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Neuroendovascular Surgery Applications in Craniocervical Trauma

Michael G. Kim, MD,[1] Jared B. Cooper, MD,[1] Namhee Kim, BA,[2] Christian A. Bowers, MD,[3] Kartik Prabhakaran, MD, MHS,[4] Rolla Nuoman, MD,[5] Krishna Amuluru, MD,[6] Chirag D. Gandhi, MD,[1] Fawaz Al-Mufti, MD.[1,7,*]

[1]Department of Neurosurgery, Westchester Medical Center at New York Medical College, Valhalla, NY, USA

[2]New York Medical College, Valhalla, NY, USA

[3]Department of Neurosurgery, University of New Mexico, Albuquerque, NM, USA

[4]Department of Surgery, Division of Trauma and Acute Care Surgery, Westchester Medical Center, Valhalla, NY, USA

[5]Department of Neurology, Maria Fareri Children's Hospital - Westchester Medical Center at New York Medical College, Valhalla, NY, USA

[6]Goodman Campbell Brain and Spine, Indianapolis, ID, USA

[7]Department of Neurology, Westchester Medical Center at New York Medical College, Valhalla, NY, USA

Abstract

Purpose— Cerebrovascular injuries after blunt or penetrating trauma to the head and neck often lead to local hemorrhage and stroke. A large number of manifestations can be seen including carotid or vertebral artery dissection, pseudoaneurysm, occlusion, transection, arteriovenous fistula, carotid cavernous fistula, epistaxis, venous sinus thrombosis, and subdural hematoma.

Methods— A selective review of the literature reporting various neuroendovascular surgery techniques in craniocervical trauma was performed. The authors conducted a PubMed search from 1989-2020 including the terms: endovascular, trauma, dissection, blunt cerebrovascular injury, pseudoaneurysm, occlusion, transection, vasospasm, carotid-cavernous fistula, arteriovenous fistula, epistaxis, cerebral venous sinus thrombosis, subdural hematoma, and middle meningeal artery embolization.

Results— An increasing number of neuroendovascular procedures are currently available to treat these traumatic injuries. Lesions can be embolized using coils, liquid embolics (onyx or n-butyl cyanoacrylate), and polyvinyl alcohol particles. Stent placement using uncovered, covered, and overlapping stents; mechanical thrombectomy using stent-retrievers or aspiration catheters; and balloon occlusion tests and supraselective angiography to assess the safety of therapeutic arterial occlusion are additional options depending upon the underlying lesion.

Conclusion— Neuroendovascular techniques can often treat conditions where surgical options are otherwise limited. However, there is limited data comparing neuroendovascular and surgical techniques in the setting of trauma.

Keywords— Endovascular, neurointerventional, trauma, craniocervical, angiography.

INTRODUCTION

Traumatic injuries to the head and neck can result in adverse neurological outcomes due to the critical neurovascular anatomy.[1] Neuroendovascular techniques have traditionally been limited to the treatment of traumatic injuries of the carotid and vertebral arteries when conservative management has failed. However, advancements in technology and improvements in the safety profile and complication rates have allowed the field to expand greatly over the past several decades.

Injuries such as blunt cerebrovascular injury (BCVI), dissection, pseudoaneurysm, vessel occlusion, carotid cavernous fistula (CCF), arteriovenous fistula (AVF), cerebral venous sinus thrombosis (CVST), epistaxis, and subdural hematomas (SDH) can be difficult to diagnose, particularly in the polytrauma patient with a depressed neurologic status. Furthermore, many of these injuries can develop over time and may not be present on initial evaluation. The detection of these injuries has increased though as comprehensive screening protocols have been implemented. For stable patients, initial

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***Corresponding Author:** Fawaz Al-Mufti, 100 Woods Road, Macy Pavilion, Suite 1332, Valhalla, NY 10595, Phone: 914-493-5216, Fax: 914-493-2424, Email: fawazalmufti@outlook.com

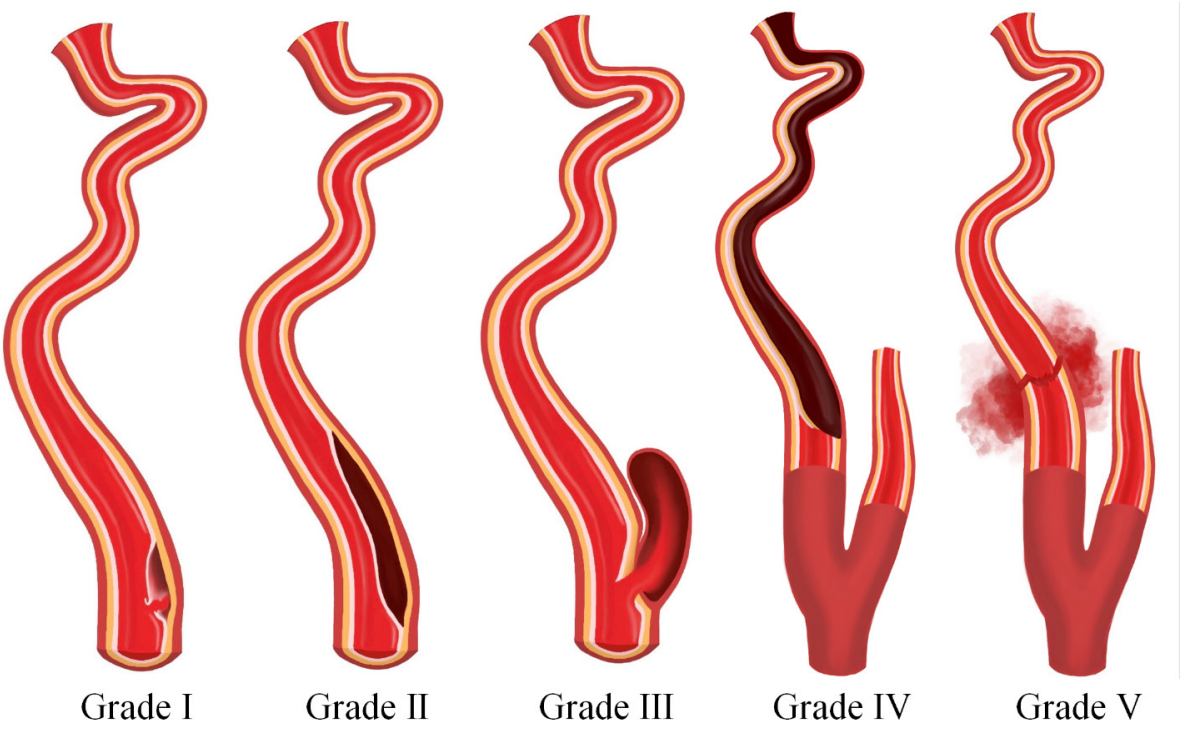

FIGURE 1: An illustration depicting the five grades of blunt cerebrovascular injury.

screening with non-invasive imaging, such as computed tomography angiography (CTA) and magnetic resonance angiography (MRA), followed by subsequent selective use of digital subtraction angiography (DSA) has been increasingly used as the management algorithm of choice.[2] However, DSA remains the gold standard for diagnosing traumatic carotid and vertebral artery injury. It provides superior visualization of both arterial anatomy and real-time blood flow, allowing the physician to visualize both obvious and subtle flow-altering injuries. These injuries can also be treated during the same procedure, offering both diagnostic and therapeutic benefits. Furthermore, the physician can examine the collateral circulation in the region supplied by traumatized vessel, which is critical in determining the optimal treatment modality.

Endovascular techniques have evolved from an adjunct to surgical treatment to now a viable alternative for initial management. They may help to reduce the morbidity and mortality rates involved in treatment of these injuries.[3] However, there are no randomized clinical trials comparing surgical and endovascular treatments of traumatic head and neck injuries, and therefore a consensus in the diagnosis and management of these injuries still needs to be established. We provide here an updated comprehensive nonsystematic review of the role of endovascular treatments in the management of traumatic cerebrovascular injuries (Table 1).

METHODS

The authors conducted a PubMed search from 1989-2020 including the terms: endovascular, trauma, dissection, blunt cerebrovascular injury, pseudoaneurysm, occlusion, transection, vasospasm, carotid-cavernous fistula, arteriovenous fistula, epistaxis, cerebral venous sinus thrombosis, subdural hematoma, and middle meningeal artery embolization.

Blunt Cerebrovascular Injury

BCVI can occur from high-energy nonpenetrating blunt force trauma to the neck and is often categorized into 5 grades according to the scheme proposed by Biffl et al.[4,5] Grade I has intimal irregularity with <25% vessel stenosis, grade II has a dissection with >25% vessel stenosis, grade III involves formation of a pseudoaneurysm, grade IV has vessel occlusion, and grade V involves transection of the vessel with active extravasation (Figure 1). BCVI is present in approximately 1-2% of blunt trauma patients and in 2.4% of trauma patients that require inpatient care over 24 hours.[6–8] The detection of these injuries has significantly risen from 0.1% to now up to 3% of polytrauma patients due to advances and availability in noninvasive imaging such as CTA and MRA as well as the development of improved screening protocols.[9–11] A multislice CTA with an 8 or greater multi-detector has been recommended as an alternative screening method to DSA with similar rates of detection by both the Western Trauma Association (WTA) and the Eastern Association for the Surgery of Trauma (EAST) in their BCVI guidelines.[12,13]

The signs and symptoms of BCVI may not always be obvious in complex trauma patients with limited neurological exams and BCVI historically carries a significant risk of morbidity and mortality with rates of 48-56% and 23-30% respectively.[6,12] One series found that patients with traumatic vertebral artery dissections had higher rates of in-hospital stroke and longer lengths of intensive care unit and hospital stay.[14] However, comprehensive screening protocols have resulted in earlier initiation of appropriate therapies in BCVI patients, leading to a significant reduction in stroke risk.[1] Studies performed by Scott et al. at their level 1 trauma center from 2003-2013 revealed a 1-1.7% stroke rate in grade I and II BCVI and 7% in grade III and IV BCVI.[15–18]

Grade I-II: Dissection

The role for neuroendovascular techniques is limited in grade 1 and 2 BCVI. The mainstay of treatment for grade I and II BCVI is an antithrombotic agent such as aspirin or heparin and therefore endovascular treatments may take a role only when these low grade injuries progress to higher grades.[12,13] Furthermore, the updated 2020 EAST BCVI guidelines now recommend against the use of routine stent placement as an adjunct to antithrombotic therapy in adult patients with grade II BCVI. Scott et al.[15] found in their cohort of 100 patients with 117 grade I and II carotid artery BCVI that 19 patients had worsening of injury on follow up imaging. 17 of those 19 patients developed pseudoaneurysms, 3 of which required endovascular stent placement for radiographic progression.[15] They had a similar series of 120 patients with 152 grade I and II vertebral artery BCVI. Of these, 9 patients had worsening injury with 7 developing pseudoaneurysms. However, none of these patients required endovascular treatment.[16]

Grade III: Pseudoaneurysm

Traumatic pseudoaneurysms arise from a disruption of the internal elastic lamina and eventually all layers of the arterial wall, resulting in the formation of a false lumen within the arterial wall. As this false lumen expands, it can compress the true lumen of the artery, resulting in stenosis and possible infarcts from ischemic or embolic events. Furthermore, the pseudoaneurysm can rupture, causing extravasation into the surrounding tissue. Therefore, patients can present with a variety of symptoms including Horner's syndrome, neck pain, pulsatile neck or scalp mass, epistaxis, dysphonia, dysphagia, upper airway compromise, hemiparesis, or even coma.[19,20]

While both the WTA and EAST guidelines agree that screening can be performed using a CTA, their guidelines for treatment differ. They both state that initial treatment should be with an antiplatelet agent or anticoagulation. However, the timing of antiplatelet or anticoagulation is frequently complicated and often delayed due to the polytraumatic injuries frequently associated with the high force mechanism injuries required to produce grade III BCVI. The WTA guidelines states that stent placement should be considered in patients with severe luminal narrowing or expanding pseudoaneurysms after a repeat CTA 7-10 days after initial diagnosis is obtained.[12] The 2020 EAST guidelines now state that routine stent placement should not be performed as an adjunct to antithrombotic therapy in grade III injuries, but that there may be select cases such as an enlarging pseudoaneurysm in which stent placement would be appropriate and potentially beneficial.[11] Neither set of guidelines give recommendations on what treatment modality should be performed. Given the lack of guidelines on the optimal technique and the complexity of polytrauma patients, treatment depends on the associated injuries and is often individualized. Complete occlusion of the pseudoaneurysm with preservation of the parent vessel is often possible and good results have been obtained using coil embolization, coil embolization with stent placement, stent angioplasty, and stent placement with covered stents or flow diverters.[20–26] Caution must be taken as stent placement requires dual antiplatelet therapy and the risk of bleeding from dual antiplatelet agents must be weighed against the risk of complications from untreated pseudoaneurysms. In cases where therapeutic occlusion of the parent vessel is necessary, a balloon test occlusion can be performed under local anesthesia to assess collateral flow both clinically and angiographically prior to therapeutic occlusion of the vessel.

Scott et al.[18] found in their series of 23 patients with grade III vertebral artery injuries that 9 patients had stable BCVI, 10 resolved, 3 improved, and 1 worsened. All except 1 patient were treated with antiplatelet therapy or anticoagulation. 1 patient developed infarcts that were felt to be due to other concurrent BCVI in the anterior circulation. None of the patients required endovascular therapy.[18] They also found in their series of 44 patients with 53 grade III carotid artery BCVI that 8 patients required endovascular treatment, 7 with stents and 1 with coil embolization. Final follow-up imaging showed that 6 resolved, 28 were stable with 8 felt to be due to the effects of treatment, and 12 worsened, which had prompted 5 patients to get treated. 3 patients developed infarcts, but did not require endovascular treatment.[17] The authors conclude that the posttraumatic infarction rate after high grade BCVI may be overestimated in the literature.

Grade IV: Occlusion

The carotid and vertebral arteries may become occluded after traumatic injury primarily due to vessel dissection or secondarily to extrinsic compression from cervical spine fractures. Occlusions caused by extrinsic compression rarely require neuroendovascular techniques, but may require therapeutic occlusion of the proximal vessel if reduction of the fracture will result in further injury to the vessel.[19] Traumatic occlusions caused by dissections are treated based on the presence of ischemic stroke and adequacy of the collateral circulation. The current WTA and EAST guidelines recommend that all patients with carotid or vertebral artery occlusions should be treated with antiplatelet agents or anticoagulation to prevent further propagation of the intraluminal thrombus and potential embolic events. Furthermore, in patients with early neurologic deficits and accessible lesions, operative or interventional repair should be considered to restore flow as long as they have not suffered a completed cerebral infarct.[12,13] Endovascular options include mechanical thrombolysis, mechanical thrombectomy, or stent angioplasty.[19,27]

Scott et al.[18] found in their series of 42 patients with 43 grade IV vertebral artery BCVI that at latest follow up, 28 had stable occlusions, 13 improved with asymptomatic recanalization, and 2 resolved completely.[18] A total of 11 patients had undergone coil embolization, but the authors stated these procedures were done early on and the practice was subsequently discontinued. 3 patients, 1 of which had bilateral vertebral artery occlusions, had infarcts that they felt were due to the BCVI with a mortality of 100%. All patients except one were treated with antiplatelet therapy or anticoagulation. In their series of 8 patients with 8 grade IV carotid artery BCVI, 3 died upon or shortly after presentation, 3 had stable occlusions, and 2 improved with vessel recanalization. The 5 surviving patients were treated medically and 1 had an asymptomatic stroke. The authors believe that all strokes caused by grade IV BCVI were likely present upon hospital admission. Furthermore, they concluded that follow-up and treatment protocols

may have to be amended because they had no delayed infarcts in the post-injury period and the majority of occlusions were either stable or improved on follow-up.

Grade V: Transection

Transection with active extravasation of the extracranial carotid or vertebral arteries after trauma is associated with high rates of morbidity and mortality and mandates immediate attempts at control due to the large amounts of blood loss.[19] The current WTA guidelines recommend urgent surgical repair for accessible lesions and endovascular techniques for inaccessible injuries.[12] However, these guidelines do not make any recommendations on which technique should be employed. Zone 2 injuries to the carotid artery in the neck are usually easily surgically accessible. Carotid artery transections in zones 1 and 3 and vertebral artery transections are more difficult to access due to the extensive exposure needed for proximal and distal control and are therefore more amenable to endovascular treatments.[19] Other treatment options include therapeutic occlusion of the parent artery to prevent blood flow and they include therapeutic occlusion of the vessel proximal to the tear with or without distal occlusion using coils or liquid embolization materials. Temporary balloon occlusion testing can be used in this setting prior to performing the therapeutic occlusion. Reconstruction of the injured vessel can be performed by stent placement. However, the placement of stents requires the use of antiplatelet medications, which needs to be weighed against the potential of bleeding complications or progression of hemorrhage, especially in the setting of active extravasation.

Intracranial Dissection

Intracranial dissections represent an uncommon and likely underdiagnosed phenomenon because of the inherent difficulty in visualizing the subtle radiographic signs in pathologic small intracranial arteries.[28] In the absence of major randomized control trials, there remains a dearth of management guidelines with no widespread consensus, particularly as patients can present either with ischemia or hemorrhage. Intracranial vessels lack an external elastic lamina and have little adventitial tissue, making these vessels prone to subadventitial dissections and therefore subarachnoid hemorrhage.[28–30] Several small case series of intracranial dissections caused by all etiologies have shown that anterior circulation dissections present in approximately 72.7-88% of cases with ischemia and 20-65% with subarachnoid hemorrhage while posterior circulation dissections present in approximately 26-62% of cases with ischemia and up to 70% with subarachnoid hemorrhage.[28] The majority of intracranial dissections are spontaneous in nature with a small minority caused by trauma. One series found that only seven of 61 patients (11.5%) with intracranial dissections were due to trauma and that all seven were located in the ICAs.[31] In the pediatric population, intracranial dissections most often involve the anterior circulation.[32] The diagnosis of these injuries can be difficult as the clinical presentation can vary widely and range from mild findings such as facial hypoesthesia or dysmetria to severe deficits such as aphasia, hemiplegia, and coma.[19] Furthermore, the majority of patients are asymptomatic in the acute phase.

The optimal treatment is currently unknown and the dissected vessels can present with varying radiographic findings from segmental stenosis and occlusion to fusiform or saccular aneurysmal dilatation.[30] Therefore, treatment of traumatic intracranial dissection is frequently individualized based on neurologic function, presence of collateral flow, and other traumatic injuries. For example, dissections with non-flow limiting stenosis or occlusions with adequate collateral circulation can often be managed medically while those with flow limiting stenosis and inadequate collateral circulation may be candidates for endovascular therapy.[19] Asymptomatic patients with stable dissections are typically treated with anticoagulation or anti-platelet agents.[19,28] However, medical therapy alone should be used cautiously given the risk of subarachnoid hemorrhage.

In patients presenting with ischemia, endovascular therapy is often limited to patients who have worsening symptoms or progression of the dissection despite optimal medical management.[28,30] Reconstruction of the vessel can be performed using flow diversion or stents to separate the false and true lumens. This method leads to a controlled thrombosis of the dissection while preserving vessel patency and therefore physiologic cerebral blood flow.[33] Overlapping stents and flow diversion without coil embolization have also been shown to be a safe option for dissecting aneurysms with a lower rate for rupture because no coils are inserted into the thin-walled aneurysm.[28] Deconstructive techniques involve vessel takedown with embolization material or detachable coils. Additionally, emergent revascularization by mechanical or chemical thrombectomy can be considered in patients who become acutely symptomatic from embolization, similar to acute ischemic stroke therapy. This maneuver can be performed by itself or in conjunction with the aforementioned techniques.[28]

Patients presenting with subarachnoid hemorrhage from an intracranial dissection are treated much more aggressively than those presenting with ischemia because up to 40% of patients rebleed within the first few days with a mortality rates of up to 50%.[28–30] The endovascular treatment options include the techniques mentioned previously and are tailored to each patient based on vessel morphology with an ultimate goal of reducing blood flow to the dissected region. Deconstructive techniques have been shown to have both rebleeding and infarction rates of <33% while reconstructive techniques have reported rebleeding rates of <50% with infarction rates of <14%.[28] Other novel approaches such as the use of Onyx (Medtronic, Minneapolis, MN, USA), a liquid embolic system comprised of an ethylene vinyl alcohol copolymer, to occlude a dissection and its associated pseudoaneurysm have been reported.[34]

Post-Traumatic Vasospasm

Secondary injury is a cause of substantial morbidity and mortality following traumatic brain injury (TBI), and can be aggravated by hypotension and hypoxia. Recently, arterial vasospasm has become a focus of interest as a potential contributor to secondary brain injury via delayed ischemia (Figure 2). Post-traumatic vasospasm is believed to result

from circulation of spasmogenic and neuroinflammatory substances generated from lysis of subarachnoid blood products following trauma.[35] In contrast to aneurysmal subarachnoid hemorrhage, surveillance for post-traumatic vasospasm is not routinely performed and its true incidence therefore is difficult to determine. As imaging technology has improved, rates of post-traumatic vasospasm detection in TBI patients range from 27% to 63%.[36,37] Diagnosis of post-traumatic vasospasm is often established using CTA/CT perfusion and DSA. Additionally, transcranial Doppler ultrasonography has been instrumental in guiding early management of TBI patients by monitoring the mean blood velocity, which acts as a surrogate for cerebral perfusion pressure.[38] Certain clinical features such as fever, low Glasgow Coma Scale score, number of cerebral lobes affected by the traumatic injury, high Injury Severity Score, and presence of an associated pseudoaneurysm or hemorrhage have been shown to correlate with an increased risk of developing post-traumatic vasospasm.[36,39–43] Currently, nimodipine is the most efficacious and widely used medication for post-traumatic vasospasm, however, interventional techniques such as balloon angioplasty and intra-arterial vasodilator administration have been used as well.[44,45] As is the case with many neurovascular injuries, no consensus guidelines exist on which patients should be screened for post-traumatic vasospasm and further study should be performed to best determine the optimal management for these patients, especially given the wide spectrum of clinical effects that vasospasm can have.

Carotid-Cavernous Fistula

The formation of a CCF can occur following cranial trauma and is typically associated with complex fractures of the skull base (Figure 3). Following injury, arterialization of the venous outflow from the cavernous sinus may result in the development of a myriad of symptoms including visual deterioration, ophthalmoplegia, diplopia, headache, conjunctival chemosis, proptosis, pulsatile tinnitus, and ocular bruit.[19,46–49] Signs and symptoms may develop days to weeks following the initial traumatic event.[19]

CCFs may be direct, in which there is a direct connection between the ICA and cavernous sinus, or indirect (dural), in which there are communications between the cavernous sinus and meningeal arterial branches.[19] Direct fistulas are high flow with rapid development of venous congestion and symptoms and are often associated with trauma, accounting for greater than 70% of cases.[46] Indirect fistulas are low flow and their etiology is often unknown.[19]

Initial evaluation initially includes standard ocular tonometry, pneumotonometry, ultrasonography, color Doppler imaging, CTA, and/or MRA.[48] If suggested by non-invasive modalities, diagnosis is confirmed by DSA. DSA allows for classification of CCFs as well as treatment planning by delineation of the arterial supply and identification of the fistulous point.[19,46,47,50]

Because of their low flow status, indirect CCFs may be managed conservatively by observation, with spontaneous closure seen in as many as 70% of cases.[51–53] Medical therapy with intraocular pressure-lowering agents, intermittent compression of the ipsilateral ICA or superior ophthalmic vein, or radiosurgery may be added as adjuvant therapy, but these are rarely used.[48] Almost all CCFs can be treated by endovascular methods including transarterial embolization, transvenous embolization, or therapeutic occlusion of vessel alone or in combination. Indications for intervention include refractory elevation in intraocular pressure, severe diplopia, proptosis, optic neuropathy, retinal ischemia, severe ocular bruit, and cortical venous drainage. With endovascular techniques, cure rates approach 100% with low rates of complications and mortality with the potential to restore oculomotor function and vision if early treatment is pursued.[48,49,54] Embolization may be achieved using detachable coils, balloons, liquid embolic agents, and/or flow diverting stents with the goal of therapy to occlude the fistulous point. Transarterial embolization is preferred, however if the fistulous point cannot be accessed via arterial catheterization, a transvenous approach may be attempted utilizing the inferior petrosal sinus or superior ophthalmic vein.[49] For cases with ICA dissections, flow-diverting stents serve as a potential adjuvant therapy to coil embolization by way of endoluminal reconstruction.[48,55,56] A disadvantage of stent placement is the need for dual antiplatelet therapy. As a last resort, therapeutic occlusion of the parent vessel may be considered if the patient has adequate collateral flow.[46]

Other Intracranial Arteriovenous Fistula

Intracranial AVF other than CCF are exceedingly rare and can result from trauma to the meningeal branches of the external carotid artery (ECA) or as the sequelae of progressive stenosis or occlusion of a dural venous sinus.[19] As pressure in the venous sinus rises, meningeal arteries form fistulous connections with dural sinuses or cortical veins. The development of the AVF can be a dynamic process as the thrombosed sinus recanalizes and additional ECA feeders are recruited.[57] The resultant venous hypertension can lead to vasogenic edema, venous infarct, and ultimately intracerebral hemorrhage. Patients can present in a delayed fashion with a variety of symptoms as they are dependent on venous congestion and mass effect from hemorrhage rather than the location of the fistula itself. While CTA and MRA can screen for the presence of an AVF, DSA remains the gold standard as it is necessary to display the architecture of the AVF and the location of the fistulous point.

Patients with AVF and cortical venous drainage with severe neurologic deficits have a poor prognosis with annual rates of intracerebral hemorrhage and neurologic deficits of 7.4% to 19% with an annual mortality rate of 3.8%. Urgent treatment is recommended in these patients to eliminate these risks. Asymptomatic patients with AVF and cortical venous drainage have a more benign course that nevertheless is still significant. Their annual rates of hemorrhage and neurologic decline are 1.4% to 1.5% with an annual mortality rate of 0% and therefore treatment is still recommended in properly selected patients.[57] Endovascular treatment options are similar to those of CCF and primarily consist of embolization with Onyx, nBCA, or coils with a primary goal to disconnect the fistulous point.

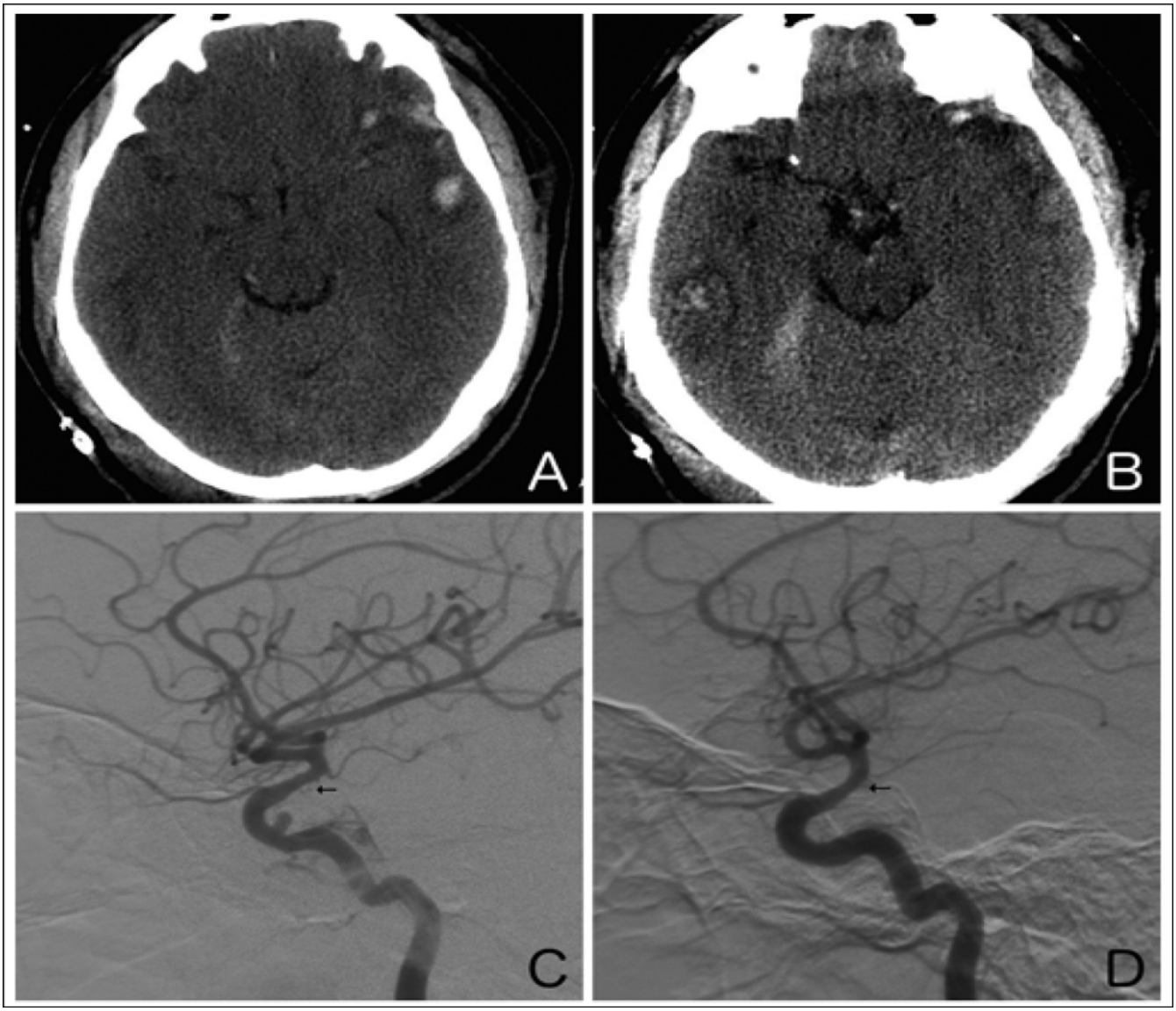

FIGURE 2: Illustrative case. A 25-year-old man presented after an assault to the head. An axial noncontrast computed tomography scan of the head showed a left temporal contusion with adjacent subarachnoid hemorrhage (A) as well as a right temporal contusion and tentorial subdural hematoma (B). Right (C) and left (D) internal carotid artery digital subtraction angiograms (lateral view) obtained on day 10 showing moderate vasospasm of both supraclinoid internal carotid arteries (arrows). Note the traumatic pseudoaneurysm of the cavernous right internal carotid artery.

Other Extracranial Arteriovenous Fistula

Extracranial AVF are rare lesions that may result from traumatic injury to the vessels in the neck, most commonly after penetrating neck injuries.[58,59] They typically arise from the vertebral artery due to its close proximity to vertebral veins and the epidural venous plexus, but they can involve other vessels such as the ECA branches, common carotid artery, and ICA.[19,60–62] Diagnosis is challenging due to an often delayed presentation and indolent course. Patients can present with a variety of symptoms depending on the location of the AVF as well as the degree of shunting and subsequent intracranial venous congestion. They can range from mild symptoms such as dizziness, bruit, and pulsatile tinnitus to more severe ones such cervical myelopathy, subarachnoid hemorrhage, and cranial nerve deficits.[19,59–62]

Non-invasive diagnostic imaging such as color Doppler ultrasonography, MRA, and CTA has taken an increasing role in the evaluation of these patients. CTA is particularly useful in the screening of penetrating trauma patients due to its ability to evaluate the entire cerebrovascular circulation from the aortic arch to the intracranial vessels.[2,63,64] DSA remains the most commonly used method for diagnosis and treatment since it can assess the arterial feeders, fistulous point, collateral circulation, and venous drainage. Furthermore, the majority of extracranial AVF arise from the vertebral artery and the anatomy of this artery makes surgical treatment difficult. Endovascular repair is a safe and feasible option with occlusion rates of up to 89%.[58] Endovascular options include detachable coils, stent placement, coil embolization, and liquid embolization with a primary goal of obliterating the fistulous point.[19,58,60–62]

Epistaxis

With extensive skull base fractures, cerebrovascular injury may occur, particularly in areas where the artery is fixed to the skull base.[65] Traumatic pseudoaneurysms may be asymptomatic or present after a contained rupture such as the case

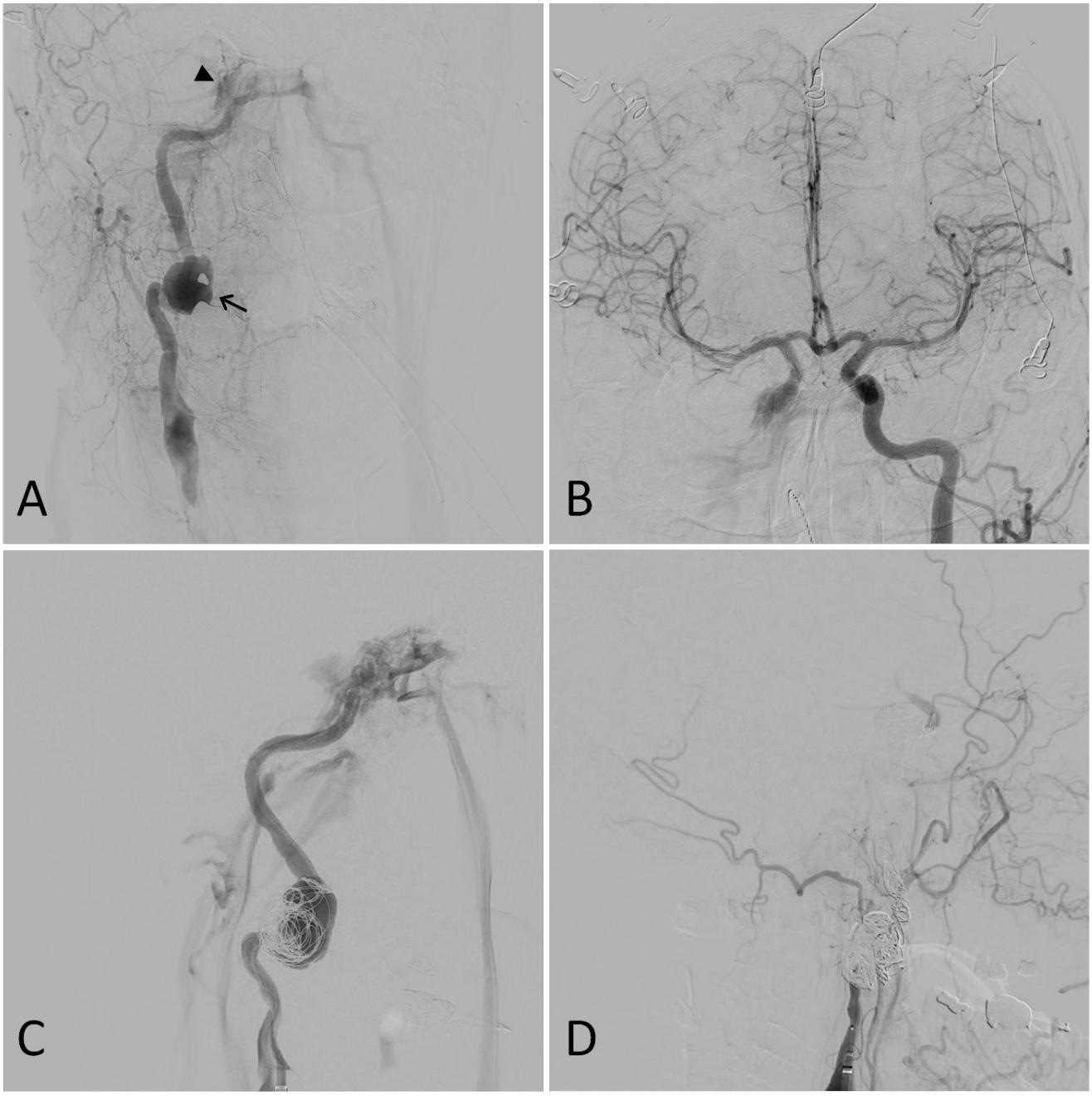

FIGURE 3: Illustrative case. A 50-year-old male presented after a motor vehicle accident. A computed tomography angiogram revealed a right internal carotid artery (ICA) pseudoaneurysm with associated stenosis. A digital subtraction angiogram showed a 27mm right ICA dissecting pseudoaneurysm (arrow) and a right cavernous-carotid fistula (CCF) (arrowhead) (A). Furthermore, the right anterior circulation was noted to fill completely through the left ICA (B). A partial coil embolization was performed at this time to protect the dome of the pseudoaneurysm (C). A repeat angiogram two weeks later showed the right ICA was dissected throughout its entire course and ended in a false lumen in the supraclinoid segment with no antegrade flow or intracranial filling. The CCF, pseudoaneurysm, and dissected ICA were then coiled. A final right common carotid angiogram showed no filling of the ICA, pseudoaneurysm, or CCF (D).

of a CCF or with severe epistaxis. The incidence of intractable post-traumatic epistaxis ranges from 1-11%.[66–68] The most commonly involved vessel is the ECA, especially the internal maxillary artery, followed by the cervical, petrous, and cavernous segments of the ICA.[21,22]

Magnetic resonance imaging and CTA are often performed first when establishing diagnosis, however angiographic imaging remains the gold standard.[22,66] CTA has an overall sensitivity of 80% and may also be useful in guiding superselective angiography necessary to demonstrate the lesion.[19,20]

Severe craniofacial injury combined with intractable oronasal epistaxis is potentially fatal, with a mortality rates reported between 30% and 50%.[69,70] Presentation may be delayed, with an interval of three days to six months, however most present within the first three weeks.[65] Nasal packing or tamponade with balloon catheters is oftentimes effective in controlling mild to moderate bleeding; however, the effectiveness is lessened with severe bleeding.[66,71] If conservative techniques fail to achieve hemostasis, surgical therapy is often necessitated.

Interventional approaches to traumatic aneurysms have evolved from Huntarian parent vessel ligation to a largely endovascular paradigm. Success has been reported with the

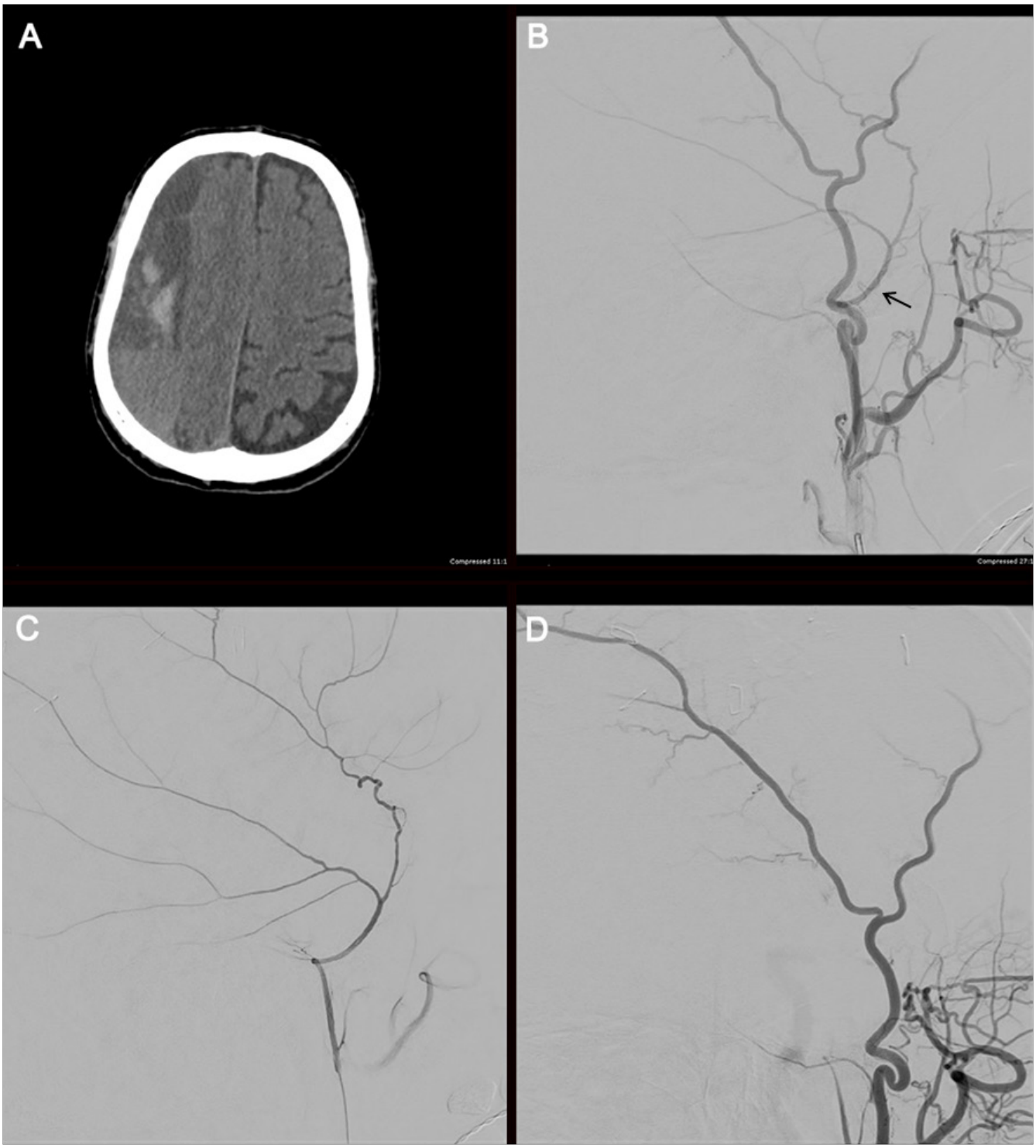

FIGURE 4: Illustrative case. Radiographic course of an 82-year-old male who presented with a large, septated, chronic right-sided subdural hematoma (A). The patient underwent a cerebral angiogram with embolization of the right middle meningeal artery using Embospheres. An external carotid artery angiogram (B) shows filling of the middle meningeal artery (arrow). A selective middle meningeal artery angiogram was then performed (C). A post-embolization angiogram of the external carotid artery shows no filling of the middle meningeal artery (D).

use of detachable coils, detachable balloons, covered stents, and flow-diverting stents.[65] Packing of the pseudoaneurysm with coils is feasible when the aneurysm neck is narrow; however, it carries risks of rupture in the acute phase.[22,23] If the aneurysm neck is narrow or the vessel is dysplastic, then balloon reconstruction, stent placement, or flow diversion is often preferred. Covered stents have shown good efficacy in managing these lesions, however these carry the risk of occluding potentially important branch vessels. For this reason, use of flow diverting stents is gaining popularity.[24,25] In either case however, endovascular stent placement necessitates dual antiplatelet therapy and the risk of bleeding in trauma patients must be weighed against risk of complications from an untreated pseudoaneurysm on an individualized basis. Parent artery occlusion is an additional management option; however extreme caution must be taken when pursuing this technique as it is laden with potential grave complications. When the lesion involves the ICA, a balloon test occlusion is necessary to demonstrate adequate collateral circulation. Even after a successful balloon test occlusion, up to 22% of

patients may still develop complications after therapeutic occlusion of the vessel.[72,73]

Cerebral Venous Sinus Thrombosis

CVST is an entity that is oftentimes associated with head trauma and TBI with an incidence reported up to 7%.[74] Clinical features of CVST vary and include headache, seizures, and focal neurologic deficits; however, CVST can carry more significant morbidity, particularly if involving the posterior third of the superior sagittal sinus or dominant transverse or sigmoid sinus. In these cases, venous infarct may result in catastrophic hemorrhage, elevation in intracranial pressure, coma, and death.[75] Poor prognostic indicators for CVST include coma at presentation, delineated infarction, and deep venous involvement.[19,76] Oftentimes, CVST is related to a skull fracture and the location of the fracture often determines which sinus is affected.[77] They may also occur secondary to external compression from an underlying hematoma.[78] A study by Hersh et al. found that of 113 patients who underwent formal venous imaging, 34% had sinus thrombosis, 17% of which demonstrated external compression by an extra-axial hemorrhage.[74]

Non-invasive imaging such as CT venography and MR venography are adequate for establishing diagnosis, with sensitivity and specificities reported up to 95%.[78–80] Interestingly, contrast-enhancing T1-weighted MR imaging is thought to be most accurate for detecting dural venous sinus and cortical venous sinus thrombosis.[81] DSA is typically reserved for patients in whom endovascular therapy is being planned.[78] Patients with skull fractures or extra-axial hemorrhages adjacent to or overlying cerebral venous sinuses or patients with a high suspicion for sinus injury should be screened.

Treatment of CVST involves aggressive rehydration and anticoagulation, which often poses as a challenge in patients with recent trauma. In patients with traumatic CVST treated with anticoagulation, rates of worsening intracranial hemorrhage have been reported in up to 14% with a 4.5% mortality.[74] Unfortunately, there are no clear guidelines delineating the appropriate time to initiate anticoagulation. However, studies suggest that commencing anticoagulation after 48-72 hours from initial injury is associated with fewer deleterious effects.[82] Optimal treatment duration is also unclear, but 3-6 months is typically accepted as an appropriate treatment course.[78]

Patients who fail to improve or who have worsening neurologic deficits in spite of medical therapy are considered for chemical or mechanical thrombolysis. Endovascular strategies include intrasinus thrombolysis, balloon angioplasty, thrombectomy, and sinus stent placement.[83,84] These techniques may be used in isolation or in combination. A systematic review demonstrated a 69% radiographic resolution rate with mechanical thrombectomy in medically refractory cases, with a 14.3% mortality rate during the follow up period.[85] Additionally, recent reports have shown an improvement in intracranial pressure following mechanical thrombectomy and stent placement of dural sinuses.[86]

Subdural Hematoma

Traumatic chronic SDH represent one of the most pervasive neurosurgical conditions, with an estimated annual incidence of 58.1-80.1 per 100,000 among individuals older than 65 years.[87–89] With the aging population, the incidence of chronic SDH is expected to double within the next 20 years.[90] Furthermore, 11-28% of patients who undergo surgical evacuation of a chronic SDH will experience recurrence.[90–92] The traditional theory that head trauma results in the disruption of bridging subdural veins has been widely contested in recent literature, which suggests that the pathophysiology of chronic SDH are the result of local inflammation, causing hyperfibrinolysis of the clot, and production of angiogenic factors, which promote neovascularization and bleeding from fragile capillaries.[93–97] Delineation of these pathophysiologic mechanisms has facilitated the emergence of new and less invasive therapies.

While surgical evacuation remains the gold standard for alleviating the mass effect from the hematoma, this does not treat the underlying neovascularization and inflammation, often allowing for the possibility of recurrence. As such, endovascular therapies are emerging as a viable treatment option. These therapies are based on the notion that embolization of middle meningeal artery (MMA) branches that supply the dura will inhibit blood influx into pathologic structures and control bleeding from the chronic SDH membrane promoting spontaneous hematoma resolution.

Embolization may be performed with microparticles, Onyx, nBCA, or coils. A recent systematic review found that PVA was the most commonly used embolic agent, however efficacy and rate of complications were not affected by choice of embolisate.[98] Before embolization, selective angiography of the MMA is performed in order to target the branches of interest and avoid potentially dangerous collaterals (Figure 4). When flow stasis is observed in the MMA, the procedure is concluded.

The largest reported series included 72 patients who underwent MMA embolization, which were compared to 469 patients who underwent conventional treatment.[99] In this study, all asymptomatic patients who underwent MMA embolization achieved spontaneous hematoma resolution. Of the 45 symptomatic patients, only one patient had hematoma re-accumulation. Importantly, treatment failure rate was lower in the embolization group as compared to the conventional treatment group at 1.4% versus 27.5%, and treatment complication rate was not different between the two groups.

A recent meta-analysis demonstrated a composite recurrence rate of 2.1% and 3.6% across double-arm and single-arm studies respectively, far lower than those reported for open surgical hematoma evacuation.[100] The modified Rankin Scale was not found to differ between embolization and conventional treatment groups, but this variable was not reported in select large studies. In the available literature on functional outcomes, good outcome (modified Rankin Scale of 0-2) has been reported in up to 85% of patients.[101]

The ideal timing for MMA embolization remains to be determined as successful outcomes have been reported for embolization both before and after surgical evacuation, or as a

stand-alone procedure. For patients with symptomatic or significant mass effect, surgical hematoma evacuation may still be necessary as a primary procedure with MMA embolization as an adjunct procedure to prevent recurrence. As this is still a developing technique, further study needs to be performed to determine which patients will best benefit from MMA embolization.

LIMITATIONS

The main limitation of this nonsystematic review is the paucity of literature and randomized controlled trials evaluating the efficacy of treatment options. Furthermore, as many of these traumatic injuries are rare, much of the literature for these disease processes stems from the non-traumatic etiologies, and the literature and subsequent treatment algorithms are extrapolated for the management of the traumatic injuries. As neuroendovascular techniques rapidly evolve, the efficacy of newer therapies has yet to be fully evaluated.

CONCLUSION

Neuroendovascular techniques have evolved tremendously to add to the armamentarium of neurosurgeons and trauma teams in the care of patients with traumatic head and neck injuries. Improvements in noninvasive imaging such as CTA and MRA have allowed for their increasing use as screening modalities. However, DSA remains the gold standard for diagnosing vascular injury and it allows for the safe and effective treatment of patients with carotid and vertebral dissections, pseudoaneurysms, various arteriovenous fistulas, and now even subdural hematomas. As neuroendovascular teams become more prevalent and available for rapid interventions, it is possible that endovascular treatments may take an expanded role in the management of acute trauma patients. However, more trials are needed to properly delineate the role of neuroendovascular techniques in the management of traumatic head and neck injuries.

CONFLICTS OF INTEREST

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REFERENCES

1. Radvany MG, Gailloud P. Endovascular management of neurovascular arterial injuries in the face and neck. *Semin Intervent Radiol* 2010;27(1):44–54.
2. Múnera F, Cohn S, Rivas LA. Penetrating injuries of the neck: use of helical computed tomographic angiography. *J Trauma* 2005;58(2):413–8.
3. Rao PM, Ivatury RR, Sharma P, et al. Cervical vascular injuries: a trauma center experience. *Surgery* 1993;114(3):527–31.
4. Biffl WL, Moore EE, Offner PJ, Brega KE, Franciose RJ, Burch JM. Blunt carotid arterial injuries: implications of a new grading scale. *J Trauma* 1999;47(5):845–53.
5. Fusco MR, Harrigan MR. Cerebrovascular dissections: a review. Part II: blunt cerebrovascular injury. *Neurosurgery* 2011;68(2):517–30; discussion 530.
6. Kerwin AJ, Bynoe RP, Murray J, et al. Liberalized screening for blunt carotid and vertebral artery injuries is justified. *J Trauma* 2001;51(2):308–14.
7. Stein DM, Boswell S, Sliker CW, Lui FY, Scalea TM. Blunt cerebrovascular injuries: does treatment always matter? *J Trauma* 2009;66(1):132–43; discussion 143-144.
8. Griessenauer CJ, Fleming JB, Richards BF, et al. Timing and mechanism of ischemic stroke due to extracranial blunt traumatic cerebrovascular injury. *J Neurosurg* 2013;118(2):397–404.
9. Harrigan MR, Falola MI, Shannon CN, et al. Incidence and trends in the diagnosis of traumatic extracranial cerebrovascular injury in the nationwide inpatient sample database, 2003-2010. *J Neurotrauma* 2014;31(11):1056–62.
10. Spaniolas K, Velmahos GC, Alam HB, et al. Does improved detection of blunt vertebral artery injuries lead to improved outcomes? Analysis of the National Trauma Data Bank. *World J Surg* 2008;32(10):2190–4.
11. Kim DY, Biffl W, Bokhari F, et al. Evaluation and management of blunt cerebrovascular injury: A practice management guideline from the Eastern Association for the Surgery of Trauma. *J Trauma Acute Care Surg* 2020;88(6):875–87.
12. Biffl WL, Cothren CC, Moore EE, et al. Western Trauma Association critical decisions in trauma: screening for and treatment of blunt cerebrovascular injuries. *J Trauma* 2009;67(6):1150–3.
13. Bromberg WJ, Collier BC, Diebel LN, et al. Blunt cerebrovascular injury practice management guidelines: the Eastern Association for the Surgery of Trauma. *J Trauma* 2010;68(2):471–7.
14. Majidi S, Hassan AE, Adil MM, et al. Incidence and outcome of vertebral artery dissection in trauma setting: analysis of national trauma data base. *Neurocrit Care* 2014;21(2):253–8.
15. Scott WW, Sharp S, Figueroa SA, et al. Clinical and radiographic outcomes following traumatic Grade 1 and 2 carotid artery injuries: a 10-year retrospective analysis from a Level I trauma center. The Parkland Carotid and Vertebral Artery Injury Survey. *Journal of Neurosurgery JNS* 2015;122(5):1196–201.
16. Scott WW, Sharp S, Figueroa SA, et al. Clinical and radiological outcomes following traumatic Grade 1 and 2 vertebral artery injuries: a 10-year retrospective analysis from a Level 1 trauma center. *Journal of Neurosurgery JNS* 2014;121(2):450–6.
17. Scott WW, Sharp S, Figueroa SA, et al. Clinical and radiographic outcomes following traumatic Grade 3 and 4 carotid artery injuries: a 10-year retrospective analysis from a Level 1 trauma center. The Parkland Carotid and Vertebral Artery Injury Survey. *Journal of Neurosurgery JNS* 2015;122(3):610–5.
18. Scott WW, Sharp S, Figueroa SA, et al. Clinical and radiological outcomes following traumatic Grade 3 and 4 vertebral artery injuries: a 10-year retrospective analysis from a Level I trauma center. The Parkland Carotid and Vertebral Artery Injury Survey. *Journal of Neurosurgery JNS* 2015;122(5):1202–7.
19. Alderazi YJ, Cruz GM, Kass-Hout T, et al. Endovascular therapy for cerebrovascular injuries after head and neck trauma. *Trauma* 2015;17(4):258–69.
20. Cox MW, Whittaker DR, Martinez C, et al. Traumatic pseudoaneurysms of the head and neck: early endovascular intervention. *J Vasc Surg* 2007;46(6):1227–33.
21. Al-Jehani HM, Alwadaani HA, Almolani FM. Traumatic intracranial internal carotid artery pseudoaneurysm presenting as epistaxis treated by endovascular coiling. *Neurosciences* (Riyadh) 2016;21(1):60–3.
22. Zhang C wei, Xie X dong, You C, et al. Endovascular treatment of traumatic pseudoaneurysm presenting as intractable epistaxis. *Korean J Radiol* 2010;11(6):603–11.
23. Chen D, Concus AP, Halbach VV, et al. Epistaxis originating from traumatic pseudoaneurysm of the internal carotid artery: diagnosis and endovascular therapy. *Laryngoscope* 1998;108(3):326–31.
24. Auyeung KM, Lui WM, Chow LCK, et al. Massive epistaxis related to petrous carotid artery pseudoaneurysm after radiation therapy: emergency treatment with covered stent in two cases. *AJNR Am J Neurora-*

diol 2003;24(7):1449–52.

25. Celil G, Engin D, Orhan G, et al. Intractable epistaxis related to cavernous carotid artery pseudoaneurysm: treatment of a case with covered stent. *Auris Nasus Larynx* 2004;31(3):275–8.
26. Amuluru K, Al-Mufti F, Roth W, et al. Anchoring Pipeline Flow Diverter Construct in the Treatment of Traumatic Distal Cervical Carotid Artery Injury. *Interv Neurol* 2017;6(3–4):153–62.
27. Nicosia G, Cicala D, Mirone G, et al. Childhood acute basilar artery thrombosis successfully treated with mechanical thrombectomy using stent retrievers: case report and review of the literature. *Childs Nerv Syst* 2017;33(2):349–55.
28. Al-Mufti F, Kamal N, Damodara N, et al. Updates in the Management of Cerebral Infarctions and Subarachnoid Hemorrhage Secondary to Intracranial Arterial Dissection: A Systematic Review. *World Neurosurg* 2019;121:51–8.
29. Krings T, Choi I-S. The many faces of intracranial arterial dissections. *Interv Neuroradiol* 2010;16(2):151–60.
30. Debette S, Compter A, Labeyrie M-A, et al. Epidemiology, pathophysiology, diagnosis, and management of intracranial artery dissection. *The Lancet Neurology* 2015;14(6):640–54.
31. Urasyanandana K, Songsang D, Aurboonyawat T, et al. Treatment outcomes in cerebral artery dissection and literature review. *Interv Neuroradiol* 2018;24(3):254–62.
32. Fullerton HJ, Johnston SC, Smith WS. Arterial dissection and stroke in children. *Neurology* 2001;57(7):1155–60.
33. Sönmez Ö, Brinjikji W, Murad MH, et al. Deconstructive and Reconstructive Techniques in Treatment of Vertebrobasilar Dissecting Aneurysms: A Systematic Review and Meta-Analysis. *AJNR Am J Neuroradiol* 2015;36(7):1293–8.
34. Oh DC, Hirsch JA, Yoo AJ. Novel use of onyx for treatment of intracranial vertebral artery dissection. *J Neurointerv Surg* 2012;4(1):31–3.
35. Provencio JJ, Vora N. Subarachnoid hemorrhage and inflammation: bench to bedside and back. *Semin Neurol* 2005;25(4):435–44.
36. Al-Mufti F, Amuluru K, Changa A, et al. Traumatic brain injury and intracranial hemorrhage-induced cerebral vasospasm: a systematic review. *Neurosurg Focus* 2017;43(5):E14.
37. O'Brien NF, Reuter-Rice KE, Khanna S, et al. Vasospasm in children with traumatic brain injury. *Intensive Care Med* 2010;36(4):680–7.
38. Ract C, Le Moigno S, Bruder N, et al. Transcranial Doppler ultrasound goal-directed therapy for the early management of severe traumatic brain injury. *Intensive Care Med* 2007;33(4):645–51.
39. Shahlaie K, Keachie K, Hutchins IM, et al. Risk factors for posttraumatic vasospasm. *J Neurosurg* 2011;115(3):602–11.
40. Armonda RA, Bell RS, Vo AH, et al. Wartime traumatic cerebral vasospasm: recent review of combat casualties. *Neurosurgery* 2006;59(6):1215–25; discussion 1225.
41. O'Brien NF, Maa T, Yeates KO. The epidemiology of vasospasm in children with moderate-to-severe traumatic brain injury. *Crit Care Med* 2015;43(3):674–85.
42. Zubkov AY, Lewis AI, Raila FA, Zhang J, Parent AD. Risk factors for the development of post-traumatic cerebral vasospasm. *Surg Neurol* 2000;53(2):126–30.
43. Al-Mufti F, Amuluru K, Lander M, et al. Low Glasgow Coma Score in Traumatic Intracranial Hemorrhage Predicts Development of Cerebral Vasospasm. *World Neurosurg* 2018;120:e68–71.
44. Harders A, Kakarieka A, Braakman R. Traumatic subarachnoid hemorrhage and its treatment with nimodipine. German tSAH Study Group. *J Neurosurg* 1996;85(1):82–9.
45. Ogami K, Dofredo M, Moheet AM, et al. Early and Severe Symptomatic Cerebral Vasospasm After Mild Traumatic Brain Injury. *World Neurosurg* 2017;101:813.e11-813.e14.
46. Mergeani A, Popescu D, Laza C, et al. A review on endovascular techniques for treatment of direct post-traumatic carotid-cavernous fistula supported by case presentations. *Maedica (Bucur)* 2012;7(4):332–8.
47. Gemmete JJ, Ansari SA, Gandhi DM. Endovascular techniques for treatment of carotid-cavernous fistula. *J Neuroophthalmol* 2009;29(1):62–71.
48. Henderson AD, Miller NR. Carotid-cavernous fistula: current concepts in aetiology, investigation, and management. *Eye (Lond)* 2018;32(2):164–72.
49. Dahlin BC, Waldau B. Surgical and Nonsurgical Treatment of Vascular Skull Base Trauma. *J Neurol Surg B Skull Base* 2016;77(5):396–403.
50. Shownkeen H, Bova D, Origitano TC, et al. Carotid-cavernous fistulas: pathogenesis and routes of approach to endovascular treatment. *Skull Base* 2001;11(3):207–18.
51. de Keizer R. Carotid-cavernous and orbital arteriovenous fistulas: ocular features, diagnostic and hemodynamic considerations in relation to visual impairment and morbidity. *Orbit* 2003;22(2):121–42.
52. Ellis JA, Goldstein H, Connolly ES, et al. Carotid-cavernous fistulas. *Neurosurg Focus* 2012;32(5):E9.
53. Liu HM, Wang YH, Chen YF, et al. Long-term clinical outcome of spontaneous carotid cavernous sinus fistulae supplied by dural branches of the internal carotid artery. *Neuroradiology* 2001;43(11):1007–14.
54. Liu Y-C, Duan C-Z, Gu D-Q, et al. The recovery time of traumatic carotid-cavernous fistula-induced oculomotor nerve paresis after endovascular treatment with detachable balloons. *J Neuroradiol* 2014;41(5):329–35.
55. Ogilvy CS, Motiei-Langroudi R, Ghorbani M, et al. Flow Diverters as Useful Adjunct to Traditional Endovascular Techniques in Treatment of Direct Carotid-Cavernous Fistulas. *World Neurosurg* 2017;105:812–7.
56. Amuluru K, Al-Mufti F, Gandhi CD, et al. Direct carotid-cavernous fistula: A complication of, and treatment with, flow diversion. *Interv Neuroradiol* 2016;22(5):569–76.
57. Reynolds MR, Lanzino G, Zipfel GJ. Intracranial Dural Arteriovenous Fistulae. *Stroke* 2017;48(5):1424–31.
58. Herrera DA, Vargas SA, Dublin AB. Endovascular treatment of traumatic injuries of the vertebral artery. *AJNR Am J Neuroradiol* 2008;29(8):1585–9.
59. Robbs JV, Carrim AA, Kadwa AM, et al. Traumatic arteriovenous fistula: experience with 202 patients. *Br J Surg* 1994;81(9):1296–9.
60. Yeh C-H, Chen Y-L, Wu Y-M, et al. Anatomically based approach for endovascular treatment of vertebro-vertebral arteriovenous fistula. *Interv Neuroradiol* 2014;20(6):766–73.
61. Felbaum D, Chidambaram S, Mason RB, et al. Vertebral-venous fistula: an unusual cause for ocular symptoms mimicking a carotid cavernous fistula. *BMJ Case Rep* 2015;2015.
62. Yu J, Guo Y, Wu Z, Xu K. Traumatic arteriovenous fistula between the extracranial middle meningeal artery and the pterygoid plexus: A case report and literature review. *Interv Neuroradiol* 2017;23(1):90–6.
63. Múnera F, Soto JA, Palacio DM, et al. Penetrating neck injuries: helical CT angiography for initial evaluation. *Radiology* 2002;224(2):366–72.
64. Múnera F, Soto JA, Palacio D, et al. Diagnosis of arterial injuries caused by penetrating trauma to the neck: comparison of helical CT angiography and conventional angiography. *Radiology* 2000;216(2):356–62.
65. Mankahla N, LeFeuvre D, Taylor A. Delayed massive epistaxis from traumatic cavernous carotid false aneurysms: A report of two unusual cases. *Interv Neuroradiol* 2017;23(4):387–91.
66. Wong C-W, Tan W-C, Yeh Y-T, et al. Transarterial embolization for traumatic intractable oronasal hemorrhage. *J Emerg Med* 2013;44(6):1088–91.
67. Ardekian L, Samet N, Shoshani Y, et al. Life-threatening bleeding following maxillofacial trauma. *J Craniomaxillofac Surg* 1993;21(8):336–8.
68. Tung TC, Tseng WS, Chen CT, et al. Acute life-threatening injuries in facial fracture patients: a review of 1,025 patients. *J Trauma* 2000;49(3):420–4.
69. Liao C-C, Hsu Y-P, Chen C-T, et al. Transarterial embolization for intractable oronasal hemorrhage associated with craniofacial trauma: evaluation of prognostic factors. *J Trauma* 2007;63(4):827–30.
70. Karamoskos P, Dohrmann PJ. Traumatic internal carotid artery aneu-

rysm and massive epistaxis. *Aust N Z J Surg* 1989;59(9):745–7.

71. Bynoe RP, Kerwin AJ, Parker HH, et al. Maxillofacial injuries and life-threatening hemorrhage: treatment with transcatheter arterial embolization. *J Trauma* 2003;55(1):74–9.
72. Eckert B, Thie A, Carvajal M, et al. Predicting hemodynamic ischemia by transcranial Doppler monitoring during therapeutic balloon occlusion of the internal carotid artery. *Am J Neuroradiol* 1998;19(3): 577-582.
73. Elias AE, Chaudhary N, Pandey AS, et al. Intracranial endovascular balloon test occlusion: indications, methods, and predictive value. *Neuroimaging Clin N Am* 2013;23(4):695–702.
74. Hersh D, Hayman E, Aarabi B, et al. 175 Safety of Anticoagulation for the Treatment of Cerebral Venous Sinus Thrombosis in Adult Trauma Patients. *Neurosurgery* 2016;63:169–70.
75. Qureshi AI, Sahito S, Liaqat J, et al. Traumatic Injury of Major Cerebral Venous Sinuses Associated with Traumatic Brain Injury or Head and Neck Trauma: Analysis of National Trauma Data Bank. *J Vasc Interv Neurol* 2020;11(1):27–33.
76. Ferro JM, Canhão P, Stam J, et al ISCVT Investigators. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke* 2004;35(3):664–70.
77. Delgado Almandoz JE, Kelly HR, Schaefer PW, et al. Prevalence of traumatic dural venous sinus thrombosis in high-risk acute blunt head trauma patients evaluated with multidetector CT venography. *Radiology* 2010;255(2):570–7.
78. Afshari FT, Yakoub KM, Zisakis A, et al. Traumatic dural venous sinus thrombosis; a challenge in management of head injury patients. *J Clin Neurosci* 2018;57:169–73.
79. Chiewvit P, Piyapittayanan S, Poungvarin N. Cerebral venous thrombosis: diagnosis dilemma. *Neurol Int* 2011;3(3):e13.
80. Khandelwal N, Agarwal A, Kochhar R, et al. Comparison of CT venography with MR venography in cerebral sinovenous thrombosis. *AJR Am J Roentgenol* 2006;187(6):1637–43.
81. Sari S, Verim S, Hamcan S, et al. MRI diagnosis of dural sinus - Cortical venous thrombosis: Immediate post-contrast 3D GRE T1-weighted imaging versus unenhanced MR venography and conventional MR sequences. *Clin Neurol Neurosurg* 2015;134:44–54.
82. Grangeon L, Gilard V, Ozkul-Wermester O, et al. Management and outcome of cerebral venous thrombosis after head trauma: A case series. *Rev Neurol (Paris)* 2017;173(6):411–7.
83. Qureshi AI. A Classification Scheme for Assessing Recanalization and Collateral Formation following Cerebral Venous Thrombosis. *J Vasc Interv Neurol* 2010;3(1):1–2.
84. Qureshi AI, Grigoryan M, Saleem MA, et al. Prolonged Microcatheter-Based Local Thrombolytic Infusion as a Salvage Treatment After Failed Endovascular Treatment for Cerebral Venous Thrombosis: A Multicenter Experience. *Neurocrit Care* 2018;29(1):54–61.
85. Ilyas A, Chen C-J, Raper DM, et al. Endovascular mechanical thrombectomy for cerebral venous sinus thrombosis: a systematic review. *J Neurointerv Surg* 2017;9(11):1086–92.
86. Chen K-W, Lin Y-H, Lee C-W. Acute Posttraumatic Cerebral Venous Sinus Thrombosis-Induced Malignant Increased Intracranial Pressure Treated with Endovascular Dural Sinus Thrombectomy and Stenting. *World Neurosurg* 2019;128:393–7.
87. Okuma Y, Hirotsune N, Sato Y, et al. Midterm Follow-Up of Patients with Middle Meningeal Artery Embolization in Intractable Chronic Subdural Hematoma. *World Neurosurg* 2019;126:e671–8.
88. Karibe H, Kameyama M, Kawase M, et al. Epidemiology of chronic subdural hematomas. *No Shinkei Geka* 2011;39(12):1149–53.
89. Kudo H, Kuwamura K, Izawa I, et al. Chronic subdural hematoma in elderly people: present status on Awaji Island and epidemiological prospect. *Neurol Med Chir (Tokyo)* 1992;32(4):207–9.
90. Ramachandran R, Hegde T. Chronic subdural hematomas--causes of morbidity and mortality. *Surg Neurol* 2007;67(4):367–72; discussion 372-373.
91. Amirjamshidi A, Abouzari M, Eftekhar B, et al. Outcomes and recurrence rates in chronic subdural haematoma. *Br J Neurosurg* 2007;21(3):272–5.
92. Jung Y-G, Jung N-Y, Kim E. Independent predictors for recurrence of chronic subdural hematoma. *J Korean Neurosurg Soc* 2015;57(4):266–70.
93. Santarius T, Kirkpatrick PJ, Kolias AG, et al. Working toward rational and evidence-based treatment of chronic subdural hematoma. *Clin Neurosurg* 2010;57:112–22.
94. Frati A, Salvati M, Mainiero F, et al. Inflammation markers and risk factors for recurrence in 35 patients with a posttraumatic chronic subdural hematoma: a prospective study. *J Neurosurg* 2004;100(1):24–32.
95. Stanisic M, Aasen AO, Pripp AH, et al. Local and systemic pro-inflammatory and anti-inflammatory cytokine patterns in patients with chronic subdural hematoma: a prospective study. *Inflamm Res* 2012;61(8):845–52.
96. Ito H, Yamamoto S, Komai T, et al. Role of local hyperfibrinolysis in the etiology of chronic subdural hematoma. *J Neurosurg* 1976;45(1):26–31.
97. Kalamatianos T, Stavrinou LC, Koutsarnakis C, et al. PlGF and sVEGFR-1 in chronic subdural hematoma: implications for hematoma development. *J Neurosurg* 2013;118(2):353–7.
98. Waqas M, Vakhari K, Weimer PV, et al. Safety and Effectiveness of Embolization for Chronic Subdural Hematoma: Systematic Review and Case Series. *World Neurosurg* 2019;126:228–36.
99. Ban SP, Hwang G, Byoun HS, et al. Middle Meningeal Artery Embolization for Chronic Subdural Hematoma. *Radiology* 2018;286(3):992–9.
100. Srivatsan A, Mohanty A, Nascimento FA, et al. Middle Meningeal Artery Embolization for Chronic Subdural Hematoma: Meta-Analysis and Systematic Review. *World Neurosurg* 2019;122:613–9.
101. Kim E. Embolization Therapy for Refractory Hemorrhage in Patients with Chronic Subdural Hematomas. *World Neurosurg* 2017;101:520–7.