Blunt Carotid Injury

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Current Treatment Options in Cardiovascular Medicine 2006, 8:167–173
Current Science Inc. ISSN 1092-8464
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Opinion statement

Blunt carotid injury (BCI) is an uncommon disorder, occurring in trauma patients as a result of cervical hyperextension, hyperflexion, or direct blow. BCI is commonly present in initially asymptomatic patients who subsequently develop devastating thromboembolic complications of their injury. Although clinical predictors of injury have been developed, they are of limited accuracy. Nevertheless, employment of clinical screening criteria is of value in identifying at-risk patients in need of diagnostic testing. Liberalized screening of these trauma patients with angiography or the latest generation (64-multidetector) CT angiography facilitates early diagnosis and provides opportunity for timely intervention in asymptomatic victims. Anticoagulation and/or antithrombotic therapy in specific categories of these patients reduces neurologic morbidity and mortality. Endovascular stenting shows promise as a treatment modality for specific subsets of individuals with BCI. Surgery remains a therapeutic option for some surgically accessible lesions.

Introduction

Although first described in detail in 1872 [1], blunt carotid injury (BCI) was until recently thought to be a rare phenomenon, the subject of isolated case reports or small case series. Analysis of a trauma registry of 14,003 patients uncovered four cases of BCI (incidence of 0.03%) [2] and a 1990 case series [3] reported an incidence of 0.08% of BCI; both examined an unscreened population. In 1996 Fabian et al. [4•], in the first large single center series on the subject, noted an overall incidence of BCI of 0.33% in all blunt trauma patients, but an incidence of 0.67% in victims of motor vehicle crashes. This data remains relevant when compared with analysis of more recent trauma data indicating an overall incidence of BCI of 0.40% in the unscreened blunt trauma patient [5].

The mechanism of blunt injury to the carotid arteries involves rapid deceleration with associated hyperflexion and/or hyperextension. Hyperextension is believed to cause BCI by stretching the vessel over the lateral masses of the third and fourth cervical vertebrae. Hyperflexion with rotation is thought to injure the artery by compressing it between the mandible and the cervical spine. Direct blow has also been known to cause BCI. Intimal injury occurs with sequela, commonly including dissection with thrombosis, and thromboembolism with secondary stroke. However, other potential sequela include pseudoaneurysm, complete vessel rupture, arteriovenous fistula, or arteriovenous-cavernous sinus fistula [4,6]. The overwhelming majority of injuries occur in the internal carotid artery, anywhere from the bifurcation to the base of the skull (or higher), with the majority being near the base of the skull [4,7]. Biffl et al. [8••] developed a grading scale for BCI based on angiographic findings, which has both prognostic and therapeutic implications (Table 1) [8••,9••,10].

The majority of studies involving BCI are related to mechanisms that involve significant energy, and many of these patients have concurrent major injuries. However, some patients have isolated BCI, perhaps involving a more minor mechanism of injury than one might expect. It is also interesting to note that carotid artery dissection due to relatively trivial trauma has been a known entity for many years. A well-known mechanism for such injury
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involves chiropractor manipulation of the neck. A current study by the Canadian Stroke Consortium (preliminary data published in letters only) \[11,12\] supports the hypothesis that sudden head movement may be a precipitating factor in many cases of "spontaneous" cervical artery dissections. Arterial structural factors, genetic and/or acquired, likely play a significant role in the etiology of such injuries. It is conceivable (but not a widely discussed theory) that such factors may play a role in certain subsets of cases of BCI in the trauma patient. Discussion of "spontaneous" carotid artery dissection is beyond the scope of this article. It should also be noted that although less common than BCI, blunt vertebral artery injury is also a well-known entity with many similarities to BCI. Although BCI and blunt vertebral artery injury are often studied and reported (and can occur) together, this paper focuses on BCI.

Perhaps the most frustrating aspect regarding the diagnosis of BCI is that historically, in an unscreened population, 70% of cases of BCI are uncovered only after onset of major neurologic sequela. Of greater significance is that in 43% of these cases, the deficit occurred after hospitalization \[4\]. The time from injury to diagnosis is often prolonged. The classic presentation of BCI is that of a neurologically intact trauma patient who subsequently develops catastrophic hemiparesis or hemiplegia. Figure 1 describes such a case with accompanying diagnostic findings. The greatest potential for successful intervention exists in those neurologically intact patients with BCI. Some of the challenges involved in early identification of BCI include relatively weak correlation between clinical predictors and presence of disease and invasiveness of the diagnostic gold standard, angiography.

Efforts have focused on developing clinical screening criteria to aid in selecting patients for diagnostic imaging. Screening criteria include anisocoria (related to Horner’s syndrome), unexplained hemiparesis, a neurologic examination unexplained by head CT scan, basilar skull fracture through or near the carotid canal, fracture through the foramen transversarium, cerebrovascular accident or transient ischemic attack, massive epistaxis, severe flexion or extension of cervical spine fracture, and massive facial fractures or neck hematoma. Implementation of such screening programs (specifically with respect to criteria unrelated to neurologic sequela) has shown promise at achieving earlier diagnosis, allowing for therapeutic interventions and improving outcomes. Employing such screening criteria, the incidence of BCI increases to approximately 1% of blunt trauma patients \[2,6,7,9\]. Cervicothoracic seat belt sign has also been studied as a predictor for BCI. It is a visible, distinct mark that can easily be identified on initial physical examination; approximately 3% of those with this finding will have BCI \[13\]. Another interesting phenomenon regarding BCI is that bilateral injuries are present in up to one third of cases \[9\]. Factors that contribute to bilateral BCIs are not well understood. One investigator comments that patients

Table 1. Denver grading system for blunt carotid injury with treatment overview

<table>
<thead>
<tr>
<th>Grades</th>
<th>Angiographic findings</th>
<th>Prognosis</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>Grade I</td>
<td>Vessel wall irregularity or dissection with &lt; 25% luminal diameter</td>
<td>Good (7% will progress)</td>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Grade II</td>
<td>Raised intimal flap, thrombus, dissection, or hematomas &gt; 25% luminal diameter</td>
<td>Fair with treatment (70% will progress)</td>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Grade III</td>
<td>Pseudoaneurysms</td>
<td>Fair with treatment</td>
<td>Anticoagulation with or without surgery or stenting</td>
</tr>
<tr>
<td>Grade IV</td>
<td>Total vessel occlusions</td>
<td>Outcome is usually assured at time of diagnosis</td>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Grade V</td>
<td>Transections</td>
<td>Very poor, high mortality</td>
<td>Surgery</td>
</tr>
</tbody>
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Figure 1. A, A 32-year-old patient was intubated and transferred to our facility hours after being involved in a bicycle crash with interim development of right hemiplegia, aphasia, lethargy, a questionable seizure, and a "blown pupil" (later appreciated to be the contralateral pupil to a Horner’s pupil). Three-dimensional reconstruction of MRI displays the location of the left internal carotid artery (ICA) injury (white arrow). B, Subsequent, four-detector CT angiography demonstrates a grade II left ICA injury (arrow) and grade I right ICA injury (arrowhead).