Blunt Craniocervical Trauma
Does the Patient Have a Cerebral Vascular Injury?

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KEYWORDS

- BCVI • Blunt • Cerebrovascular injury • Carotid injury • Vertebral artery injury • CT angiography
- Trauma

KEY POINTS

- Screening for blunt cerebrovascular injuries in high-risk patients decreases morbidity and may be cost effective (moderate evidence).
- Digital subtraction angiography remains the gold standard for diagnosing vascular injury following blunt trauma (moderate evidence).
- Many institutions have adopted multidetector computed tomography angiography as the primary diagnostic modality for blunt cerebrovascular injury screening.
- Recent evidence supports the potential of multidetector computed tomography angiography as a reference standard in the future.

INTRODUCTION

Blunt cerebrovascular injury (BCVI) involves injury to the carotid and/or vertebral arteries sustained via generalized multitrauma or directed blunt craniocervical trauma, and reflects an increasingly common, potentially catastrophic injury. Stroke remains the most consequential outcome of BCVI, causing 80% of deaths,[1] and is largely responsible for the overall mortality rate of 20% to 30%,[2,3] and permanent neurologic morbidity rate of 37% to 58%.[3]

Motor vehicle collisions contribute to 80% of BCVI; less common causes include a fall from any height, sports related injuries, assault, strangulation, or chiropractic spinal manipulation.[4] Accounting for 5% of all adult trauma,[5] up to 90% of pediatric trauma,[6] and the most common cause of death in young adults,[7] the ubiquitous nature of blunt trauma impacts health care across all levels. Once thought to account for less than 0.1% of all blunt trauma patients,[8] systematic review of 122,176 patients estimates BCVI to account for 0.18% to 2.70% of blunt trauma admissions.[9] Although estimates continue to vary slightly, most new studies support this trend of increasing incidence.[9–11] Concordantly, a 2017 systematic review demonstrated an incidence increase from 0.33% to 2.00% ($P<.001$) from 1995 to 2015.[12]

This trend seems to be at least partially attributable to a heightened awareness of BCVI, the increasing role of noninvasive imaging strategies,[2,4,13–18] and the expanding implementation of more sensitive screening protocols to identify patients at risk for BCVI.[10,12,14,18–30] Optimum treatment for BCVI is not yet known, although the importance of timely diagnosis and initiation of

any treatment before the development of neuro-logic complications has a well-established role in the reduction of morbidity and mortality. Stein and colleagues[31] studied 147 patients with BCVI at R. Adams Cowley Shock Trauma, and reported stroke rates of 25.8% in untreated patients (owing to contraindications) and 3.9% (P = .0003) of treated patients, independent of treatment type. Similarly, the recent systematic review by Shahan and colleagues[12] reported BCVI-related stroke mortality to have decreased from 37% to 5% (P<.001) over a 30-year duration.

Recent widespread acceptance of multidetector computed tomography (CT) angiography (MDCTA) as an accurate diagnostic modality for BCVI has overcome many of the inherent disadvantages of digital subtraction angiography (DSA); however, disagreement persists for defining the appropriate population to screen via MDCTA. As a noninva-sive, rapid, readily accessible, and cost-efficient modality, capable of detecting associated injuries throughout the body, CT now accounts for the greatest source of radiation exposure in trauma patients.[32] Recently, pervasive efforts to keep ra-diation dose as low as reasonably achievable have shifted, although not supplanted, the essen-tial role of careful clinical examination. This article presents the evidence and controversies sur-rounding the optimization of diagnostic imaging for patients with suspected BCVI. Discussion cen-ters on the increasing reliance on MDCTA for BCVI screening, also considering the relevant clinical criteria for determining the appropriate patient population to screen.

ANATOMY

An understanding of the anterior and posterior cir-culation is helpful in evaluating for BCVI. The ante-rior circulation comprises the bilateral common carotid arteries, from their origins from the brachio-cephalic artery (right) or aorta (left) to the carotid bulbs, and the internal carotid arteries (ICA) from the bulbs to their terminus. The ICA most frequently arises at the C3 to C5 vertebral level. The Bouthillier classification[33] divides the ICA into 7 segments: the cervical segment (C1) from the carotid bulb to skull base; the petrous segment (C2), coursing through the skull base in the carotid canal; the lac-erum segment (C3), a short segment extending above the petrous apex to the cavernous sinus; the cavernous segment (C4), running through the cavernous sinus; the clinoid segment (C5), ending as the ICA enters the arachnoid space; the supra-clinoid segment (C6) extending until the origin of the posterior communicating artery; and the termi-nal segment (C7) giving rise to the posterior communicating artery, anterior choroidal artery, and finally the anterior and middle cerebral arteries. With the exception of the C7 segment, only the even numbered segments have branches.

The posterior circulation comprises the bilateral vertebral arteries and the basilar artery terminating in the posterior cerebral arteries. The vertebral ar-teries generally arise from their respective subcla-vian arteries and are divided into 4 segments: the preforaminal segment (V1), from their origin until they enter the transverse foramen, generally at the C6 level; the foraminal segment (V2), continuing until the C2 level; the Atlantic or extra-dural segment (V3), extending from the C2 level until the artery passes through the dura; and the intracranial or intradural segment (V4), the intra-dural extent until they join to form the basilar. Numerous muscular and spinal branches are given off by the vertebral arteries as they ascend the neck, as well as numerous branches originating from the V4 segments and the basilar artery to supply the posterior fossa and the bilateral occip-ital lobes.

Although BCVI can occur anywhere along the length of the carotid or vertebral arteries, certain anatomic and mechanistic considerations lead to injury more frequently occurring at some sites than others.

ICA are most frequently injured just below the skull base,[34–36] within the cervical segment, and within the petrous and cavernous segments.[34–36] In vertebral artery injury, the V2 segments encom-passed by the transverse foramina of C3 to C6 are the most common sites of injury, followed by the V3 segments.[35,36]

PATHOPHYSIOLOGY

Many mechanisms for the pathophysiology of BCVI have been proposed, several sharing the basic theory of disrupting 1 or more layers of the vascular wall through stretching them beyond their physical limits.[36] The pathophysiology of BCVI seems to be a dynamic process, causing grades of injury to change with time, sometimes progress-ing in a predictable manner. Minimal intimal injury, the least severe form of BCVI described by Biffl and colleagues,[21–24] may result in disruption of the vasa vasorum, commonly progressing to dissection and intramural hematoma. Alterna-tively, minimal intimal injury may simply expose the underlying thrombogenic subendothelial ma-trix, causing thrombosis and potential distal embolization.

Blunt carotid artery injury primarily involves a direct cervical blow or cervical hyperexten-sion/hyperflexion complicated by a rotational