Blasts are the most prevalent mechanism of injury during war times, concomitant with the increase in terrorist activity and their terrorists’ preference for explosive devices over chemical, biological, and radiation means of attacks. Recent figures of combat casualties during the War on Terrorism in Afghanistan and Iraq estimate a total of 17,501 casualties with 303 soldiers who “died of wounds” (deaths after arriving to a medical facility) and 1266 soldiers who were “killed in action” (deaths before reaching a medical facility). Comparative analyses against World War II and Vietnam suggest a 6% decrease in the killed-in-action rate and a 9 and 6% decrease in case fatality rate, respectively. However, there has been an increase in the estimated percentage of deaths after reaching a medical facility, indicating patients being treated earlier with a greater burden of injury. In addition, TBI cases have increased to an estimated 22% of military admissions in Germany during combat in Iraq and Afghanistan, compared with the 12–14% of cases during the Vietnam War. The direct effect of traumatic blast shock waves becomes more apparent as morbidity remains high, including a higher incidence of recognized postinjury vasospasm and as many as 25% of blast-injured patients die.

Several reports have observed patients with blast-induced trauma, especially seen during war times, presenting and responding in various ways compared with most civilian craniofacial trauma and traumatic brain-injured patients. Despite the lack of longitudinal studies, growing evidence demonstrates distinct features of blast-induced TBI as evidenced clinically and in the laboratory, and this may lend evidence to delayed neurological deterioration after blast-induced cerebrovascular trauma, which is not well understood. Ling et al. suggested blast-induced TBI as a separate classification because it shares hemorrhage and cerebral edema (features of brain-tissue deformation seen in penetrating TBI) and vascular and white matter injury (features of closed-head TBI), but that diffuse edema and hyperemia are quite often seen early along side SAH and delayed neurological deterioration as commonly unique features of blast-induced TBI. Furthermore, the military has reported performing fewer decompressive cranietomies in cases of penetrating or closed head–induced TBI than in cases involving this blast-induced TBI presentation. Laboratory studies have demonstrated unique features of persistent, slowing electroencephalographic activity after missile-induced high-impact injuries and degeneration in white matter tracts without gross associated cellular loss, features different from those of direct contusion or fluid impact injury models. In addition to the knowledge that severe TBI disrupts cerebral autoregulation and the growing evidence that cerebral vasculature is a conduit for blast-impact waves, it is not surprising that in blast-induced trauma, especially seen during war times, presenting and responding in various ways compared with most civilian craniofacial trauma and traumatic brain-injured patients.
induced penetrating and nonpenetrating TBI a significant incidence of traumatic cerebral vasospasm and delayed neurological deterioration has been demonstrated.\(^2\)

Understanding the complex nature of blast-induced cerebrovascular trauma may help us identify vascular compromise in the acute and delayed setting. During the Iran-Iraq conflict, Amirjamshidi et al.\(^1\) reported the need for early angiography after evaluating 31 cases of traumatic cerebral aneurysms and 3 associated CCFs, 1 of which was followed for spontaneous resolution and the other 2 of which were trapped and embolized for occlusion.\(^2\) Here, we describe the cases of 2 patients with blast-induced craniofacial injuries and delayed-presentation traumatic CCF.

**Case Reports**

**Case 1**

*History.* This 36-year-old man presented with an improvised explosive device blast injury to his face and jaw. The result was a comminuted mandibular fracture and soft-tissue neck injury at the level of the cricoid cartilage. In the combat theater he underwent surgical exploration bilaterally of the common cervical carotid artery bifurcation and open reduction and rigid internal fixation to stabilize the mandibular fracture (Fig. 1A). No external injury to the carotid arteries was appreciated (Fig. 2A). On arrival, his GCS score was 11 (combined scores of E4, V1, and M6) and neurological examination unremarkable.

*Examination.* Two weeks later, while at the National Naval Medical Center, serial TCD ultrasonography demonstrated significant abnormalities, including reduced right middle cerebral artery velocities and reversal of flow in the right ophthalmic artery. This steal phenomenon was likely indicative of a CCF. The patient had no ophthalmological signs of orbital hypertension, and no audible bruit. A CT angiogram confirmed this suspicion with an additional cervical pre-petrous right ICA dissection and Type A high-flow separate cavernous-seg-
Delayed detections of CCFs associated with craniofacial trauma

Fig. 2. Case 1. Endovascular treatment and follow-up of the complex blast-induced CCF. A: Mandibular ramus fixator in place with an associated distal ICA subintimal dissection, missed during open neck exploration. B: Endovascular vessel reconstructed with a combination of a self-expanding stent for the cervical dissection and a stent/coil buttress occlusion of the direct CCF with parent vessel preservation. C: Twelve-week follow-up angiograms demonstrating obliteration of the CCF, parent vessel preservation, and endovascular reconstruction.

The cervical dissection was treated with a self-expanding stent, the venous pouch of the CCF was treated with coil occlusion, and stent reconstruction was performed to preserve the cavernous-carotid artery. After endovascular treatment, transient partial cranial nerve III and VI palsies were noted. The patient’s condition remained stable and his GCS score continued to be 11.

Postoperative Course. Two weeks after surgery, the patient returned to the neurointerventional operative suite for repeated angiography. Transvenous inferior petrosal microcatheterization was used to re-coil the residual persistent CCF, with drainage via the pterygoid venous plexus as well as retrograde filling via clival venous plexus into the jugular bulb. Completion angiography demonstrated resolution of the dissection with a widely patent lumen (Fig. 2B) and near obliteration of the fistula with stasis of flow in the cavernous sinus. At 12-week follow-up, angiography revealed complete obliteration of the fistula and the patient’s cranial nerve deficits had resolved (Fig. 2C).

Case 2

This 25-year-old male platoon leader, whose vehicle was struck by a rocket-propelled grenade (RPG), sustained a depressed frontal sinus fracture and a complex flail jaw fracture involving the maxilla and mandible. He underwent an emergency cricothyroidotomy and a subsequent elevation of the frontal sinus fracture with epidural hematoma evacuation after the initial head CT scans. His mandible and zygomatic arch were stabilized and fixed with internal plates and screws. Although the patient was initially neurologically intact, a delayed right gaze diplopia developed 6 weeks after injury, and the patient began to notice a marked pulsatile machine-like bruit. There was no evidence of chemosis or exophthalmos. Color CT angiography and angiography at that time revealed a giant pseudoaneurysm at the proximal portion of the cavernous ICA segment, with both cortical venous reflux into the postero-fossa veins via the superior petrosal vein and superior orbital vein engorgement, consistent with venous

Fig. 3. Case 2. A delayed CCF appearance after blast injury to the head. Color CT angiograms (A–D) and repeat angiogram (E) 6 weeks postinjury revealing a giant ruptured pseudoaneurysm with marked filling of the cavernous sinus, superior petrosal sinus, cerebellar veins, as well as filling anteriorly to the superior ophthalmic vein, inferior ophthalmic vein, and pterygoid veins (gold arrows, A). F: One-month postoperative angiogram demonstrated no filling of the aneurysm, maintenance of ICA patency, and complete resolution of the CCF.
hypertension (Fig. 3A–E). Selective catheterization dem-onstrated marked filling of the cavernous sinus, superior petrosal sinus, cerebellar veins, and filling anteriorly to the superior ophthalmic vein, inferior ophthalmic vein, and pterygoid veins. Rapid filling was noted through the CCF, with filling of the cavernous sinus, superior inferior ophthalmic veins, and predominately into the posterior fossa and veins of the lateral mesencephalic region of the cerebellum and brainstem. Transarterial coil embolization was performed using 210 cm of Matrix G coils to occlude the pseudoaneurysm and prevent filling of the CCF. One month follow-up angiography demonstrated no fill-ing of the pseudoaneurysm, maintenance of ICA patency, and complete resolution of the CCF (Fig. 3F). The cranial nerve VI palsy had completely resolved, and the officer returned to full duty at 6 months without limitations.

Discussion

These cases highlight the need for a high degree of suspicion associated with neurovascular injuries in the presence of blast-induced maxillofacial injuries. The delayed detection of CCFs becomes more challenging in the absence of ophthalmic findings. The use of TCD ultrasonography and CT angiography allowed for the identification of the fistula, and endovascular techniques permitted successful safe treatment. A high likelihood for such injuries should exist in the presence of facial fractures such as mandibular, maxillary, and skull base injuries as in these cases. The absence of ophthalmic features indicates that the fistula is draining through intracranial or pterygoid venous channels, and thus, there is the risk of cortical venous drainage. Such venous drainage may result in delayed intracranial hemorrhage or intraoral bleeding.

Both of the CCFs in the present cases can be classified as Barrow Type A high-flow fistulas. Type A fistulas are caused by a direct tear of the cavernous-carotid artery. This can result from traumatic skull base injuries due to the relative high mobility of the cavernous-carotid artery compared with the points of carotid artery fixation in the petrous and clinoid segments. Generally these lesions have a low rate of spontaneous resolution and require definitive treatment, of which endovascular options are preferable. In trauma CCFs are infrequently associated with concomitant traumatic cerebral aneurysmal rupture, which is more commonly seen in cases of spontaneous Type A fistulas. In contrast, Barrow Type B–D fistulas, which generally exhibit dural derivation, frequently resolve spontaneously. Interestingly, in both cases reported here the patients did not initially present with typical suspicious findings of classically described traumatically induced CCFs, including the lack of pulsatile exophthal-mus, penetrating trauma, SAH, stroke, or chemosis. Evi-dence of craniofacial trauma and its association with a pseudoaneurysm in both cases suggested blast-induced CCFs. The blast-wave effect on the mobile segment of the cavernous-carotid artery seems a likely cause for a direct tear and resulting high-flow fistula.

A blast wave is described as having 2 important char-acteristics: 1) a high-pressure shock wave, along with 2) a subsequent inflow of air. This combination allows for high-pressure differences, especially deleterious at air-fluid interfaces. Blast injuries have been classified into 4 categories according to the origin of the blast effect. Primary blast injuries include direct effects of pressure often seen in blunt (nonpenetrating) injuries. Secondary blast injuries include penetrating and projectile fragments entering the body, producing injury alongside blast pressure. Tertiary blast injuries are based on structural collapse of building or structures with people in or on them, allowing these persons to be thrown by blast winds and also encompassing in some respects the effects of primary and secondary blast injuries as well. Quaternary blast injuries include exposure to toxic inhalants producing burns and asphyxia. Blast-induced cerebrovascular injury, seen in the present cases, may have evolved by frank arterial disruption that resulted in a brief subarachnoid hemorrhage due to tamponade or by partial arterial wall disruption, including the subadventitial layers, while allowing for a prolonged series of associated unregulated vasculogenic responses in attempts for arterial recovery. Despite the number of patients with blast-induced TBI and facial injuries, the rate of CCF formation remains low. In the series of 187 patients undergoing cerebral angiography at the National Naval Medical Center and Walter Reed Army Medical Center, only 6 total fistulas were identified: 3 cavernous, 1 orbital, 1 vertebral, and 1 scalp.

The current military experience has witnessed earlier neurosurgical intervention in theater with improved airlift emergency triaging and early access to state-side neurointerventional treatments. Our indications for cerebral angiography have expanded to include maxillofacial injuries and penetrating neck injuries. Screening with CT angiography may be useful in patients without significant metallic artifact obliterating the imaging quality. How-ev er, the timing of the contrast bolus, the artifact from bone, metal, and foreign debris, as well as the technical aspects of workstation 3D reconstructions, limit its use. Neck exploration as demonstrated in Case 1 does little to identify a subintimal dissection. Because this is most likely to occur between the mobile segment of the artery, its entrance to the petrous bone, and contact with the sty-loid process, extraluminal exploration is ineffective. Early out-of-theater angiography and continued repeat imag-ing becomes essential in those thought to have sustained blast-wave propagation (impact) within the cerebrovascu-lar pathways and/or delayed neurological compromise.

Here, we observed 2 patients with delayed identification for CCF. The use of TCD ultrasonography is an essen-tial part of our screening regimen and has helped us expand our evaluation of wartime patients with maxillofacial trauma. We speculate here that blast waves and blunt forces, with the resulting collision and impact, may result in cerebral vasculature injury in the mobile segments of the vessel between points of fixation. Additional theories include the brachiocephalic vasculature as a conduit of blast-wave propagation as a pulse wave from the heart to the cranial cavity. Greater understanding of this blast-wave propagation within the cerebral vascula-ture becomes even more important in light of recent work from our group in which the incidence of cerebrovascular injury was as high as 27% in this war setting, almost 10-
fold higher than the previously reported incidence. The 2 cases here highlight the need for a high degree of clinical suspicion for cerebrovascular injury in patients with maxillofacial fractures caused by blast-induced trauma. Further studies both in the clinical and laboratory setting to investigate the pathophysiology behind these blast-induced neurovascular injuries may help identify the cause and ultimate prevention of such injuries.

Conclusions

Blast-induced maxillofacial injuries and TBI may develop delayed CCFs. Endovascular approaches that involve coil occlusion and stent reconstruction appear well tolerated and are an effective measure for blast-induced CCFs and traumatic dissections. Neuroendovascular techniques provide a definitive means of treatment for blast-induced cerebrovascular trauma as demonstrated in these 2 cases. The use of TCD screening combined with CT angiography allowed the detection of these lesions and should be used in patients with craniofacial trauma when neurovascular injuries may be located beneath the disrupted contour of the craniofacial skeleton.

Disclosure

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