benefit to the single therapy.

OPEN SURGICAL REPAIR

Direct surgical repair is advocated for discrete lesions at the carotid bifurcation or lesions below the base of the skull making it amenable for proximal and distal control. Most of the time, open surgical repair is rarely considered for patients with BCVI for the reasons mentioned above.[3,7] Management of pseudoaneurysms is less controversial and most authors recommend surgical repair whenever it is technically feasible.[24,29,33] Small or inaccessible pseudoaneurysms have been managed by anticoagulation with or without proximal ligation, or by the rarely used extracranial-intracranial bypass.[7,35]

ENDOVASCULAR THERAPY

With the development of the less invasive endovascular techniques, most carotid pseudoaneurysms and dissection flaps that result from BCVI have been managed successfully with angioplasty and endoluminal stenting.[36–38] Endovascular therapy is an alternative to open surgical repair and of great value in the distal carotid lesions which are not amenable for open surgical repair. This therapy is recommended in cases where there is a contraindication for anticoagulation. Bare or covered stents, either balloon expandable or self-expanding, have been used extensively with good outcome and no further neurological events reported.[36,39,40] As compared to data on peripheral stenting, antiplatelets

therapy (aspirin and clopidogrel) is advised after endoluminal stent therapy to prevent stent thrombosis and embolic ischaemic events.[41] Balloon occlusion techniques are a well established mode of treatment for carotid-cavernous sinus fistulae, and the results are fair to good.[13,29–32]

Outcome

The prognosis of BCVI is generally poor. All reported cases of complete arterial disruption have been fatal. Other injuries of dissection, thrombosis and pseudoaneurysm formation carry variable outcomes depending on the site of the injury as well as the time of detection of these injuries. Mortality rates after BCVI have been reported to be 5–40% and reasonable neurologic recovery in only 20–60% of all survivors.[13,29–32] Although there is some evidence to show improved outcomes with anticoagulation and antiplatelets therapy and in selected cases of surgical and endovascular interventions, the outcome of BCVI depends more on the speed of diagnosis.[12,32,33] A high index of suspicion and the maintenance of aggressive evaluation protocols for patients with possible BCVI will definitely avoid diagnostic delay and may improve the overall outcome.

Conclusion

Blunt cerebrovascular injury (BCVI) may be overtly present in more than 1% of patients with blunt trauma. Aggressive screening strategies uncover injuries in up to 44% of those screened. If not appropriately diagnosed and treated in a timely manner, many such injuries are responsible for significant morbidity and mortality. Aggressive screening protocols are now feasible using newer generation, multidetector helical scanners capable of detecting small intimal defects in a relatively non-invasive manner.

Treatment has focused on reducing the atheroembolic tendency of the disrupted vessel wall. Anticoagulation with heparin and anti-platelet agents has been used; however, in a multitrauma patient, the risks of bleeding and the need for immediate surgery must be taken into account. Certain injuries to cervical vessels may be amenable to endovascular therapy. Therefore a multi-disciplinary team consisting of the emergency physician, trauma surgeon, vascular surgeon, interventional radiologist and intensivist is recommended for immediate and follow-up care. A high index of suspicion and maintaining aggressive evaluation protocols for patients with possible BCVI will definitely avoid diagnostic delay and may improve the overall outcome after BCVI. Further research into optimal treatment strategies is warranted.

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