Vascular Complications of Penetrating Brain Injury

I. RECOMMENDATIONS

A. Standards

The available data are not sufficient to support a practice Standard for vascular complications of penetrating brain injury.

B. Guidelines

The available data are not sufficient to support a practice Guideline for this topic.

C. Options

Computed tomographic (CT) angiography and/or conventional angiography should be considered to identify a traumatic intracranial aneurysm (TICA) or arteriovenous fistula in patients with a penetrating brain injury (PBI) involving an orbitofacial or pterional injury, particularly in patients harboring an intracerebral hematoma. When a traumatic intracranial aneurysm or arteriovenous fistula is identified, surgical or endovascular management is recommended.

II. OVERVIEW

In PBI, mechanical loading of the cerebral vasculature, either by the contact forces of the projectile or the shearing forces of a pulsating temporary cavity, could cause partial or complete transection of an arterial wall. This damage can result in subarachnoid hemorrhage (SAH) and/or intracerebral and intraventricular hematomas.

Damage to an arterial wall may cause a TICA. Although rare cases of partial damage to the arterial wall have been reported (true TICA), traumatic intracranial aneurysms caused by PBI are primarily false aneurysms. A sac made of fresh or organized blood clot makes the TICA especially vulnerable to rupture and delayed traumatic intracerebral hematoma. SAH, or both, potentially causing significant mortality and morbidity. Recognizing these vascular injuries before they rupture and excluding them surgically from the cerebral circulation could significantly reduce the morbid effects of TICA rupture.

III. PROCESS (METHODOLOGY)

A MEDLINE search from January 1966 to January 2000 was conducted using the following key terms: wounds, gunshot, and brain injuries or head injuries. The search was limited to human subjects and included English language literature only. This identified 382 articles, 88 of which were rejected on the basis of irrelevant titles. An additional 33 articles were found in the bibliography of a review chapter on penetrating craniocerebral injury. This primary selection process thereby identified 327 articles for further review. Two reviewers selected 65 articles from this group for inclusion. Articles were rejected on the basis of relevance to the topic (e.g., pediatric population, non-English language, case reports, irrelevance to the project, atypical mechanisms of injury, and series of less than 10 subjects). From these 65 articles, 19 were found appropriate for inclusion in this chapter by the reviewers. In the literature search, nine articles were included for review in this section. Two articles were subsequently discarded because they did not report any actual data analyses or numerical data on vascular complications in PBI.

IV. SCIENTIFIC FOUNDATION

Traumatic Intracranial Aneurysm

Between 0.4% and 0.7% of all intracranial aneurysms are caused by trauma. Approximately 20% of traumatic aneurysms after traumatic brain injury are caused by PBI. The incidence of TICAs caused by PBI is uncertain, and the discovery of a TICA may depend on the timing of the angiogram. TICA incidence is reported to be between 3% (7 of 222 patients, when angiography was performed an average of 16 days after PBI) and 33.3% (4 of 12 patients, when angiography was performed within 48 hours after PBI) of the PBI population. A single negative angiogram does not exclude the possibility of TICA. Haddad et al. reported the delayed appearance of a new TICA 2 weeks after an initial angiogram that showed only a single TICA. Angiography remains the best diagnostic procedure to detect TICAs. However, since CT scanning has largely replaced angiography in PBI, the detection of a TICA can be increased by selective use of angiography on the basis of the presence of certain CT scan features. These include the presence of delayed intracerebral hemorrhage or subarachnoid hemorrhage, especially when associated with deterioration of neurologic function.

Peripheral branches of the middle cerebral artery and the anterior cerebral artery are more vulnerable in PBI, respectively, than the internal carotid artery. Traumatic aneurysms could heal, change in size over time, or rupture. Cerebral angiography (CT or conventional) remains the usual technique to detect intracranial aneurysms. The diagnostic value of magnetic resonance angiography when the patient is not harboring a ferromagnetic projectile remains to be elucidated. (For further details on the use of angiography in PBI, see this...
Guideline’s section, “Neuroimaging in the Management of Penetrating Brain Injury.” In one Class III article, patients with facial, orbital, and pterional injuries and those with an intracerebral hematoma or a wound profile in which the fragment crossed into another dural compartment were at greater risk of developing a TICA. Since the majority of TICAs are not true aneurysms, excluding the aneurysm by clipping may not be possible and may require trapping between clips on the parent vessel.

Subarachnoid Hemorrhage

In the PBI literature, the relative incidence of traumatic SAH is unclear. The incidence of SAH after PBI ranges from 31% to 78%, on the basis of CT scan data. These incidences are derived from studies using case series design, which may under- or overestimate the true incidence of SAH. The presence of SAH after PBI has been shown to correlate significantly with mortality in three Class III studies1,2 (for additional information, see the section on prognosis in these Guidelines).

Vasospasm

Patients with SAH may exhibit cerebral vasospasm on transcranial Doppler. However, no differences in outcome (3-month Glasgow Outcome Scale) were found in PBI patients with or without vasospasm.9 No specific evidence shows a benefit from treatment of vasospasm in these patients with PBI, although it is common clinical practice for patients with true aneurysmal SAH.

V. SUMMARY

In PBI, physicians should maintain an index of suspicion for the presence of vascular injury, traumatic SAH, and vasospasm. When these are detected, therapeutic measures analogous to those used outside the setting of trauma are indicated. However, outcome data to judge the efficacy of these interventions are limited and do not support recommendations stronger than treatment options.

VI. KEY ISSUES FOR FUTURE INVESTIGATION

The incidence, diagnosis, prophylaxis, and treatment of posttraumatic vasospasm need further study. Research on the interaction of injury mechanisms (e.g., ballistics) and the mechanism of traumatic aneurysm formation is needed.

Techniques to screen for traumatic aneurysms would be helpful. Investigation of vasospasm, correlating angiography and transcranial Doppler (TCD), to determine inci-

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dence of vasospasm in the presence of associated pathology may be useful.

Randomized, controlled trials of the therapeutic benefit of pharmaceutical agents, such as calcium channel blockers and/or hypertensive, hypervolemic, and hyperdynamic therapy, should be performed. A prospective study should be considered to determine the true incidence and timing of cerebral aneurysm development after PBI.

REFERENCES


