Airbag Associated Bilateral Internal Carotid Artery Dissection with Hyperdense Middle Cerebral Artery Sign - A Case Report
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INTRODUCTION
Traumatic bilateral dissection of the internal carotid artery is a devastating injury. Although it leads to serious consequences, it remains underreported because it is overlooked. The mechanisms that cause this injury vary widely. Horseback riding, rollercoasters, strangulation, softballs, and airbags, to name a few, have been named as culprits in the literature. As illustrated by our case, airbag deployment can result in injury as serious as bilateral carotid dissection and can be associated with a grim outcome. It is our intention to raise awareness to such presentations in order to facilitate diagnosis, since full recovery requires prompt recognition and treatment.

CASE REPORT
A 29-year-old female was involved in an early evening motor vehicle accident in which her airbag deployed. According to paramedics, she briefly lost consciousness at the scene. She was transferred to our hospital from another primary center, where a non-contrast computed tomography (CT) had been performed. In the ER, her Glasgow Coma Scale (GCS) was 14, her pupils were 2.5mm and equally reactive, and she moved all four extremities spontaneously. The patient was admitted to the trauma service for observation. Her vital signs were normal, and a complete blood count showed all values to be normal, including hematocrit. Although neurologically intact, our patient was consistently agitated and no explanation could be provided at the time. A head CT at admission was unremarkable. Several hours later, the patient became increasingly agitated and confused. A repeat head CT at 1:00 am showed mild cerebral edema and a bilateral hyperdense middle cerebral artery (MCA) that was originally attributed to contrast administration (figure 1). By 6:00 am the patient was comatose and a head CT performed at that time showed loss of the gray-white matter interface in both hemispheres (figure 2). An angiogram performed following the CT revealed bilateral carotid dissection (figures 3-6); several hours later, the patient expired from a massive bilateral stroke despite aggressive medical treatment.
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Figure 1
Fig. 1. Bilateral hyperdense middle cerebral arteries, a classic sign of internal carotid artery dissection.

Figure 2
Fig. 2. Brain CT showing loss of the grey-white interface in the region of the anterior circulation, indicating ischemia.

Figure 3
Fig. 3. Angiogram showing contrast in the left common and left external carotid arteries, but absent in the left internal carotid artery.
Figure 4
Fig. 4. Angiogram demonstrating contrast in the right common and right external carotid arteries, with absent flow in the right internal carotid artery.

Figure 5
Fig. 5. Cerebral angiogram showing contrast perfusion only in the distribution of the right external carotid artery with no contrast in the right internal carotid artery territory.

Figure 6
Fig. 6. Cerebral angiogram showing contrast perfusion only in the area of the left external carotid artery. Note the absence of contrast in the area of the left internal carotid artery.

DISCUSSION
Although airbags are proven to decrease morbidity and mortality, attention is now being brought to their potential as a source of injury. Risk factors for airbag-induced injury include airbag deployment without seatbelt use, improper
The extracranial portion of the internal carotid artery carries...
DIAGNOSIS

It has been suggested that patients with certain types of injuries be screened for traumatic dissection of the internal carotid arteries. Appropriate screening in a timely manner is imperative to avoid potential neurological consequences. Once dissection is suspected, there are several methods to diagnose the condition. Possible options include duplex ultrasound, angiography, CT angiography (CTA) and magnetic resonance angiography (MRA). According to a recent multicenter review, although ultrasound is non-invasive, it has an 86% sensitivity for identifying internal carotid artery injuries. Most injuries that were missed by ultrasound screening were distal lesions near the base of the skull. MRA may be a non-invasive and contrast-free alternative, with a sensitivity that exceeds 87%. Another advantage is that the length of the dissection can also be shown on MRA. However, MRA is expensive and may not be immediately available in the trauma setting.

Ideally, a non-contrast CT of the head should be performed first, as it is a common test in the trauma setting to rule out intracranial bleeding. If a diagnosis of dissection is considered, CTA, as of late, is thought to be the best method of diagnosis. CTAs are convenient because trauma patients may already be making a trip to the CT scanner for imaging of other areas, it is a rapid test, and high-speed scanners decrease the amount of contrast needed. Suspicious lesions on a CTA or MRA can then be confirmed with a cerebral angiogram, which is considered to be the gold standard of diagnosis.

One of the hallmark signs of carotid dissection is the finding of a hyperdense MCA on a non-contrast enhanced CT scan. Gacs et al. first defined it in 1983, thought to be associated with an internal carotid artery dissection. The arterial dissection can lead to thrombus formation and vessel occlusion, raising the local hematocrit. When present, it is an early sign, indicating impending thrombosis. A hyperdense MCA sign is rare, but its bilaterality in the absence of other contributing factors is exceptional. A highly specific sign, its identification can be used to start therapy in the hyperacute post-traumatic stage. Unfortunately, this is a transient sign that is present on CT for only a few hours, making it difficult to detect. The hyperdense MCA sign, however, must be differentiated from a pseudohyperdense MCA sign whose causes include increased hematocrit, atherosclerotic changes and contrast administration. An MCA may also falsely appear hyperdense due to hypodensity of surrounding brain tissue, which may occur post-traumatically. It is important to differentiate between these two signs in order to properly suspect internal carotid artery dissection. A hyperdense MCA on CT is reversible whereas a pseudohyperdense MCA is not. Also, a pseudohyperdense artery affects all cerebral vasculature, while a hyperdense MCA is specific to the MCA.

Due to the rare occurrence of bilateral internal carotid dissections, the best method of treatment has yet to be found. Table 1 shows treatments and outcomes for bilateral internal carotid dissection in previous case reports. Treatment can be either medical or surgical. Medical therapy includes anticoagulation and antiplatelet medications. Anticoagulation is usually used as a first line of therapy and has been demonstrated to decrease morbidity and mortality. Heparin has been associated with increased survival, improved neurological outcome and a higher GCS. It has been suggested that treatment be dependent on symptoms. In the absence of symptoms, anticoagulation has been found to reduce neurological complications, provided there are no contraindications. In 2007, Edwards et al. demonstrated that both anticoagulation and antiplatelet therapy had equal efficacy in treating dissections as well as pseudoaneurysms, a potential complication of dissection.

Surgical treatment includes endovascular and surgical treatment. Endovascular treatment has been considered to be the best nonmedical treatment. Common forms of endovascular treatment include stenting with balloon-expandable, self-expanding or covered stents. DuBose et al. published a review article in 2008 addressing the recent use of stents for the treatment of traumatic internal carotid injuries, including dissections. Their use was associated with favorable results, with a follow-up patency of 79.6% and no stent-related mortality. Use of a stent is useful in intracranial or high extracranial lesions. A downside to stenting is the requirement of dual antiplatelet therapy, which may be problematic in patients with multiple injuries, those with intracranial hemorrhage and those requiring surgery. Stent procedures may be associated with complications at the local access site and require a high level of expertise. Thrombosis of the stent may be associated with
small vessel size, under dilation of the stent, as well as a proximal or distal dissection.\textsuperscript{28}

Surgical intervention is also an option, but surgical access can sometimes be difficult. Modalities include vessel ligation, direct suturing of intimal injury, replacement of injured vessels with interposition grafts and bypass grafting. Carotid ligation can be complicated by ischemic stroke in 3-10%. There is also an increased risk of hypertension and de novo intracranial aneurysm formation. Direct repair is associated with cranial nerve injury.\textsuperscript{19} In comparison, surgery has an associated mortality of up to 22%, while stenting is much lower, 0.9%. The associated morbidity, however, is only 3.5% for stents, and between 0 and 21% for surgical intervention.\textsuperscript{27}

In summary, our case shows that a patient with seemingly normal, reactive, equally sized pupils might be suffering from sympathetic nerve plexus injury. A hyperdense MCA on CT should raise the suspicion of imminent thrombosis, which in our case was originally credited to contrast administration. Our bilateral findings are significant as they have never been published in the literature. Given the difficulty in identifying the symptoms of dissection in a timely manner as well as the necessity in recognizing possible coexisting injuries, the diagnosis requires an astute physician. As bilateral internal carotid dissection is a rare diagnosis without a standard of treatment, it is necessary to further study possible treatment options to maximize positive outcomes for patients.

References

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