Early diagnosis and successful management of traumatic carotid artery dissections require a high index of clinical suspicion. The diagnostic study of choice is cerebral arteriography. In this paper, 24 cases of traumatic carotid artery dissection are described. Presenting signs and symptoms include Horner's syndrome, dysphasia, hemiparesis, obtundation, and monoparesis. Patients detected early with mild neurological deficits fared well with treatment, while those with profound neurological deficits and delayed diagnoses had poor outcomes. Aggressive nonsurgical treatment is advocated including anticoagulation therapy for prevention of progressive thrombosis and arterial occlusion and/or distal arterial embolization with resultant cerebral ischemia. Direct surgical thromboendarterectomy is considered to carry high morbidity and mortality rates.

Key Words · carotid artery · trauma · arteriography

Illustrative Case Report

This 40-year-old woman was involved in a motor-vehicle accident. She was admitted to the local medical facility where her initial neurological examination was normal. Shortly after admission, she was noted to have decreased movement on the right side and she was transferred to the Elvis Presley Memorial Trauma Center for treatment. Her trauma injuries included closed head injury, facial fracture, bilateral pneumothoraces with flail chest, liver laceration, splenic laceration, compound tibia and fibula fractures, right humeral fracture, cardiac contusion, and multiple lacerations.

Upon arrival at the trauma center, she was found to have aphasia and a right hemiparesis in addition to the injuries described above. No carotid bruits were present. Computerized tomography of the brain was normal. Prior to laparotomy, she underwent urgent cerebral arteriography which confirmed bilateral carotid artery dissections and a distal intraluminal thrombus in the carotid siphon on the left side (Fig. 1). The patient was taken to the operating room where she underwent emergency laparotomy for repair of liver laceration, removal of the ruptured spleen, bilateral tube thoracostomies, and immobilization of long-bone fractures. At the conclusion of these procedures, the patient was anticoagulated with intravenous heparin and closely monitored by the trauma and neurosurgical services. Her course was stable with no new neurological deficits. She was subsequently discharged to the rehabilitation center with oral anticoagulation for 6 months. Follow-up arteriography confirmed recanalization of the dis-
Traumatic carotid artery dissection

Fig. 1. Cerebral arteriograms. Left: Anteroposterior view showing tapering stenosis of cervical internal carotid artery (ICA) to dissection point (1), a pseudoaneurysm (2), anddelayed intracranial ICA flow (3). Right: Lateral view showing retained contrast material in the cervical intimal dissection (1) and intraluminal thrombus in the carotid artery siphon (2).

Our center has adopted an aggressive approach to the diagnosis of this clinical entity in the trauma setting. Any trauma patient who presents with a focal neurological deficit unexplained by findings on CT brain scans, spinal injury, or peripheral nerve injury is evaluated with urgent cerebral arteriography. This approach has led to the diagnosis of traumatic carotid artery dissection in 24 patients in the 4-year period between December, 1984, and December, 1988. All patients were evaluated and treated at the Elvis Presley Memorial Trauma Center. This appears to be the most concentrated experience with this problem thus far reported. We have treated 115 patients who were suspected of having carotid artery dissections and therefore underwent arteriography; 24 (20.8%) proved to have dissections.

Age

As in most trauma patients, the majority of these patients are much younger than the usual patient with a cerebrovascular accident. Eighteen (75%) of the 24 patients were between the ages of 15 and 35 years (average 33 years). The mechanism of injury was most often a motor-vehicle accident but other modes of injury included motorcycle accidents, three-wheeler recreational vehicle accidents, airplane crashes, and personal altercations such as strangling or fist fight.

Sex and Race

Fourteen male and 10 female patients were diagnosed with traumatic carotid artery dissections. Since male patients are more commonly victims of trauma, the relatively high number of female patients raises the question of whether females may be more vulnerable to this arterial injury. There were 17 Caucasian patients and seven blacks.

Signs and Symptoms

Presenting signs and symptoms of patients were generally cerebral ischemic events in the distribution of the affected artery or arteries. These included coma, lethargy, hemiparesis, aphasia, and Horner's syndrome. Glasgow Coma Scale scores ranged from 4 to 15, with the average score being 11. No patient was found to have an audible neck bruit on the distribution of the dissected artery. Two patients had spinal fractures without cord injury, one in the cervical region and the other in the upper thoracic region.

Diagnostic Studies

All patients underwent CT scans of the head within four hours of admission. Seventeen (71%) of the 24 patients had normal initial scans; of these, 12 (70%) subsequently showed hypodense areas on CT scanning consistent with cerebral infarctions. Eleven patients had right carotid artery dissections, seven had left carotid artery lesions, and six had bilateral carotid artery dissections. The follow-up period in this series ranged from 3 weeks to 3 years.

Treatment

Treatment was individualized depending upon the anatomy of the lesion. The majority of the patients were immediately heparinized. Subsequent to an initial 3-week period of heparinization, 11 patients were changed to oral anticoagulation with Coumadin (sodium warfarin) for approximately 6 months. Seven patients were placed on antiplatelet therapy alone. No patient underwent surgery. Complications of heparinization included gastrointestinal hemorrhage in two patients and an acute subdural hematoma in one patient. No complication of long-term treatment has been identified, and no subsequent ischemic events have occurred. Thus far no complication of a pseudoaneurysm has been identified.

Results of Treatment

Outcome has related to the severity of the presenting ischemic event. Therapy was considered successful if
deterioration or subsequent ischemic events were prevented. Other factors determining outcome were coexisting injuries and conditions. Three patients have normal neurological examinations, six have minimal neurological sequelae and are independent, nine have moderate residual deficits and are disabled (four of whom are less than 1 year postinjury), one is in a persistent vegetative state, and five have died (Table 1). Two patients died of concomitant primary brain trauma, one of sepsis, and two of massive stroke due to bilateral carotid artery dissections and total occlusions.

Discussion

A review of the literature shows that there is significant variation in the treatment modalities for this entity. Yamada, et al.,17 reviewed 52 cases including their own surgical case and showed that better results were obtained with surgical treatment than without surgery. Biller, et al.,2 reported on a small series, yet their results were more favorable with supportive therapy. Batzdorf, et al.,1 described a series in which better results were obtained by either supportive therapy or by trapping of the dissected artery than by direct thromboendarterectomy of the dissected vessel. Other reports of surgical repair and thromboendarterectomy have generally been unfavorable.8,13 Stringer and Kelly,14 advocated conservative therapy and demonstrated good results in all of their six patients. Other authors have also recommended conservative therapy.5 All reported series have emphasized the importance of a high index of suspicion for carotid dissection in the trauma patient and have recommended arteriography for evaluation.

The results of this study demonstrate that carotid artery dissection is more common than has been recognized. This diagnosis should be considered in patients experiencing deceleration injuries, particularly in patients with focal neurological deficits. Patients with focal neurological deficits and normal CT scans of the brain should not be designated as having focal contusions, concussions, brain-stem injuries, or peripheral nerve stretch injuries unless cerebral arteriography has ruled out a traumatic carotid artery dissection. Urgent cerebral arteriography should be utilized when this diagnosis is entertained. Treatment is directed toward arresting the propagation of thrombus, thereby reducing or preventing distal arterial embolization and/or occlusion. It is considered that early diagnosis and early intervention with anticoagulation are likely to reduce secondary injury and, as a result, to decrease the risk of associated morbidity and mortality. Asymptomatic pseudoaneurysms may be observed; however, if symptoms of distal embolization from the aneurysm sac, bleeding, or enlarging neck mass occur, the treatment outlined by Sundt, et al.,15 should be considered. This would entail resection of the pseudoaneurysm or trapping of the internal carotid artery segment with or without extracranial-intracranial bypass. We believe that thromboendarterectomy in the setting of acute traumatic carotid artery dissection is related to excessive morbidity and mortality rates, and is likely to have poorer results than the treatment approach of anticoagulation and therapy as presented here.

Acknowledgment

We express our appreciation to Ms. Christie Beaty and Ms. Lyse Magness for assistance in manuscript preparation.

References


Manuscript received November 14, 1988.
Accepted in final form June 21, 1989.

This paper was presented at the Annual Meeting of the American Association of Neurological Surgeons, Toronto, Ontario, Canada, April 24-28, 1988.

Address reprint requests to: Clarence B. Watridge, M.D., Department of Neurosurgery, University of Tennessee, 956 Court Avenue, Room A-202, Memphis, Tennessee 38163.