BLUNT CEREBROVASCULAR INJURY MANAGEMENT: IS THERE A RIGHT ANSWER?

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Introduction

Blunt cerebrovascular injury (BCVI) occurs as a result of blunt non-penetrating trauma to the head or neck causing hyperextension or hyperflexion and consequently injury to the cerebral vasculature, namely the carotid and vertebral arteries[1]. This causes intimal tear with exposure of subintimal layers to the blood flow and consequently thrombus formation, wall hematoma, dissection, pseudoaneurysm and even lumen occlusion [2]. Overall incidence of BCVI in the in-hospital trauma population is 1-2% and as high as 9% in patients with severe head injury [2].

Most patients with BCVI are asymptomatic [3]. However, its most severe manifestation is stroke-like neurologic deficits. Before widespread access to imaging modalities, BCVIs were not detected until after patients had suffered a stroke [3].

There is no consensus on optimal management of BCVI. Studies have employed watchful waiting, antiplatelet/anticoagulant use, and interventional radiological (IR) techniques without clear guidelines on what to employ, when to employ, or overall effect on patient outcomes.
In this study, we aimed to identify the incidence, management, and outcomes of BCVI at USA Health University Hospital, a level 1 trauma and stroke hospital in south Alabama.

Methods

- This was a retrospective review of the trauma registry at USA Health University Hospital from March of 2016 through December of 2021
- International Classification of Diseases (ICD) codes were used to identify patients that had BCVI on imaging
- Manual chart review was performed to confirm the presence of BCVI
- The primary endpoint identified was the presence of symptoms with imaging (CTA Head and Neck with contrast) diagnosis of BCVI.
- Additional endpoints included interventions performed—medical (anticoagulant/antiplatelet), IR, and patient outcome.

Objective

The purpose of this study was to evaluate the incidence, management, and outcomes as it relates to BCVI at a level 1 trauma and stroke center.

Results

Study Demographics Overview

Demographic	BCVI Patients	Demographic	BCVI Patients
White	60	Stabs	0
Black or African American	27	Falls	14
Asian	3	Other MOI	8
Other Race	7	Avg ISS	23.23
Male	70	Symptomatic	16
Female	27	Asymptomatic	81
MVCs	69	Deceased	6
GSWs	6	Total BCVIs	97

Table 1 displays the overall demographics of BCVI patients in this study including race, gender, mechanism of traumatic injury (MOI), the average injury severity score (ISS), the amount of symptomatic (of stroke) vs. asymptomatic patients as well as the total amount of BCVI patients in the study.

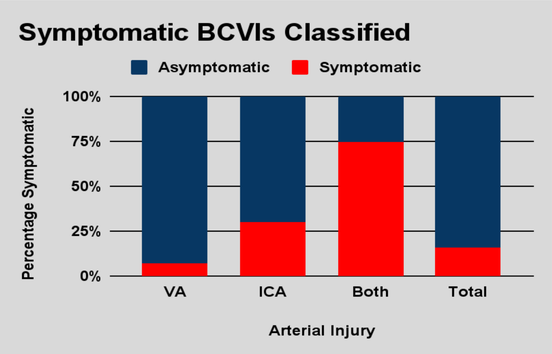

Figure 1 depicts the distribution of injury between the vertebral artery (VA), internal carotid artery (ICA) or both and the proportion of each that were symptomatic.

Results

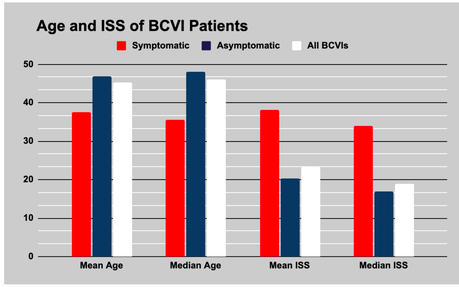

Figure 2 displays a comparison of the mean and median age and ISS of symptomatic, asymptomatic and total BCVI patients. The age range of symptomatic patients was 12-71 years old, while the age range of asymptomatic patients was 5-91 years old. The ISS range of symptomatic patients was 9-75, while the ISS range of asymptomatic patients was 4-75.

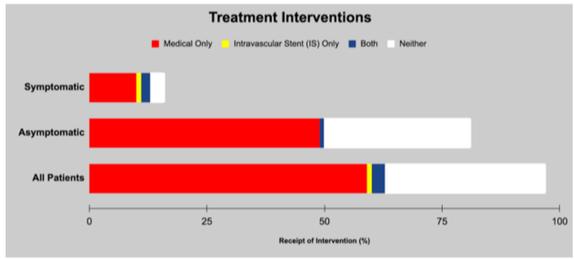

Figure 3 shows the breakdown of all interventions utilized for the management of BCVI. Approximately 61% of patients received medical management (MM) alone. 3.1% received intravascular stent (IS) in conjunction with MM. 1% underwent IS alone, and 35% did not receive any treatment.

Deceased Patients

There were 6 mortalities of patients in this study. 2 of the 6 patients were deemed symptomatic. One patient had a devastating stroke as a result of their BCVI. The remaining 5 deaths were due to etiology unrelated to the BCVI.

Discussion

We identified 144 patients whose charts were associated with ICD diagnosis pertaining to injury to cerebral vasculature, and after chart review 47 were excluded as they did not qualify as BCVI. The majority of patients were asymptomatic. 65% of patients received some sort of intervention, and those who did not either did not qualify for intervention, per the BCVI management protocol, or did not receive treatment per the discretion of the treating physician. Location of injury was predictive of symptom development. As depicted in Figure 1, injury to the ICA or both ICA and VA were more likely to result in BCVI symptoms. The determination of symptom resolution was challenging due to poor patient follow up. Furthermore, many patients with BCVI had concurrent traumatic brain injuries, making it difficult to determine whether residual symptoms were a direct result of the BCVI. Therefore, it is difficult to determine if the interventions influenced patient outcome.

Conclusions

There remains variability in BCVI management. Treatment of BCVI is crucial as the resultant neurologic deficits can have life altering effects. Stroke-like symptoms noted on patients with imaging findings of BCVI might be attributable to presence of other traumatic injuries. Larger studies are necessary to better capture the outcomes after BCVI treatment in order to establish clear management guidelines.

Bibliography

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