



Carotid Artery Dissection and Cerebral Infarction Secondary to Blunt Trauma

Künt Travmaya Bağlı Gelişen Karotid Arter Diseksiyonu ve Serebral İnfarkt

Burcu Gökçe, A. Kemal Erdemoğlu

Kırıkkale University, Faculty of Medicine, Department of Neurology, Kırıkkale, Turkey

Summary

Traumatic carotid artery dissection may appear after blunt head or neck traumas. Patients can be asymptomatic or clinical symptoms may include headache, transient ischemic attack, stroke, loss of consciousness, hemiparesis, aphasia and Horner syndrome, typically occurring within hours to days. Prognosis is good if diagnosed and treated early. As cerebral ischemia and neurological deficits may develop subsequently, it is essential that carotid artery dissection should be considered, and possible cases evaluated and diagnosed appropriately. In this article, we present a case of internal carotid artery dissection with diagnostic neuro-imaging findings, that occurred after 6 hours of blunt trauma and subsequent cerebral infarction following a car accident. Clinical features, neuro-radiological diagnostic methods and treatments options are discussed with relevant literature. (*Turkish Journal of Neurology 2012; 18:162-167*)

Key Words: Trauma, carotid artery, dissection, stroke

Özet

Travmatik internal karotid arter diseksiyonu baş veya boynun künt travmalarından sonra meydana gelebilir. Hastalar asemptomatik olabileceği gibi klinik bulgular saatler veya günler içinde ilerleyen baş ağrısı, geçici iskemik atak, inme, Horner sendromu ve şuur bulanıklığını içerebilir. Erken tanı ve tedavi sağlanırsa prognoz iyidir. Olgularda ilerleyen dönemlerde serebral iskemi ve nörolojik defisit gelişebileceğinden şüpheli hasta grubunda karotis diseksiyonu akla gelmeli ve hastaların tanı ve tedavi için uygun şekilde araştırılması gereklidir. Görüntüleme bulguları eşliğinde sunduğumuz olguda araç içi trafik kazası sonrası 6. saatte künt post-travmatik internal karotid arter diseksiyonu gelişmiştir. Hastalığın klinik özellikleri, nöroradyolojik tanı metotları ve tedavi yöntemleri literatür gözden geçirilerek ele alınmıştır. (*Türk Nöroloji Dergisi 2012; 18:162-167*)

Anahtar Kelimeler: Travma, karotid arter, diseksiyon, inme

Introduction

Dissection is accumulation of blood within the arterial wall as a result of a tear in the blood vessel wall (intramural hematoma). This causes luminal stenosis following collection of blood between the intima and media or aneurismal dilation occurs if the build-up is between the media and the adventitia. Traumatic internal carotid artery dissection is one of the frequent causes of stroke seen in young individuals and causes approximately 20% of all strokes. Blunt traumas of the carotid artery are rare and although most patients experience symptoms within the first 24 hours following the trauma, some may have them weeks later, whereas 5% are asymptomatic and are diagnosed by chance (1). As early diagnosis

and treatment will change the outcome of mortality and morbidity, carotid dissection should be considered, investigated and treated in suspected patients.

Here we present with relevant literature, a case admitted to the emergency room with right hemiparesis following a motor vehicle accident, and diagnosed with posttraumatic internal carotid artery dissection after further investigation.

Case Presentation

Forty-one year old male patient presented at the emergency room with complaints of confusion, aphasia, and weakness in the right extremities developing 6 hours after the motor vehicle

Address for Correspondence / Yazışma Adresi: A. Kemal Erdemoğlu MD, Kırıkkale University, Faculty of Medicine, Department of Neurology, Turkey

Phone.: +90 232 388 41 77 E-posta: akerdemoglu@yahoo.com

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accident. There was nothing to note in his medical history or his family history. His blood pressure was 120/70 mm Hg, heart rate was 76/minute and respiratory rate was 20/minute. His neurological examination showed a tendency to sleep, but his response to verbal stimuli was assessed as complete. Pupils were isochoric and light reflex was bilateral. Strength in upper and lower right extremity was 0/5, and there was an increase in deep tendon reflexes. There was an extensor response and a flexor response for the plantar skin reflex in the right and left foot, respectively. There was a right hemi-hypoesthesia and right central facial paralysis. The NIHSS (National Institutes of Health Stroke Scale) score was found to be 18. There was no pathology in the initial computerized tomography (CT) scan taken in the emergency room (Image 1) and the patient was admitted in the neurology clinic with an preliminary diagnosis of stroke. His routine blood chemistry and hemogram values were within the normal range and INR (International Normalized Ratio) value was 0.87. His cranial magnetic resonance imaging (MRI) scan showed a field of hyperintensity in T2-FLAIR sections consistent with ischemia involving the mid cerebral arterial area in the left parietal lobe, globus pallidus, caudate nucleus, putamen and lateral thalamus in the inferior from the level of semioval centrum on the left (Image 2a) and the Diffusion Weighted Brain MRI (DWI) showed hyperintensity (Images 2b and 2c). The cranial magnetic resonance angiography (MRA) showed that there was no flow in the left internal carotid artery until the mid cerebral artery bifurcation level and in the left mid cerebral artery (Image 3a) and there was occlusion in the left internal carotid artery (Image 3b). In the computerized neck tomographic angiography (CTA) the left internal carotid artery was found to be occluded from the bifurcation level almost completely, and 1 cm from the proximal completely, and no contrast material was seen (Image 3c). Doppler ultrasonography (USG) of the carotid and vertebral arteries was consistent with complete thrombosis at the approximately 1.5 cm segment starting at the bifurcation level of the left internal carotid artery. Electrocardiography and echocardiography were normal. Vasculitis markers and investigations for stroke etiology did not show any pathology. The patient was heparinized and anticoagulant treatment was initiated with warfarin 5 mg/day tablets. His neurological examination performed on the tenth day of his hospitalization showed that lower right extremity paresis of 0/5 had improved to 3/5 and NIHSS score to 9. His discharge neurological examination showed that motor aphasia continued and the Modified Rankin Scale (MRS) value was 3. The patient was discharged on anticoagulant and antiaggregant treatment with an INR value of and asked to return for a follow-up visit a month later. Although he was referred to the interventional neuroradiology unit for stent placement, he was not found to be suitable for endovascular intervention indication and a stent was not inserted. Follow-up MRI scan four months later showed that ischemic stroke had completely settled and recanalization had not occurred (Images 4a and b).

Discussion

Carotid artery dissection is seen at a frequency of 2.5% among all causes of stroke. It is mostly seen between the ages of 35 and 50

and is the cause of 15-20% of all strokes below to age of 45 (2). Carotid artery dissection is thought to have an annual prevalence of 1.7 to 3.0/100,000. Internal carotid artery dissection may be spontaneous or traumatic, which is caused by a penetrating or blunt trauma targeting the area the carotid artery passes through (3). High speed penetrating traumas cause vessel injury directly or via bone fractures or impact on adjacent structures. Blunt traumas, on the other hand, may cause vessel injury through direct compression and detachment of the vessel wall or tissue fragments perforating the vessel wall (including bone fragments, especially in basilar skull fractures) (4).

Carotid artery dissection is seen in 1% of all patients with a blunt trauma and is seen in men and women equally. Although it is rare, it can be serious and may cause high mortality and morbidity rates if not treated (5). Cerebral ischemic events and mortality may occur following carotid artery dissection at a rate of 40-80% and 25-60%, respectively (6,7). Partial or complete occlusion are frequent (33%) lesions following blunt trauma. Penetrating injuries may cause arteriovenous fistula and pseudo-aneurysm in the long term (8). Lesions developing following a blunt trauma can be classified under 5 groups (Table 1) (9).

The main traumatic mechanism is a tear in the intima layer of the internal carotid artery and blood flowing from that area forward between the subintimal or subadventitial layers, and thus creating a pseudo-lumen (5). Hematoma may detach the intima and the internal elastic membranes and cause luminal narrowing or dissect the vessel wall within the subadventitial area and create an adventitial sac hanging outside the vessel wall. As a result, the true lumen of the carotid artery narrows, and cerebral hypoperfusion may develop due to this narrowing or embolic causes.

The bifurcation of the carotid artery is very sensitive to trauma at the level of the C1-C2 vertebra level. Dissections usually occur at the transition from the cervical to the petrous, and from the petrous to the cavernous portion of the carotid artery. The media and adventitia layers of the intracranial vessels are thinner and there is no external elastic laminae. The prognosis, mortality and morbidity of intracranial dissections are more severe. It is not always possible to distinguish between traumatic and spontaneous etiology. Therefore, arterial pathology, fibromuscular dysplasia, collagen tissue disorders including Marfan and Ehlers-Danlos syndrome, and other causes such as syphilis and systemic hypertension that could predispose to dissection should be investigated in these patients (10). Investigations assessing the etiology did not reveal any of the above conditions. One of the conditions predisposing internal carotid artery dissection in external mechanical injury is structural vessel wall pathologies such as tortuosity of the blood vessel.

Motor vehicle accidents are the most common traumatic causes (between 53 and 82%) of carotid dissections (3). Motor vehicle accidents commonly cause flexion, extension and rotation of the neck, the most common cause of blunt internal carotid arterial traumas. Therefore, patients admitted following a vehicle accident should be investigated with this condition in mind (11).

Clinical presentation in carotid artery dissection depends on the localization and the vessel involved, with varying clinical

findings. Symptoms are usually due to distal embolization or hypoperfusion. The findings most commonly accompanying carotid artery dissection are unilateral motor and/or sensory loss, aphasia, amaurosis fugax and related visual changes, incomplete Horner's syndrome, tendency to sleep, vertigo, dysphasia, seizures, transient ischemic attacks and pulsatile tinnitus. The most frequent local findings are swelling and local tenderness in the neck, and the most common symptoms are head and neck pain (12).

Clinical findings vary in spontaneous and traumatic internal carotid artery dissections. While focal cerebral ischemic symptoms are frequently seen in the traumatic group, unilateral headache accompanied by Horner's syndrome is the most common symptom in the spontaneously developing group (12).

Symptoms may arise instantly or onset may vary between 1 hour and a few weeks in patients presenting with a latent period (13). Symptoms are seen in 10%, 55% and 35% of patients developing a carotid artery dissection within 1 hour, 24 hours and after 24 hours of the trauma, respectively. There are reports in the literature of 11-58% of patients with internal carotid artery dissection having Horner's syndrome (14). Our patient's neurological examination did not reveal Horner's syndrome or amaurosis fugax, and his medical history showed that he developed confusion, weakness in his right side and aphasia 6 hours after the accident.

When the clinical picture suggests carotid artery dissection, imaging methods such as non-invasive doppler USG, MRI and CTA are used for definitive diagnosis. CTA allows detection of pathologies developing as a result of vessel injury in the craniocervical region (partial or complete occlusion, pseudoaneurisms, dissection, intimal flap and arteriovenous fistula) and is particularly valuable in diagnosis due to its ability to evaluate the extracranial internal carotid artery (11). The diagnostic value of carotid doppler USG is not clear because it is not efficient in demonstrating the skull and neck junction where dissections are most commonly seen. However, it may be used, due to its convenience in daily practice and high sensitivity and specificity in extracranial carotid artery pathologies. The sensitivity and specificity of cranial MRI and MRA in carotid artery dissection is over 95% (15). MRA is the most popular non-invasive method in the diagnosis of carotid artery dissection (16). FLAIR and Diffusion MRI provides information about the probable etiology (coexistence of hemodynamic and embolic etiology) (17). Internal

or external irrigation area lesions are seen at a rate of 16-50% in extracranial internal carotid artery dissection. Our patient had a similar radiologic image (Pictures 4a and b). Carotid digital subtraction angiography (DSA) is the gold standard in demonstrating carotid artery pathologies. DSA allows evaluating the pattern of internal carotid artery dissection (stenotic, occlusive and aneurismal), thrombo-embolic material, possible bilateral carotid involvement, accompanying possible vascular pathologies in various areas and cerebral hemodynamics. DSA must be used as a further investigation method in cases where vessel injury is suspected clinically, but non-invasive imaging methods are not adequate (18).

The treatment of carotid artery dissection is essentially medical; surgical procedures are rarely used. Currently the main therapy is supportive treatment and anticoagulant and antiaggregant treatments alone or in combination. The patient must be under hemodynamic control in the acute stage, bed rest must be recommended and neck movements must be restricted; the patient must be followed in an intensive care unit, if possible. Study data show that antiaggregant and anticoagulant treatments are effective in preventing the development of cerebral stroke following dissection. It is shown that stroke developed in 26% of non-treated, and only 4% of treated patients. The most preferred treatment modality in first-step treatment is heparinization. Antiaggregant treatment is preferred in patients who are not suitable for anticoagulation (19). There is no randomized study comparing the efficacy of anticoagulant and antiaggregant treatments. Antiaggregants are recommended in cases where the NIHSS score is 15 and above, where local compression syndromes not causing ischemic events accompany the main event and where the risk of bleeding has increased. On the other hand, anticoagulant treatment is recommended in cases where the NIH score is below 15, in pseudo-occlusion, in patients with a high signal intensity in doppler USG, and when there is more than one ischemia and thrombus in the same circulation area. Currently there is no clear data about the early treatment of cerebral infarct developing after dissection. There is also no clear answer to the question, "Antiaggregants or anticoagulants?" Studies on this subject ("International Study on Cervical and Intra-Cranial Artery Dissections, and the Cervical Artery Dissection in Stroke" çalışması) are continuing.

Table 1. Classification in blunt carotid injury

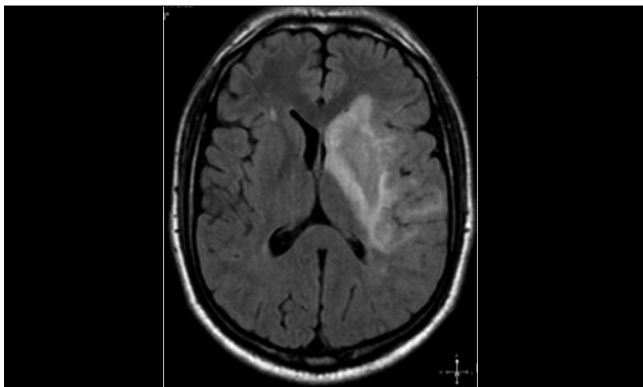
Class	Description
I	Luminal irregularity or <25% stenosis accompanying dissection (i.e. intimal injury)
II	Intramural hematoma, intraluminal thrombus or raised intimal flap accompanying dissection or >25% luminal stenosis
III	Pseudo-aneurism
IV	Occlusion
V	Transection accompanying free extravasation



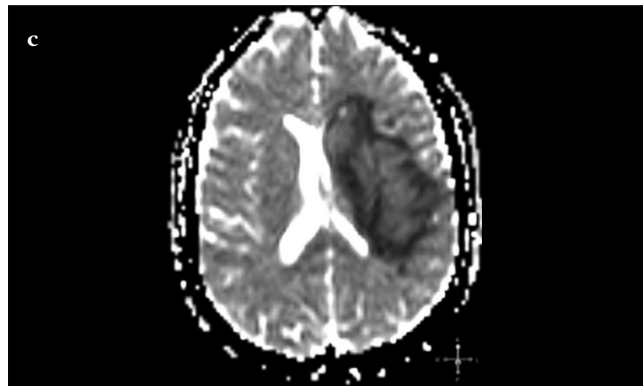
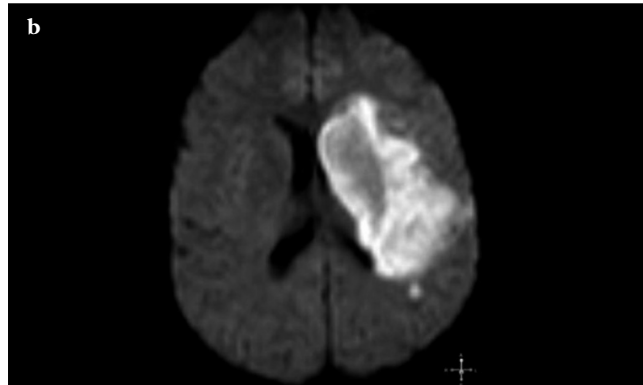
Images 1. Normal brain CT scan taken on Day 1.

In blunt carotid artery injury, if there is a contraindication to anticoagulant or antiaggregant treatment, endovascular treatment and carotid artery stent placement should be considered in case of expanding pseudoaneurism or extending dissection or lesions that cannot be reached by surgery (Table 1 and Figure 1). Currently the alternatives to endovascular treatment are balloon angioplasty, stent placement, coil embolization for dissecting aneurisms, stent placement with intraarterial thrombolysis and therapeutic occlusions. DuBose et al. report stent placement in penetrating or

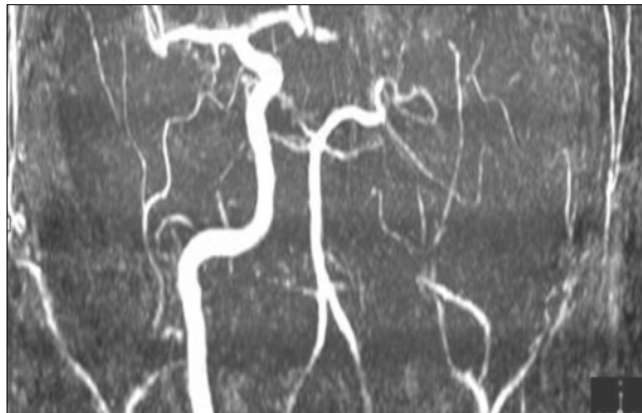
blunt carotid artery injury provides 80% recanalization and there is no mortality due to stent placement, but de novo neurologic deficit may develop following stent placement at a rate of 3.5% (16). Recent studies have reported differing opinions on carotid artery stenting. Cothren et al. found that 21% and 45% of the carotid artery dissection patients who had stenting developed complications and carotid artery stenosis, respectively. This rate was 5% in patients who did not have a stent but received



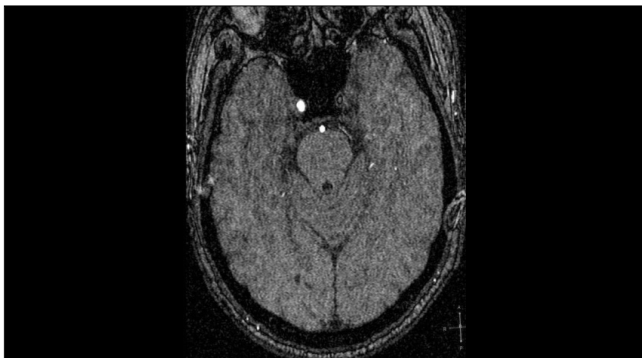
Images 2a. Cranial MR MR T2 FLAIR image; hyperintensity consistent with ischemia involving the mid cerebral arterial area in the left parietal lobe, globus pallidus, caudate nucleus, putamen and lateral thalamus in the inferior from the level of semioval centrum on the left.



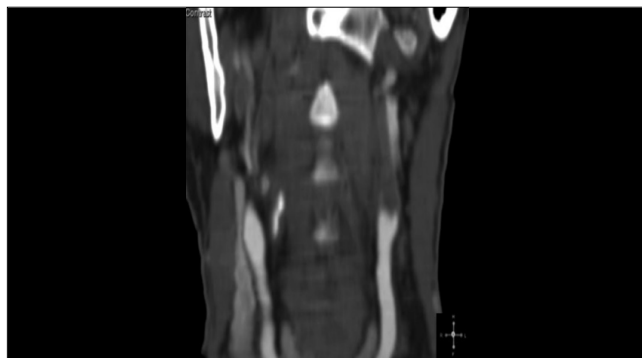
Images 2 b-c. Hyperintensity seen in the Diffusion Weighted MRI (DWI) consistent with ischemia and hypointensity seen in the "Apparent diffusion coefficient" (ADC) MRI image.



Images 3 a. Cranial Magnetic Resonance Angiography; no flow in the left internal carotid artery until the mid cerebral arterial bifurcation level and in the left mid cerebral artery.



Images 3b. Cranial Magnetic Resonance Angiography; occlusion in the left internal carotid artery.



Images 3c. Computerized neck tomographic angiography; complete occlusion in the left internal carotid artery from the bifurcation level.



Images 4a. Brain MR (T1) image taken 4 months later. Encephalomalacic area in the left MCA area.



Images 4b. Brain MR angiography taken 4 months later. Left Carotid artery cannot be seen.

antiaggregant or anticoagulant treatment. As a result, they concluded that carotid stenting alone cannot be accepted as a safer alternative to anticoagulant or antiaggregant treatment, but early anticoagulant or antiaggregant treatment is safe and effective. It has been emphasized that the role of carotid artery stenting is still not clear, and controlled studies are needed on this subject. Vascular specialists experienced in carotid stenting technique will ensure fine results during administration. There is no consensus on the appropriate treatment following endovascular procedure; although some authors recommend lifelong antiplatelet treatment, some advise continuing dual antiplatelet treatment for at least 3 months (clopidogrel 75 mg/day and aspirin 81 mg/day). The timing of stent placement in literature is also controversial. Whereas some authors believe delaying carotid stenting will decrease the risk of embolic and thrombotic side effects due to catheter manipulation in the acutely damaged artery, others think carotid artery stenting may be delayed at most 1 week (20).

In conclusion, in patients with a head and neck trauma, young stroke or normal CT scan, but neurologic and local findings, an etiology of internal carotid artery dissection must be suspected and early treatment must be initiated following diagnosis.

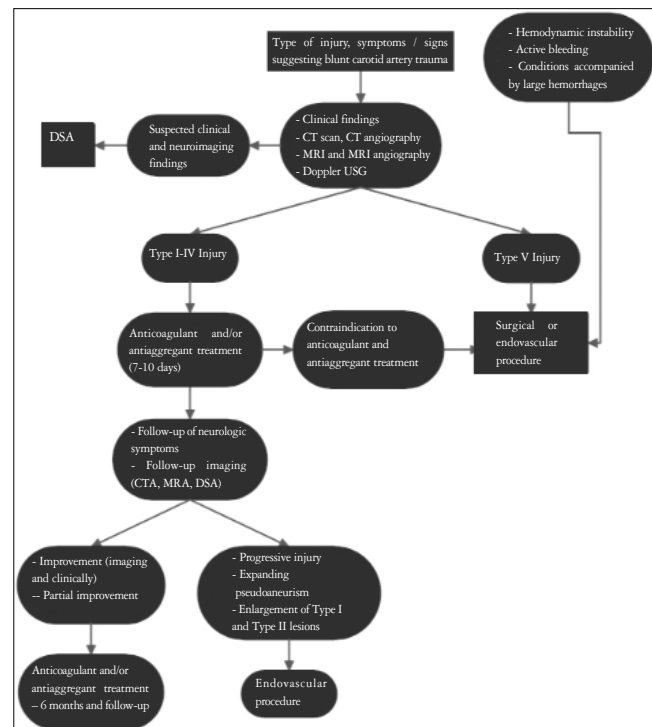


Figure 1. Treatment Algorithm in Blunt Carotid Trauma

References

- Zetterling M, Carlstrom C, Konrad. Internal carotid artery dissection. *Acta Neurol Scand* 2000;101:1-7.
- Putala J, Metso AJ, Metso TM, Konkola N, Kraemer Y, Haapaniemi E, et al. Analysis of 1008 consecutive patients aged 15 to 49 with first-ever ischemic stroke: the Helsinki young stroke registry. *Stroke* 2009;40:1195-203.
- Yang ST, Huang YC, Chuang CC, Hsu PW. Traumatic internal carotid artery dissection. *J Clin Neurosci* 2006;13:123-8.
- Öç M, Uçar Hİ, Doğan R. Management and Treatment modalities of cervical vascular injuries. *Turkiye Klinikleri J Cardiovasc Sci* 2006;18:125-31.
- Biousse V, Touboul PJ, D'Anglejan-Chatillon J, Levy C, Schaison M, Bousser MG. Ophthalmologic manifestations of internal carotid artery dissection. *Am J Ophthalmol* 1998;126:565-77.
- Bromberg WJ, Collier BC, Diebel LN, Dwyer KM, Holevar MR, Jacobs DG et al. Blunt cerebrovascular injury practice management guidelines: The East Association for the Surgery of Trauma. *J Trauma* 2010;68:471-7.
- Utter GH, Hollingworth W, Hallam DK, Jarvik JG and Jurkovich GJ. Sixteen-slice CT angiography in patients with suspected blunt carotid and vertebral artery injuries. *J Am Coll Surg* 2006;203:838-48.
- Yavas S, Saritas A, Kervan U, Bardakci H, Iscan HZ, Akgul A et al. False aneurysm of the internal carotid artery after a penetrating injury: case report. *Turkiye Klinikleri J Cardiovasc Sci* 2007;19:193-6.
- Biffi WL, Moore EE, Offner PJ, Burch JM. Blunt carotid and vertebral arterial injuries. *World J Surg* 2001;25:1036-43.
- Walton KA, Buono LM. Horner syndrome. *Curr Opin Ophthalmol* 2003;14:357-63.
- Guyot LL, Kazmierczak CD, Diaz FG. Vascular injury in neurotrauma. *Neurol Res* 2001;23:291-6.
- Nunnink L, Abu-Zidan F. Accidental carotid artery injury caused by a horse rope. *Br J Sports Med* 2003;37:460-1.
- Testerman GM. Bilateral carotid stenting after a motor vehicle crash. *South Med J* 2010;103:253-5.

14. Lévy C, Laissy JP, Raveau V, Amarenco P, Servois V, Bousser MG et al. Carotid and vertebral artery dissections: three-dimensional time-of-flight MR angiography and MR imaging versus conventional angiography. *Radiology* 1994;190:97-103
15. Provenzale JM, Sarikaya B. Comparison of test performance characteristics of MRI, MR angiography, and CT angiography in the diagnosis of carotid and vertebral artery dissection: a review of the medical literature. *AJR Am J Roentgenol* 2009;193:1167-74.
16. DuBose J, Recinos G, Teixeira PG, Inaba K, Demetriades D. Endovascular stenting for the treatment of traumatic internal carotid injuries: expanding experience. *J Trauma* 2008;65:1561-6.
17. Payton TF, Siddiqui KM, Sole DP, McKinley DE. Traumatic dissection of the internal carotid artery. *Pediatr Emerg Care* 2004;20:27-9.
18. Fleck SK, Langner S, Baldauf J, Kirsch M, Rosenstengel C, Schroeder HW. Blunt craniocervical artery injury in cervical spine lesions: the value of CT angiography. *Acta Neurochir* 2010;152:1679-86.
19. Mayberry JC, Brown CV, Mullins RJ, Velmahos GC. Blunt carotid artery injury: the futility of aggressive screening and diagnosis. *Arch Surg* 2004;139:609-12.
20. Cothren CC, Moore EE, Ray CE Jr, Ciesla DJ, Johnson JL, Moore JB, Burch JM. Carotid artery stents for blunt cerebrovascular injury: risks exceed benefits. *Arch Surg* 2005;140:480-5
21. Hershberger RC, Aulivola B, Murphy M, Luchette FA. Endovascular grafts for treatment of traumatic injury to the aortic arch and great vessels. *J Trauma* 2009;67:660-71.