REVIEW

Current Diagnosis and Management of Blunt Internal Carotid Artery Injury

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Background. Blunt carotid artery injury (BCI) is a rare but potentially devastating injury. When undiagnosed it can result in severe disability or death.

Methods. A Medline-based literature search was performed using key words ‘blunt carotid injury’ and cross-referenced with further original papers obtained from the references from this search.

Results and conclusions. The incidence of BCI is very low. However, given the serious consequences of a missed injury, recent efforts have focussed on targeted screening for this injury in trauma patients. Conventional angiography remains the investigation of choice but may be superceded in the future by non-invasive methods such as magnetic resonance angiography or CT angiography. Operative intervention is rarely required and anti-coagulation remains the treatment of choice where dissection or pseudoaneurysm is diagnosed. The role of anti-platelet therapy is currently being investigated. Endovascular management using stents has been described but medium to long term results are not yet available.

Key Words: Blunt carotid artery injury; Angiography; Computed tomography.

Introduction

Blunt carotid artery injury (BCI) was first described by Yamada et al. in 1967.¹ The incidence in the recent literature varies widely between different centres. The diagnosis is often delayed and usually not made until after onset of neurological symptoms and signs. Neurological morbidity rates of 40–80% in all patients presenting with blunt carotid artery injury and mortality rates of 5–40% have been reported.²–¹¹ Recent literature shows an increasing frequency of blunt carotid injury without significant improvement in rates of pre-stroke diagnosis.¹²

The purpose of this paper is to review the current literature relating to diagnosis and management of blunt carotid artery trauma and to discuss the newer imaging and treatment modalities available for its management.

Epidemiology

There are only a few small number of publications relating to the incidence of BCI. Reported incidence from retrospective studies, range from 0.08 to 0.27% with motor vehicle accidents being the most common mechanism of injury (69%). BCI was the most common form of injury (75%) with one quarter of these having an associated pseudoaneurysm.⁹,¹¹–¹³ However, Fabian et al. published a large single-institution series of BCI, with a reported incidence three times that cited in previous reviews (0.67% of patients after a motor vehicle accident) using an aggressive diagnostic approach.¹⁴ Aggressive screening of patients with blunt head and neck trauma using four-vessel cerebral angiography and helical CT angiography identified an incidence of 1.03% of BCI.¹⁵

Aetiology and Pathogenesis

Types of vascular injury found associated with BCI include intimal flap/dissection, occlusion/thrombosis, pseudoaneurysm, carotid cavernous fistula...
formation, complete transection or a combination of these lesions. Various mechanisms of injury have been postulated, but they probably occur secondary to a stretching injury as seen with rapid deceleration. The internal carotid artery is thought to stretch over the lateral masses of the third and fourth cervical vertebrae producing an intimal tear, most often located in the distal internal carotid artery. Dissection occurs when circulating blood penetrates into the arterial wall through such an intimal tear. A false lumen appears if this blood re-enters the true lumen of the internal carotid artery. The intramural clot may also extend subadventitiously causing a dissecting aneurysm.

Cerebral ischaemia following BCI may result from one of the two mechanisms. Damage to the arterial wall can cause internal carotid artery dissection resulting in haemodynamic disturbance. Damage to the arterial intima exposes subendothelial collagen, a thrombogenic surface and potent platelet aggregator. This can cause transient cerebral or retinal ischaemic attacks. Most cerebral infarctions are caused by this phenomenon, rather than by haemodynamic disturbance.

Aetiology

The mechanism of injury resulting in internal carotid artery dissection following BCI is usually direct trauma resulting from motor vehicle accidents, assault and falls.

More unusually, cases of BCI have been described following minor head and neck trauma, or associated with coughing or vomiting. BCI may follow uncustomed physical exercise such as bodybuilding or car pushing. Cases have also followed chiropractic manipulation. Rhythmical flexion and extension of the neck to music (‘head-banging’), prolonged telephone usage with flexion in the neck or child-birth are considered provoking factors in 25–41% of patients with BCI.

Only one case of traumatic dissection following a minor head injury has been reported in a patient with a pre-existing diagnosis of fibromuscular dysplasia.

Symptoms and Signs

Presentation of ICAD following BCI is highly variable. Clinical symptoms and signs may develop hours or weeks after the injury. Delay in recognition is common as the injury itself is often asymptomatic or is confounded by alcohol or drug intoxication, head injury, extremity or spinal cord injury or shock. Less frequently, only local manifestations are present, and their presentation can be delayed for up to 60 days after the initial insult.

The most common local symptoms of BCI are headache and/or cervical pain. The next most common symptoms are cerebral or retinal ischaemia. Horner’s syndrome associated with pain is characteristic of BCI. Other less common manifestations are described in Table 1. Despite the small numbers of patients reported with BCI, there is data on proportions of patients presenting with major and minor strokes. In a series of 27 patients described by Miller et al., three of 3 (100%) with occlusion following trauma developed stroke; five of 22 (23%) with internal carotid artery dissections and one of 2 (50%) with carotid-cavernous fistula developed major strokes.

Horner’s syndrome (oculosympathetic paresis) is related to the involvement of the internal part of the pericarotid sympathetic plexus. The nerve injury is postulated to be caused by a disruption of the blood supply (vasa nervorum) to the superior cervical ganglion or by direct injury to the sympathetic nerve plexus. It is important to note that Horner’s syndrome is not pathognomonic for carotid artery trauma and that it may also be a sign of vertebral artery dissection.

Cranial nerve palsy is seen in 5–12% of cases. The hypoglossal nerve is the most commonly affected (5%). It has been suggested that this is due to an impairment of the blood supply by mechanical, embolic, or haemodynamic mechanisms. The III, IV and VI cranial
nerves may also be affected when the dissection extends into the cavernous sinus.\textsuperscript{16,39}

**Screening for Blunt Carotid Injury**

Aggressive screening for BCI has been advocated in order to increase early diagnosis of the condition and improve outcome. Screening protocols have been shown to identify less severe injuries which would otherwise have been missed.\textsuperscript{40} Screening has been shown to double the rate of vertebral artery injury diagnosis but has not yet been shown to decrease the risk of stroke.\textsuperscript{15}

A recent study has identified specific patterns of injury associated with intracranial and extracranial carotid injury. Suggested triggers for screening of suspected BCI include cervical spine fracture, Horner’s syndrome, Le Fort II or III facial fracture, skull base fracture involving the foramen lacerum and neck soft tissue injury.\textsuperscript{15} Extracranial injuries were more commonly occult, predicted by a Glasgow Coma Scale (GCS) of $\leq 8$ and thoracic injury. Intracranial injuries on the other hand were frequently detected on initial investigations and were associated with GCS $\leq 8$ and facial injuries.\textsuperscript{41} More liberal screening for BCI has been proposed by Kerwin \textit{et al.} who suggest expanding the criteria for screening to include all patients with basilar skull fractures and all patients with unstable cervical spine fractures.\textsuperscript{42}

**Grading of BCI Injury**

Discrete BCI types may behave differently and, may therefore warrant individualised treatment. Until recently, the absence of a formal BCI grading scale has been a major impediment to formulating proper management protocols.\textsuperscript{10,53} Biffl \textit{et al.} designed such a grading scale based on arteriographic appearance of the lesions (Table 2).\textsuperscript{2}

<table>
<thead>
<tr>
<th>Injury grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>Luminal irregularity or dissection with $&lt; 25%$ luminal narrowing</td>
</tr>
<tr>
<td>II</td>
<td>Dissection or intramural haematoma with $\geq 25%$ luminal narrowing</td>
</tr>
<tr>
<td>III</td>
<td>Pseudoaneurysm</td>
</tr>
<tr>
<td>IV</td>
<td>Occlusion</td>
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<tr>
<td>V</td>
<td>Transection with free extravasation</td>
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**Investigations**

**Conventional angiography**

Conventional carotid angiography is considered the gold standard for diagnosis of BCI.\textsuperscript{12} It demonstrates the extent of proximal and distal injury, intracranial thromboembolic material, the anatomy of the intracranial circulation, bilateral injuries and associated vertebral injuries. Typical findings are irregular and often tapered stenoses with a characteristic ‘string sign’. The stenosis usually begins 2–3 cm distal to the bifurcation and often extends to the base of the skull where it ends with an abrupt luminal reconstitution. The dissection can also extend through the carotid canal and involve the intracranial arteries. The pathognomonic sign of a carotid double lumen is rare (4%). Tapered ‘flame shaped’ occlusions, aneurysmal dilatations or distal branch embolisations are other typical findings.\textsuperscript{16} Occasionally, irregularity of the vessel wall may be the only finding.\textsuperscript{32} Internal carotid artery redundancies (loops, coils and kinks) are significantly correlated with the occurrence of BCI.\textsuperscript{43} Appearances are less specific for intracranial than extracranial dissections.\textsuperscript{16}

**Computed tomography (CT)**

CT scan is not used to diagnose BCI but can be adjunct to its management. CT scan may show a focal low-density area within the middle or anterior cerebral artery territory suggesting a stroke due to BCI. The use of CTA was found to increase the rate of detection of blunt vertebral injury and reduce the time from injury to detection.\textsuperscript{44} However, the rate of missed injury using CTA remains uncertain.\textsuperscript{45} Therefore, conventional angiography remains the recommended diagnostic modality and any newer non-invasive techniques will require validation against it. Advantages of CTA include the short examination time, reduced dye load and the potential to acquire three-dimensional images. Associated facial, basilar skull fractures and brain injuries can be assessed concurrently. A significant disadvantage with CTA is its tendency to underestimate the degree of stenosis.

**Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA)**

MRI can obtain morphological data and MRA reflects intraluminal blood flow. MRI can define the length of the dissection,\textsuperscript{46} measure arterial wall thickness\textsuperscript{47} and estimate age of haematomata.\textsuperscript{48} MRA can demonstrate a
carotid double lumen and string sign. Typical MR findings in the presence of BCI are an increase in external diameter of the artery and narrowing of the lumen. Levy et al. established that in patients with BCI, MRI showed a diagnostic sensitivity and specificity of 95 and 99%, respectively, whereas in MRA it was 84 and 99%, respectively, when compared with four-vessel angiography.

MRA is a safe and non-invasive procedure. Two- or three-dimensional time-of-flight images with or without contrast are obtained. MRA permits examination from an infinite number of projection angles, avoids vessel overlap and bony interference. Furthermore, diffusion-weighted sequences allow identification of ischaemic infarction within minutes, compared to 24–48 h using CT scan. However, factors such as cost, expediency and the need to monitor and resuscitate critically ill patients limit its applicability. Further limitations with its use include difficulty in accurately diagnosing a haematoma, difficulty in grading and a tendency to overestimate the degree of stenosis. It may also give a false appearance of vessel widening and of luminal narrowing of the carotid artery as it enters the petrous canal. However, the technology is rapidly developing and today many of the aforementioned problems can be overcome.

Ultrasound

Duplex ultrasound is more useful for the assessment of penetrating than blunt carotid artery injury. Its usefulness as a non-invasive modality is limited by the absence of acoustic windows into the mediastinum, skull base, supra-mandibular and intra-cranial regions. There are no pathognomonic signs, but highly suggestive ones include tapering of the internal carotid artery lumen distal to the bulb and an intimal flap or membrane creating a double lumen. It provides indirect evidence of more distal injuries by detecting turbulence and other flow disturbances. However, findings are not reliable in the presence of stenoses <60%. Ultrasound is useful for follow-up of patients following BCI to detect rates of healing.

Trans-cranial Doppler ultrasound (TCD) can be used to assess the haemodynamics of the intracranial circulation and also to detect embolisation from a dissected vessel. The differences in the middle cerebral artery velocities reflect compensatory responses in the circulation which depend on change in perfusion pressure above the lesion.

Treatment of Blunt Carotid Artery Injury

To date there have been no randomised controlled trials to establish optimum management of BCI. Currently available treatment modalities are anticoagulation, antiplatelet therapy and open surgery. More recently, reports of endovascular treatment of these injuries have appeared and this may in time provide a viable alternative to the more invasive surgical options.

Anticoagulation and anti-platelet therapy

Anti-coagulation

The rationale for anticoagulation therapy is to prevent cerebral embolisation from the dissected artery and avoid permanent occlusion in tight stenoses (grades I–IV). Patients treated with systemic heparin have an improved neurological outcome. Heparin therapy has been shown to prevent injuries progressing to a higher injury grade, reduces the number of strokes and prevents neurological deterioration from injury diagnosis to discharge. However, bleeding complication rates as high as 40% have been reported with heparin therapy. These include intracranial, retroperitoneal, and gastrointestinal bleeding complications. Low molecular weight heparin (LMWH) has not yet been used in large numbers of patients. However, with its excellent anti clotting properties and lower bleeding complication profile, it may prove useful in the management of these patients.

In patients with major cerebral infarction, the risk of haemorrhagic transformation is considerable and may outweigh the expected benefits of anticoagulation. Other risks include a possible increase in wall haematoma and delay in healing of the false lumen. Furthermore, heparin may be totally contraindicated where the risks of bleeding outweigh the benefits of anticoagulation therapy. In a study of 22 patients, Wahl et al. found that 14 (63.6%) were unsuitable for anticoagulation therapy due to bleeding from other sites. On the other hand, Biffl et al. reported the outcome in eight patients with grade IV injuries. Four patients, not commenced on anticoagulation suffered severe neurological deficit while the remaining four patients treated with heparin immediately upon diagnosis survived without deficit.

There have been no data to establish the optimal duration of anticoagulation. However, 3–6 months is probably suitable in most cases. It may be stopped earlier if complete recanalisation is demonstrated on duplex or MR follow-up, and may be...
continued if there is persistent aneurysm or remaining wall irregularities.\textsuperscript{16,35} Despite anticoagulation, new symptoms occur in 7–14% of patients.\textsuperscript{21,22,32}

\textbf{Anti-platelet therapy}

Antiplatelet agents can be considered as an acceptable alternative when anticoagulation is contraindicated.\textsuperscript{16} Their use is especially recommended in grade I lesions.\textsuperscript{2} However, more data are needed from multi-institutional studies to support their use. Most patients treated with anti-platelet agents are deemed unsuitable for full anticoagulation due to associated injuries. Reports from small series of patients provide support for the use of anti-platelet agents in patients with Grade I–II injuries.\textsuperscript{52} A larger retrospective series reported by Wahl \textit{et al.} showed a higher number of bleeding complications among patients treated with heparin.\textsuperscript{57} They concluded that anti-platelet agents could be used in patients at high risk of bleeding complications from either intracranial or major torso injuries with heparin reserved for those patients with isolated blunt carotid injuries.

\textbf{Surgical procedures}

The need for surgical intervention for BCI is rare but several techniques are available. The requirement for surgery is determined by the thrombogenicity of the injured carotid artery, the state of the collateral circulation to the brain, presence of an expanding haematoma or worsening neurological symptoms despite anticoagulation.

\textbf{Extracranial–intracranial bypass}

Extracranial–intracranial bypass has proven a useful means of revascularisation.\textsuperscript{55} The currently preferred method is excision of the damaged arterial segment with reconstruction of the ICA to preserve flow if at all possible.\textsuperscript{57}

\textbf{Carotid ligation}

Carotid ligation or permanent balloon occlusion of the proximal ICA can be safe and simple assuming the presence of adequate collateral flow has been established. However, delayed ischaemia because of propagation of thrombus or embolisation is a potential threat in these patients, in whom intravenous heparin therapy is administered after ligation.\textsuperscript{57} Although carotid occlusion remains a reasonable option in some patients, they may be at increased risk of developing cerebral ischaemia and perhaps intracranial aneurysm later in life.\textsuperscript{58} This is pertinent, as these patients are usually young or middle-aged adults with life expectancies similar to the general population.\textsuperscript{59}

Sacrifice of the ICA would obviously be contraindicated in patients with bilateral BCI.

ICA pseudoaneurysms may develop as a result of BCI and cause symptoms despite adequate anticoagulation. Several surgical techniques to exclude ICA pseudoaneurysms have been described and may involve either a direct or indirect approach.

\textbf{Direct approach}

This involves pseudoaneurysm resection and reconstruction. Arterial, synthetic or saphenous vein grafts are used. The major morbidity associated with high cervical approach is damage to the pharyngeal and superior laryngeal branches of the vagal nerve, resulting in dysphagia and dysphonia. Facial weakness, cranial nerve palsies and deafness are other potential complications.\textsuperscript{57}

\textbf{Indirect approach}

This procedure is preferred when the pseudoaneurysm is not accessible through a high cervical approach or when the morbidity associated with this approach is considered prohibitive.

\textbf{Endovascular stenting}

Several recent reports have recommended endovascular stenting in combination with coil placement, for pseudoaneurysms associated with BCI such as in grade III lesions.\textsuperscript{2,60,61} Current indications for stent placement include enlarging pseudoaneurysms and dissections that progress and threaten to limit flow despite full anticoagulation. This treatment has been shown to be efficacious in 89% of grade III BCI.\textsuperscript{2} It has also been shown to prevent late complications in one small series.\textsuperscript{62}

When considering endovascular stenting, two important points need to be remembered. Firstly, the acutely injured ICA should not be manipulated within 48–72 h of the event. This is because of the risk of stroke from manipulation of catheters in the acutely injured vessel. The current recommendation is to wait 7 days before attempting to stent BCI.\textsuperscript{63} Secondly, endovascular stents placed in traumatised arteries should be treated adjunctively with full systemic anticoagulation to prevent stent occlusion resulting in stroke.\textsuperscript{2} Recent evidence suggests that anti-platelet therapy alone may be adequate to prevent thrombotic complications for carotid artery stents.\textsuperscript{64}

Stent procedures have the obvious advantage of avoiding complex base of skull surgery. Furthermore, they can be carried out under local anaesthesia, which
allows for ongoing neurological monitoring of the patient. To date, all series are small and describe the use of either uncovered stents or stents covered with autologous vein or synthetic material. Endovascular stenting alone,65,66 or in combination with coil embolisation has been described.81,82 Bush et al. described a 100% primary technical success rate in five patients using the combined approach.68 No patient suffered permanent neurological sequelae.

Further developments in this area will include the development of endoluminal stent grafts designed specifically for the carotid artery which would exclude the aneurysm sac while maintaining antegrade flow in the internal carotid artery.69 As most patients with BCI are young, concern has been raised about the possibility of stenosis or occlusion related to intimal hyperplasia consequent to the placement of a stent in the internal carotid artery. Long term data are not yet available.

Prognosis

BCI is generally considered to have a favourable prognosis. Complete or almost complete recovery occurs in 23–85%,21,22,29 but severe persistent deficits occur in 16–37%.21,64 The big difference in prognosis between the various studies is probably due to selection of patients and diagnostic difficulties. Recanalisation occurs in 68–100% of stenosis and 25–43% of occlusions.16 One study showed no ultrasonographic progression from stenosis to occlusion.29 The probability of recanalisation cannot be predicted. It usually occurs within the first 2 months but can take up to 6–12 months from the dissection event.30,70

Conclusion

Information on BCI is limited and where available is usually limited to small series. The larger studies suggest that it is not an uncommon cause of ischaemic stroke, especially in younger patients. The true incidence is unknown but appears to occur in less than 1% of blunt trauma patients. Patients usually develop focal neurological deficits prior to diagnosis. Clinical suspicion is essential if these lesions are to be detected early. Conventional angiography remains the diagnostic modality of choice, but this may be superceded in time by MRA technology advances. Treatment must be considered on an individual patient basis depending on the presentation, grade and morphology of the lesion. Although no level I clinical trials exist, anticoagulation seems to be the treatment of choice in most cases and surgical intervention is rarely indicated. Endovascular stenting is a new and exciting area in the treatment of BCI but at present long term outcome data are not available.

References

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