The Heart Surgery Forum #2008-1020
11 (4), 2008 [Epub August 2008]
doi: 10.1532/HSF98.20081020

Online address: http://cardenjennings.metapress.com

Carotid Artery Disease Development after Traumatic Injury Inflicted by a Ram's Horn

I. Haberal,[1] M. Kaplan,[2] Y. Zorman,[1] T. Aksoy,[1] E. Erturk,[1] M. Akyildiz[1]<<Q1>>

[1]Maltepe University School of Medicine, Maltepe, Turkey; [2]Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey

ABSTRACT

A 36-year-old man presented for treatment of headache, paresthesis, and weakness of his right arm and leg. Examination revealed an atherosclerotic plaque in the left common carotid artery, lying to the left internal carotid artery and resulting in 99% stenosis. The plaque was removed successfully by carotid endarterectomy. Pathological examination of the tissue confirmed the diagnosis of an atherosclerotic plaque. Detailed investigation of risk factors led to the conclusion that the stenosis of the carotid artery was due to blunt trauma caused by an injury that had occurred 30 years previously when the patient was gored by a ram's horn.

CASE REPORT

A 36-year-old male patient presented with headache, paresthesis, and weakness of his right arm and leg. He reported that nearly a month previously he began experiencing episodes of loss of power in his right arm and facial paralysis that lasted for only 4 to 5 minutes and remitted spontaneously. One week later the patient suffered recurrent right hemiplegia and disarthria, which lasted for 10 minutes. He sought treatment at a neurology outpatient clinic, where bilateral carotid doppler and vertebrobasillary ultrasonography were performed. Ultrasonography revealed a hypoecoic (unstable) plaque in the bulbus of the left common carotid artery, lying next to the internal carotid artery. The plaque measured 4 mm at the thickest part. The maximum velocity value was increased focally to 172 cm/sec, and a high-resistance flow pattern was present distally. The vertebrobasillary system was normal. Further investigation with carotid digital subtraction angiography revealed 50% stenosis in the left common carotid artery and 99% stenosis in left internal carotid artery. Treatment options offered to the patient were surgery or placement of an endovascular stent with a high risk.

Received February 19, 2008; received in revised form March 28, 2008; accepted April 7, 2008.

Correspondence: Ismail Haberal, Şemsettin Günaltay cad. Derya Ap. No.10/2 D:34 Erenköy Kadikö, Istanbul, Turkey; +90 216 3999750- 2109; fax : +90 216 3684784 (e-mail: doktorhaberal@hotmail.com).

An important event in the patient's medical history was that nearly 30 years previously, when he was 5 years old, a ram gored him in the neck with its horns. Only minimal bleeding occurred with this trauma and was stopped easily with external compression, and a scar of approximately 3 cm was present on his neck (Figure 1). The remainder of the patient's history was unremarkable.

Preoperative cranial magnetic resonance imaging revealed no intracerebral pathology. Because the patient complained of recurrent neurological symptoms, we performed echocardiographic screening for emboli resulting from cardiac pathology. Echocardiography results for heart spaces and cardiac wall movements and thicknesses were normal. Interventricular and interatrial septa were intact. No thrombus or vegetations were seen. The patient's brother was receiving antihyperlipidemic medications for hyperlipidemia, so the case patient's serum cholesterol profile and lipoprotein (a) levels were investigated and found to be normal.

Surgical Technique

After the completion of routine preoperative tests, the patient underwent a left carotid endarterectomy with local anesthesia. We removed a 2.5-cm long plaque that started at the bifurcation of common carotid artery and extended to the internal carotid artery. The artery was sutured primarily with 5.0 prolene (Figure 2).

Histopathological Evaluation

The removed tissue was analyzed at the pathology laboratory. The result was reported as atherosclerotic plaque with intensive calcification (Figure 3).

Follow-up

The patient experienced no complication after surgery and was discharged from the hospital on postoperative day 3. At a follow-up examination 4 weeks later, the patient had no neurological deficits, and the incision site was clean and without signs of infection.

A control carotid doppler ultrasonography examination revealed 2 plaques with fibrofatty structure and smooth surfaces, located in the bulbus of the left common carotid artery. This finding was not associated with significant clinical symptoms or stenosis. The measured velocities in both the internal and common carotid arteries were normal bilaterally.

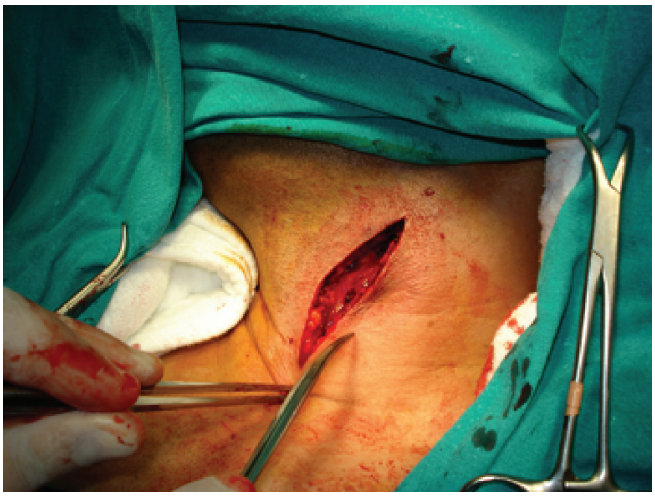

Figure 1. Visible scar over the left carotid artery from blunt trauma inflicted on the patient by a ram's horn.

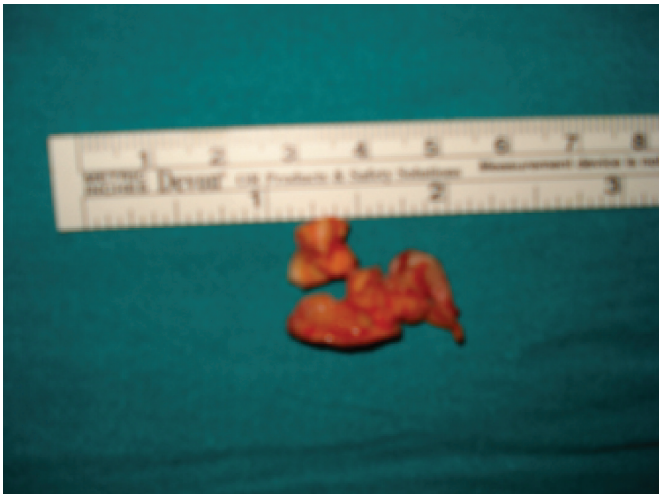

Figure 3. Material removed during endarterectomy.

DISCUSSION

Severe neurological complications and early mortality are often associated with carotid artery disease resulting from carotid artery trauma. Rates of 30% early mortality and 42% permanent neurological deficits were reported for a series of 96 cases [Mears 1988], and Perry et al [1980] reported 40% mortality. In a study that showed 38% mortality and 82% severe neurological deficits in a series of 51 cases, Yamada et al [1967] observed that in 94% of patients there was a latent interval before the onset of neurological deficits. This interval could be a few hours or a few days. The rate for onset of the neurological symptoms was 6% a few hours after the trauma, and 35% after the first day. In our case patient we concluded that the atherosclerosis was attributable to posttraumatic intimal damage. In this case the latent period for the onset of neurological symptoms resulting from carotid artery disease was nearly 30 years.

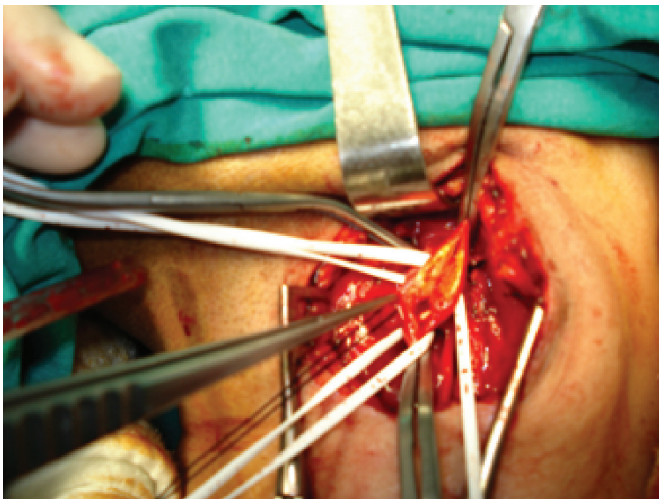

Figure 2. Carotid endarterectomy.

Blunt carotid artery trauma may cause severe permanent or reversible neurological deficits and has a high mortality rate. Neurological symptoms develop after a latent period and are usually irreversible. Craniocervical trauma may lead to the development of hematoma, monoplegia, hemiplegia, facial paralysis, disarthria, or Horners syndrome. The diagnosis of conditions occurring from blunt carotid artery trauma is usually delayed because a latent period precedes the onset of symptoms. The lengths of the latent periods reported in the literature vary from a few hours to a few days, and in our case the latent period lasted 30 years.

After trauma, intimal damage develops at the wall of the artery, and this damage in turn activates the platelets, monocytes, and T lymphocytes, resulting in arterial wall proliferation and contraction in smooth muscle cells. The final stage of traumatic intimal damage is the development of a fibrous plaque. Symptoms usually occur because of complications such as rupture of the plaque, bleeding into the plaque, thrombosis, or emboli [Calldwell 1948; Little 1969; Towne 1972]. In our case patient, we concluded that the reason for the central nervous system symptoms was progressive embolization of the thrombosis in the carotid artery. Because of the level of the cerebral embolization in this case, the symptoms occurred contralaterally. Cerebral embolizations may also cause bilateral cortical infarcts and focal retinal ischemia.

Arteriography is the best method for diagnosis in patients with posttraumatic carotid artery disease. To evaluate cerebral pathology, cranial computerized tomography and magnetic resonance imaging should also be performed.

In a series of 96 patients studied by Yamada et al [1967], lesions in the bifurcation of the carotid artery and internal carotid arteryZwere seen after nonpenetrating cervical trauma, which resulted in stenosis in the internal carotid artery with a rate of 38%. These results are in agreement with the findings for our case.

Endarterectomy performed to treat carotid artery disease caused by blunt trauma may lead to complications ranging in severity from minimal to severe neurological deficits [Leonard 1980]. These complications may be attributable to surgical technique or the nature of the plaque. In our case patient the procedure was performed successfully and without any complications.

In patients whose symptoms are due to neurological damage occurring before carotid endarterectomy, clinical status rapidly improves in a few hours or days after the operation.

In conclusion, blunt trauma may cause carotid artery stenosis. In this patient the stenosis developed years after the trauma caused by the ram's horns. It is important to be aware that trauma of this type may cause intimal damage leading to carotid artery stenosis.

REFERENCES

Calldwell HW, Hadden FC. 1948. Carotid artery thrombosis: report of eight cases due to trauma. Ann Int Med 28:1132-42.

Little JM, May J, Lamond S, Vanderfield GK. 1969. Traumatic thrombosis of the internal carotid artery. Lancet 2:926-30.

<<Q2>>Mears GD, Leonard RB. 1988. Blunt carotid artery trauma: a case report. J Emerg Med 6:281-4.

Perry MO, Snyder WH, Thal ER. 1980. Carotid artery injuries caused by blunt trauma. Ann Surg 192:74-7.

Towne JB, Neiss DD, Smith JW. 1972. Thrombosis of the internal carotid artery following blunt cervical trauma. Arch Surg 104:565-8.

Yamada S, Kindt GW, Youmans JR. 1967. Carotid artery occlusion due to nonpenetrating injury. J Trauma 7:333-42.