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Cluster Headache, Hemicrania, and Other Head Pains: Morbidity of Carotid Endarterectomy

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SUMMARY Carotid endarterectomy has become a widely used approach to the treatment of cerebrovascular disease. In spite of increasing experience, a significant and varied morbidity remains attached to the procedure. A poorly recognized complication is postoperative headache.

In a series of 57 endarterectomies in 50 patients, 24 patients experienced postoperative headaches encompassing the entire spectrum of vascular headaches: nonspecific diffuse headaches, severe hemicranias, cluster headaches occurring early and delayed, chronic paroxysmal hemicranias, carotidynia, and Eagle's syndrome.

Five patients had hemicranias, and all were homolateral to the endarterectomy. Therefore, we hypothesize that the spontaneously occurring hemicranias, the counterparts of postsurgical headache syndromes, also may be due to some overt or occult injury or disease of the carotid vessels or carotid sheaths in the region from the carotid bifurcation to the base of the skull.

STENOTIC LESIONS of the common carotid artery or the internal carotid artery near the bifurcation are well recognized causes of cerebrovascular ischemia. Carotid endarterectomy is gaining increasing recognition as a surgical approach in the prophylaxis and treatment of stroke. The surgical procedure is complicated by mortality and morbidity.¹

Aside from the general postoperative morbidity attendant upon any major surgical procedure, the structures at risk in the surgical field in carotid endarterectomy include other major vessels, nerves (including cranial nerves), and supportive bony and soft tissue structures. Some attention has been directed to local anatomic involvement and concomitant morbidity.^{1, 2}

Cranial nerve involvement can vary from mild hoarseness to difficulty in swallowing and aspiration as surgical manipulation injures the hypoglossal, superior laryngeal, and glossopharyngeal nerves, and the recurrent branch of the vagus nerve.^{1, 2} Also at risk are the greater auricular nerve and the marginal mandibular branch of the facial nerve.¹ Horner's syndrome, from involvement of the carotid sheath, is also recognized.¹

Those postoperative complications that develop some time after carotid endarterectomy or that could suggest a specific syndrome to the neurologist might well escape the immediate attention of the vascular surgeon.

Postoperative complications to carotid endarterectomy that are less well recognized and yet are of considerable interest both clinically and theoretically, are the development of particular patterns of headache. We have found limited reference in the literature to severe headache following carotid endarterectomy or similar surgery involving the carotid sheath. Toole et al.¹ attributed postoperative headache to vasodilation. Leviton³ reported 2 cases of severe self-limited headache following endarterectomy and attributed the

headache to a transient disturbance in cerebral autoregulation.

We observed that some patients developed headaches following carotid endarterectomy or related surgery which were often indistinguishable from the classically described cluster type of vascular headache. This led us to speculate on the etiology of cluster headaches in the general population and to postulate a unifying hypothesis as to the pathophysiology of other vascular headaches and hemicranias.

In the past 5 years 50 consecutive patients were submitted to 57 endarterectomies; 1 additional patient had a right neck dissection for metastatic disease. The patients were evaluated as to their preoperative and postoperative neurological status. Among 24 patients who presented with significant headache in the postoperative period, 7 had mild to moderate, poorly localized, diffuse headaches, 3 developed nonspecific, acute headaches, 9 noted onset of severe headache with retrobulbar pain homolateral to the site of surgery; 1 patient had postoperative severe retrobulbar pain without significant associated headache, 3 patients presented with headaches following retrograde angiography preparatory to endarterectomy, and 1 patient postoperatively complained of severe neck pain alone, not related to wound healing.

Among the 24 patients with headache, 5 qualified as severe hemicranias homolateral to the site of surgery. One patient 2 days postoperatively had a typical cluster attack (Case 1). Another had delayed cluster headaches (Case 2). Another had chronic hemicranias (Case 3). Another had intermittent left hemicranias and a 3-week episode of carotidynia (Case 4) and the fifth patient had severe chronic paroxysmal, irreducible hemicrania (Case 5).

Case Histories

Case #1

A 56-year-old white man was admitted 4-11-77 for elective repair of a left inguinal hernia. He was in good general health but had a background of rheumatoid arthritis with involvement of the knees and a possible myocardial infarction 11 years before. General ex-

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amination and routine laboratory evaluation including chest x-ray and ECG were unremarkable except for a left carotid bruit without any evidence of neurological involvement either by history or examination. There was no history of migraine or even common headaches.

The patient had an uneventful hernia repair. Postoperatively, ophthalmodynamometry revealed marked difference in ophthalmic artery pressures and a Doppler examination was consistent with occlusion of the right internal carotid artery and severe stenosis of the left internal carotid artery. A bilateral carotid arteriogram confirmed occlusion of the right internal carotid artery just above the bifurcation with collateral circulation to the right cerebral hemisphere coming from the external carotid artery and ophthalmic system. There was a severe, 95% or greater, ulcerated stenosis at the origin of the left internal carotid artery. An EEG was normal.

On 5-3-77 the patient went a left carotid endarterectomy; no shunt was utilized. An ulcerated plaque was removed resulting in a good, wide, patent internal carotid artery. The immediate postoperative course was unremarkable and a postoperative Doppler examination on the left carotid artery was within normal limits.

Two days postoperatively in the morning the patient had an acute onset of very severe, nonthrobbing, left-sided headache with nausea. Blood pressure was elevated, 172/108. For the next 5 days he had frequent recurrences of severe left frontotemporal headaches with retrobulbar radiation of acute onset, nonthrobbing, and of short duration. The left eye conjunctiva was injected, however no pupillary asymmetry was noted. The patient remained neurologically stable and a repeat Doppler was again within normal limits on the left. On 5-7 he was started on propranolol 10 mg qid. On 5-10 the severe headache seemed to abate, blood pressure returned to normal, and the patient was discharged. When seen as an outpatient on 5-16 he was generally comfortable but with residual left temporal headache. He was readmitted in October, 1977 for right knee synovectomy; no further mention of headache was found in the records.

Case #2

A 57-year-old white man had 2 previous VA Madison admissions for multiple vascular disease manifestations, cardiac and peripheral. Between 1973 and 1976 he had an aorta-femoral bypass and had been submitted to a double coronary bypass. The patient had adult onset of diabetes, essential hypertension and Type IV hyperlipoproteinemia.

He had had bilateral carotid bruits since 1970 but there was no clinical evidence of neurological dysfunction. When readmitted in November, 1976, however, he had a 4 to 5 months history of episodes of dizziness and numbness of the left hand. Examination showed a minimal right facial paresis, some paresthesia of the right forehead, and the left pupil was slightly smaller than the right. Bilateral loud carotid bruits were again

heard, left louder than right. The rest of the neurological examination was unremarkable. Carotid arteriograms revealed bilateral internal carotid stenosis, right more severe than left. The patient had never had any particular headache complaint.

A right carotid endarterectomy was performed on 12-10-76. The right internal carotid vessel was very small with an outside diameter of 3 mm; pre-endarterectomy blood flow was 60 ml per minute. Shunting was impossible due to the small vessel size but the endarterectomy was accomplished uneventfully and post endarterectomy blood flow was 150 ml per minute. The postoperative course was unremarkable and the patient was discharged a week after surgery.

He was readmitted 4-25-77 with a chief complaint of severe right forehead pains since February, 2 months postendarterectomy. He described them as severe, right-sided, radiating to the right eye and even to the jaw, occurring 2 to 3 times a week acutely, nonthrobbing, and of 1 to 2 hours duration. During that hospitalization the significant findings were elevated blood sugar (198 to 531 mg %), variable glycosuria, and elevated erythrocyte sedimentation rate (ESR) of 47 mm/hr. A temporal artery biopsy was unremarkable but the patient was given a trial treatment of prednisone 100 mg daily for 7 days, then tapered off, with improvement in the headaches. ESR at discharge 5-27 was 27 mm/hr. When seen a month later he had no complaint of headache; however, 3 months later some general headaches were present but on subsequent visits headache was no longer present.

Case #3

A 67-year-old white man was admitted November, 1975 to the VA Hospital, Madison, with a history of episodic right arm weakness. He had a history of longstanding hypertension. He had been submitted to surgery for an abdominal aortic aneurysm but in his past history no evidence of headaches was recorded.

The admission examination was unremarkable except for poor vision in the left eye, the result of an old injury and not related to the present illness. There was no evidence of any residual neurological deficit; however, a carotid bruit was heard on the left. A Doppler examination showed a significantly decreased flow on the left and a retrograde arteriogram revealed stenosis and plaque ulceration in the left internal carotid. A left carotid endarterectomy was performed on 12-23-75. The postoperative period was uneventful and the patient was discharged a few days later on December 29. There was no record of morbidity or headache during that hospitalization, but on 1-15-76 the patient was seen in another hospital complaining of severe left-sided headaches with left retro-orbital, ear, facial, and even jaw radiation; he also complained about pain along the healed incision.

On 4-27-76 he was admitted for the second time with the chief complaint of left-sided headache and retro-orbital pain with left facial tenderness which apparently had been present since a few days after the

endarterectomy. He appeared to be in acute distress from the pain; blood pressure was 190/90 and pulse was 80. General examination was unchanged from previous evaluation. Neurological examination showed an alert, oriented, cooperative man without any obvious neurological deficits. Laboratory values were unremarkable except for an elevated ESR of 48 mm/hr. Repeat carotid Doppler ultrasound studies revealed a patent left internal carotid artery. Isotope flow study of the carotid system showed right-sided perfusion dominance with relative underperfusion of the left cerebral artery territory. EEG was normal.

Supportive care and local injections of Xylocaine to the trigger areas in the left jaw and neck failed to control the patient's pain more than 1 day at a time. However, a course of prednisone 60 mg daily for 4 days with tapering off for a week seemed to bring about relief of the severe pain with only intermittent headaches from then on. Following discharge in April, 1976, the patient did not have any recurrence of headache or face pain.

Case #4

A 63-year-old white man was admitted to the Madison VA Hospital in November, 1977 for a sudden onset of aphasia without any other associated distress or deficits. The patient had been well until 1969 when he experienced episodes of left eye blindness. In 1971 a left carotid endarterectomy was done. Later that year he had onset of numbness of the right side of the body. A repeat transfemoral retrograde arteriogram was complicated by arterial emboli requiring an endarterectomy in the right leg. In 1974 he was re-evaluated in another hospital because of recurrent numbness of the right extremities but apparently no further therapy for the numbness was administered.

On admission to our hospital the patient's general examination was unremarkable. Neurological examination showed a severe nonfluent aphasia with apparent good comprehension. No other deficits were noted except for a left carotid bruit, decreased left radial and brachial pulses, and a left subclavian bruit. Doppler studies were consistent with bilateral carotid disease. EEG showed delta focus in the left temporal area. Isotope brain scan was normal. Following some recovery of speech the patient gave a history, corroborated by his wife, of onsets of episodic left hemiparesis since the left endarterectomy in 1971 which had persisted for years. Following the 1977 admission the patient suddenly developed severe sustained left hemiparesis with pain behind the left eye, the face, and occasionally the jaw, and associated with tenderness over the left neck and on swallowing. This severe hemiparesis persisted for 3 weeks, then receded without any residual.

Case #5

A 49-year-old man had surgery in November, 1976, for carcinoma in the right retromolar trigone. A biop-

sy in June, 1977, showed recurrent invasive carcinoma. He was treated with 3800 Rads preoperatively and in October, 1977, he was submitted to a composite resection of the right mandible. The patient had had a myocardial infarction in 1975 with subsequent stable angina. The patient did not have any history of headaches.

Two weeks after the composite resection of the mandible with right neck dissection, the patient presented with an acute onset of severe right supra-orbital headache lasting from a few minutes to 45 minutes and recurring 3 to 6 times daily. The post-operative course was otherwise unremarkable. The neurological examination was negative except for the expected tissue changes in the right neck and a 20-year-old right eye injury with no relevance to the present illness. The patient continued to have severe right cluster headaches daily and he was admitted to Neurology following discharge from the ENT service. While under neurological observation he continued to have daily severe headaches. Propranolol, which the patient had been taking prior to surgery for his heart disease, did not seem to be of any benefit for the headaches, and ergot preparations were contraindicated by the presence of coronary artery disease. Finally, after nearly 4 months of chronic paroxysmal hemicrania, the patient entered a remission phase and became comfortable. However, a month later in March, 1978, he presented with a recurrence of severe right paroxysmal headaches occurring as many as 8 times a day, lasting from a few minutes to 30 or 40 minutes, radiating to the right eye and still not responsive to propranolol.

Discussion

Cluster headaches and hemicranias are presently classified as variants of vascular headaches.⁴ An exhaustive comparison of cluster headache with chronic paroxysmal hemicrania, and the more common vascular headache variants classified under the heading of migraine, is beyond the scope of this report.

Some authorities consider cluster headache and migraine to be different manifestations of the same disease,^{5, 6} while others suggest on clinical and biochemical grounds that separation of the clinical entities is justified.⁷⁻⁹ The episodic nature of the pain in cluster headache, the occasional dilation of extracranial vessels during an episode, and partial relief by ergotamine suggest a relationship between cluster headache and the varied presentations of migraine. However, the brevity of the painful episodes, the compression in time of attacks, i.e., the "clustering" of attacks, the presence of autonomic phenomena, the invariable unilaterality of pain and the overwhelming male predominance (4 to 1) in cluster as compared to female predominance in migraine, all attest to the possibility of distinct etiologies in cluster and migraine.⁶⁻¹⁰ In addition, Anthony and Lance^{7, 9} have recently delineated distinctive biochemical events in migraine and cluster headaches. Histamine levels in blood rise rapidly during an attack of cluster head-

ache whereas the rise is small and slow to occur in migraine attacks.⁷ Furthermore, plasma serotonin levels show a mild increase during an attack of cluster headache while in migraine there is a sudden, statistically significant fall during the headache.^{7, 9} To date the evidence is inconclusive, but a case can be made on clinical and biochemical grounds for the separation of migraine and cluster headache as distinct entities.

A key diagnostic feature of classical migraine is the familial occurrence and the alternating site of pain. But cluster headache is not familial; the location of pain is stereotyped, always unilateral, occurring in cycles with symptom-free intervals varying from weeks to years.⁶ It is particularly interesting from an epidemiological point of view that the population at risk for cluster headache appears to be assertive, aggressive, athletic males¹⁰ who can be assumed to be at greater risk from trauma.

The pattern of headaches occurring in our patients following surgery involving the carotid vessels and our review of the related literature suggests to us the hypothesis that cluster headache and related hemicranias are a direct result of some pathologic state involving the carotid sheath or the vessel itself in the region of the common carotid and the carotid bifurcation. In reviewing the incidence and characteristics of our postoperative headache cases, it becomes evident that these headaches cover the entire spectrum generally classified under vascular headaches with the exception of the specific genetic classical migraine. Patients presented with diffuse headaches, nonspecific hemicranias, severe hemicranias with radiation to eye, face, jaw, ear, of either a chronic sustained nature or episodic paroxysmal hemicranias or combination of chronic hemicranias with superimposed bursts of acute pain. Two patients presented with what appeared to be typical cluster headache and, finally, 1 patient seemed to correspond to the syndrome of carotidynia with headache, face pain, neck tenderness, and pain on swallowing. All patients presented were post-endarterectomy cases except for one who had a neck dissection for carcinoma of the right retromolar trigone, this case further providing evidence that surgical trauma to the neck in the territory of the carotid vessels can produce the same morbidity as carotid endarterectomy. During endarterectomy, dissection of the carotid vessels is often pursued to the base of the skull, and the tip of the mastoid styloid and its attachments, which are in close proximity to the carotid sheaths, might be traumatized and produce the symptoms of the Eagle syndrome of the styloid process carotid artery type^{11, 12} which, in fact, is indistinguishable from carotidynia¹³ and paroxysmal episodic hemicrania.^{14, 15} One patient (Case #4) who had a hemicrania with eye, face, and jaw pain also had pain on swallowing associated with neck pain. The Eagle syndrome thus completes the series of vascular headache which can be seen.

The association of various clinical patterns of headache or head pain with structural disease in the neck is not new. Fay reported in 1932 the occurrence of head pain following electrical stimulation of the carotid sheath.¹⁶ More recently, Vijayan¹⁷ has reported headache following soft tissue injury to the neck.

Raskin and Prusiner have recently discussed a possible relationship between vascular headache and local involvement of the carotid arteries. They point out that similar etiologies might be inferred from the similar response of carotidynia and vascular headaches to the same drugs.¹³

It is our hypothesis that cluster headaches and other severe hemicranias are a clinical manifestation of circumscribed carotid vessel disease frequently posttraumatic in nature. This is supported by our observations on our own patients, all of whom may be considered to have suffered significant trauma to the carotid during surgery, and none of whom had previously suffered this type of headache. Supportive evidence arises from consideration of the population at risk for the usual typical cluster headache, typically aggressive and athletic males, presumably more subject to trauma than the population at large.

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