Scholars Journal of Applied Medical Sciences (SJAMS)
Abbreviated Key Title: Sch. J. App. Med. Sci.
ISSN 2320-6691 (Online)
ISSN 2347-954X (Print)

A Unit of Scholars Academic and Scientific Society, India
www.saspublisher.com

Anesthesiology

Right Carotid Artery Dissection Associated With Safety Belt

Emine Aslanlar, Zeynep Cura, Omur Ilban, Muhammed Emin Zora, Ozkan Onal*, Jale Bengi Celik
Medical Faculty, Anesthesia and Reanimation Department, Selcuk University, Turkey

Case Report

***Corresponding author**
Ozkan Onal

Article History
Received: 10.02.2018
Accepted: 22.02.2018
Published: 30.03.2018

DOI:
10.21276/sjams.2018.6.3.45

Abstract: As a results of high energy traumas on head and neck, blunt carotid artey dissection (CAD) may occur. The incidence of CAD among all patients with blunt trauma is estimated to be 0.08–1.2%. Motor vehicle accidents are the cause of carotid dissections at rates varying between 53% and 82%. CAD may lead to ischemic stroke and subsequent disability or death. Case fatality has been reported in up to 31% of the patients with CAD, and severe disability in up to 56% of the survivors. To prevent these serious complications, early recognition of this injury is important. In the present case, the patient with general trauma after motor vehicle accident was brought to emergency service with open consciousness. Glascow Coma Score was 13 and no pathology was seen in brain and cervical tomography images. Due to blunt abdominal injury, owing to sudden fall in hemoglobin values, the patient underwent emergency operation by general surgery team. In postoperative period, he was transferred to anesthesia intensive care unit. Anisocoria in pupillae was seen on 3rd day of admission. Diffusion MR investigation was carried out. Widespread diffusion restriction suggested carotid lesion. Cerebellar tonsillar herniation was present. In repeat cerebral angiography, contrast filling was not observed in both internal carotid arteries. The patient died on the 24th day of admission. In patients admitted to emergency service after motor vehicle accidents, artery dissection should always be kept in mind. What is challenging in such cases is to diagnose CAD before the onset of neurological symptoms. Latent interval between the injury and emergence of symptoms may be misleading. It should be borne in mind that, particularly in patient followed under sedation, diagnosis may bemused easily as neurological symptoms can not be observed clearly.
Keywords: carotid artey dissection; motor vehicle accidents; blunt trauma; delayed neurological symptoms.

INTRODUCTİON

As a results of high energy traumas on head and neck, blunt carotid artey dissection (CAD) may occur. The incidence of CAD among all patients with blunt trauma is estimated to be 0.08–1.2% [1]. Motor vehicle accidents are the cause of carotid dissections at rates varying between 53% and 82% [2]. CAD may lead to ischemic stroke and subsequent disability or death. Case fatality has been reported in up to 31% of the patients with CAD, and severe disability in up to 56% of the survivors [3,4]. To prevent these serious complications, early recognition of this injury is important [5].

Serious cervical hyperextension, particularly in combination with complex and dislocated midfacial or mandible fractures, a GCS < 6, closed head injury, skull base fractures involving the carotid canal or fracture of the petrous bone, Le Fort II or III fracture, or a cervical spine fracture are specific risk factors for the occurrence of CAD [3,6–9].

In dissection, with the impact of trauma, tear develops in intima layer of internal carotid artery. Blood moves along the artery from this region and produces a false lumen in subintimal or subadventitial layers [10]. Hematoma may lead true lumen to be narrowed by separating intima and internal elastic membrane or causes an adventitial pouch from vessel wall within subadventitial area. In addition, tear in, intima produces a thrombogenic surface, which gives rise to platelet aggregation and embolus development secondary to thrombus formation [11]. Consequently, cerebral perfusion may develop in association with the narrowing of true lumen or embolic causes.

Symptoms such as unilateral motor and/or sensation loss secondary to distal embolization or hypoperfusion, aphasia, amorozis fugax or similar visual changes, incomplete Horner syndrome, tendency to sleep, vertigo, dysphasia, convulsion, syncope, transient ischemic attack (TIA) and pulsatile tinnitus may occur. Swelling on the neck and local tenderness are the most common local findings while head and

neck pain are the most common symptoms [12]. As our patient was kept sleeping due to unstable hemodynamics after undergoing operation for abdominal injury, neurological symptoms were masked and could not be recognised until the development of anisocoria.

Latent period between the occurence of injury and emergence of cerebrovascular symptoms is the characteristic feature of these traumas [11] and it may range from a few hours to several weeks [13]. In 80% of patients, there is no marked neurological clinical picture at first presentation [11]. Of patients with carotid artery dissection, 10% becomes symptomatic within the first hour of trauma, 55% within first 24 hours of trauma and 35% after 24 hours [14]. Consciousness was open in the present case at presentation and there was no neurological complaint.

CASE REPORT

A 21 year old patient with general trauma after motor vehicle accident was brought to emergency service with open consciousness. Glascow Coma Score was 13 and no pathology was seen in brain and cervical tomography images. Due to blunt abdominal injury, widespread bleeding was observed intrabdominally in abdominal CT. Owing to sudden fall in hemoglobin values, the patient underwent emergency operation by general surgery team. A cut was observed in mesentery artery and vein and jejunum and ileum resection was carried out. In postoperative period, he was transferred to anesthesia intensive care unit in intubated position and with dopamine and adrenaline support. Light reflex was negative and papillae middilated .On the 3rd day of admission, hemodynamics improved and sedation was stopped. Anisocoria in pupillae was seen on 3rd day of admission. Diffusion MR investigation was carried out. The patient was consulted with radiology and neurosurgery departments. Widespread diffusion restriction suggested carotid lesion. Cerebellar tonsillar herniation was present and interventional radiology team carried out angiographic examination and placed supraaortic stent to right carotid artery with traumatic dissection. On 4th day, papillae were fixed dilated and light reflex was -/- . In repeat cerebral angiography, contrast filling was not observed in both internal carotid arteries. The patient died on the 24th day of admission.

DISCUSSION

In CAD, as imaging methods, Doppler ultrasonography, MRA and CT are employed. CT makes it possible to detect pathologies such as partial or complete occlusion, pseudoaneurisma, dissection, intima flap or arteriovenous fistula and has an important role in making the diagnosis of dissection, especially as it is able to evaluate extracranial internal carotid artery [15]. Carotid Doppler ultrasonography may be used since its is practical and has high specifity and sensitivity in extracranial carotid artery pathologies, but it is not efficient at demonstrating the junction of neck and skull where dissections occur most commonly. Cranial MRG and MRA has sensititivity and specifity over 95% in carotid artery dissection [16]. MRA, is the most popular noninvasive method in the diagnosis of carotid arety dissection [17]. FLAIR and diffusion MRG sequences helps to reveal the probable etiology of the stroke developing in association with internal carotid artery dissection (coexistence of hemodynamic and embolic etiology) [18]. Gold standard in demonstrating carotid artery pathologies is "Digital Subtraction Angiography (DSA)". DSA, makes it possible to evaluate the pattern of internal carotid artery dissection (stenotic, occlusive and aneurism) thrombo-embolic material, probable bilateral carotid involvement, accompanying vascular pathologies at diferent areas, and cerebral hemodynamics. DSA should be used as an advanced investigation method in cases when vessel injury is suspected clinically but can not be be adequately detected by non invasive imaging methods [19].

The treatment of carotid artery dissection is basically medical. Surgical methods are employed rarely. There are studies demonstrating that early recognition of CAD and institution of treatment with anticoagulants and antithrombocytes decrease the number of cerebral ischemic events with improvement in outcome, but they remain to be corroborated [20,21]. In addition, whether anticoagulant or antiplatelet treatment should be initiated and how long the treatment should last is also controversial. In these patients, trauma associated closed head trauma, solid organ injury or accompanying pelvic fractures may hinder the early initiation of anticoagulation treatment [22].

In blunt carotid artery injury, endovasular treatment and stenting of carotid artery may be considered in conditions when anticoagulant or antiaggregant treatment is counterindicated, and in the presence of expanding pseudoaneurism, or progressing dissection, and lesions not accessible by operation [17]. Similarly, in the present case, stent was placed on carotid artery.

During motor vehicle accidents, dissection may occur due to the sudden pressure exerted on the neck by safety belt. In patients admitted to emergency service after motor vehicle accidents, artery dissection should always be kept in mind. What is challenging in such cases is to diagnose CAD before the onset of neurological symptoms. Latent interval between the injury and emergence of symptoms may be misleading. It should be borne in mind that, particularly in patient followed under sedation, diagnosis may bemused easily as neurological symptoms can not be observed clearly.

REFERENCES

1. Biffl WL, Moore EE, Offner PJ, Burch JM. Blunt carotid and vertebral arterial injuries. World J Surg. 2001;25:1036–43.
2. Yang ST, Huang YC, Chuang CC, Hsu PW. Traumatic internal carotid artery dissection. J Clin Neurosci 2006;13:123-8.
3. Hughes KM, Collier B, Greene KA, Kurek S. Traumatic carotid artery dissection: a significant incidental finding. Am Surg. 2000;66:1023–7.
4. Kerwin AJ, Bynoe RP, Murray J, Hudson ER, Close TP, Gifford RR, Carson KW, Smith LP, Bell RM. Liberalized screening for blunt carotid and vertebral artery injuries is justified. J Trauma. 2001;51:308–14.
5. van Wessem KJP, Meijer JMR, Leenen LPH, van der Worp HB, Moll FL, de Borst GJ. Blunt traumatic carotid artery dissection still a pitfall? The rationale for aggressive screening. Eur J Trauma Emerg Surg. 2011;37:147–54.
6. Biffl WL. Diagnosis of blunt cerebrovascular injuries. Curr Opin Crit Care. 2003;9:530–4.
7. Miller PR, Fabian TC, Croce MA, Cagiannos CC, Williams JS, Vang M, Qaisi WG, Felker RE, Timmons SD. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. Ann Surg. 2002;236:386–95.
8. De Borst GJ, Slieker MG, Monteiro LM, Moll FL, Braun KPJ. Bilateral traumatic carotid artery dissection in a child. Pediatr Neurol. 2006;34:408–11.
9. Cothren CC, Moore EE, Ray CE Jr, Johnson JL, Moore JB, Burch JM. Cervical spine fracture patterns mandating screening to rule out blunt cerebrovascular injury. Surgery. 2007;141:76–82
10. Biousse V, Touboul PJ, D'Anglejan-Chatillon J, Levy C, Schaison M, Bousser MG. Ophtalmologic manifestations of internal carotid artery dissection. Am J Ophtalmol 1998;126:565-77
11. Biffl WL, Moore EE, Ryu RK, Offner PJ, Novak Z, Coldwell DM, Franciose RJ, Burch JM. The unrecognized epidemic of blunt carotid arterial injuries: early diagnosis improves neurologic outcome. Ann Surg. 1998;228:462–70
12. Nunnink L, Abu-Zidan F. Accidental carotid artery injury caused by a horse rope. Br J Sports Med 2003;37:460-1.
13. 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. Guyot LL, Kazmierczak CD, Diaz FG. Vascular injury in neurotrauma. Neurol Res 2001;23:291-6.
16. 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.
17. 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.
18. Payton TF, Siddiqui KM, Sole DP, McKinley DF. Traumatic dissection of the internal carotid artery. Pediatr Emerg Care 2004;20:27-9.
19. 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.
20. Edwards NM, Fabian TC, Claridge JA, Timmons SD, Fischer PE, Croce MA. Antithrombotic therapy and endovascular stents are effective treatment for blunt carotid injuries: results from longterm followup. J Am Coll Surg. 2007;204:1007–13.
21. Cothren CC, Moore EE, Ray CE, Ciesla DJ, Burch JM. Screening for blunt cerebrovascular injuries is cost-effective. Am J Surg. 2005;190:849–54
22. Arthurs ZM, Starnes BW. Blunt carotid and vertebral artery injuries. Injury. 2008;39:1232–41.