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Arterial surgery for sporadic hemiplegic migraine: preliminary results

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Abstract

My aim was to report for the first time (to my knowledge) the successful treatment of 3 patients with sporadic hemiplegic migraine by surgical cautery of terminal branches of the external carotid artery under conscious sedation. Since the operations (between 1 and 2 years) none of the patients have had further attacks of migraine or hemiplegia. This preliminary report suggests that for patients with sporadic hemiplegic migraine with a confirmed arterial component, surgical cautery of selected terminal branches of the external carotid artery may be effective treatment.

Keywords: Sporadic hemiplegic migraine; External carotid artery; Vascular surgery; Migraine surgery

Introduction

Sporadic hemiplegic migraine is defined as migraine with an aura including motor weakness but with no first- or second-degree relative having an aura that included motor weakness.[1]

Attacks have the same clinical characteristics as those of familial hemiplegic migraine, namely motor weakness and at least one of the following: fully reversible visual symptoms including positive features (such as flickering lights, spots, or lines) or negative features (such as loss of vision), or both; fully reversible sensory symptoms including positive features (such as pins and needles) or negative features (such as numbness), or both; or fully reversible dysphasic speech disturbance.

They also have at least two of the following: at least one symptom of aura that develops gradually over 5 min or more, or different symptoms of aura that occur in succession over 5 min or more, or both; each symptom of aura lasts more than 5 min and less than 24 h; and headache that fulfills criteria B–D for migraine without aura,[1] begins during the aura, or follows the onset of aura within 60 min.

Because sporadic hemiplegic migraine is rare there are no clinical trials of treatment available so the treatment is based on empirical data, the personal experience of the treating neurologist, and it involves trial and error. Acetaminophen and non-steroidal anti-inflammatory drugs are often the first choice of treatment for an acute attack. An effective treatment for the severe and often prolonged symptoms of aura is more warranted, but currently no such treatment is available.[2] Prophylaxis can be considered when the number of attacks exceeds 2/month, or when severe attacks pose a greater burden that requires reduction of severity and frequency. Propranolol,[3] flunarazine,[4] naloxone,[5] and verapamil[6] have all been reported to have a good therapeutic effect, but the studies were all too small to achieve a confirmed significant effect.[7] Sodium valproate, lamotrigine, and acetazolamide have also been suggested, and while less evidence is available for prophylactic treatment with topiramate, candesartan,

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and pizotifen, these drugs can also be considered.[2] None of these treatments, however, have been shown to be particularly effective.

Methods

To find out whether the pain originates in the extracranial vessels, it is necessary to examine the patient while the pain is present. The diagnosis can be confirmed if the pain diminishes when blood flow to the painful area is interrupted by digital compression of the relevant vessels. When the pressure is removed the pain returns, usually within a few seconds. The vessels most frequently associated with the pain are: the main trunk of the superficial temporal artery; the frontal and parietal branches of the superficial temporal artery; and the occipital, maxillary, angular, and posterior auricular arteries. In most patients, multiple vessels are involved in the pain and all must be cauterized. All 3 patients were examined during an attack.

In patients with unilateral pain, the scalp vessels are still cauterized bilaterally, because there are extensive side-to-side anastomoses of some of the terminal branches of the external carotid artery,[8] and cautery of the vessels only on the affected side is sometimes ineffective.[9] It is only necessary to cauterize the maxillary artery on the affected side in side-locked unilateral headache. The techniques for cauterizing these vessels have been described elsewhere.[9,10]

Case reports

Case 1

A 38-year-old woman had had left sided migraine since childhood. Initially her main trigger was the hot sun, but when she reached puberty, hormone fluctuations and stress began to be more important. The first attack that was accompanied by paralysis occurred in 2003 when she was 30 years old. She experienced a stabbing pain on the left side of her head and fainted. When she regained consciousness the left side of her face was paralyzed. She could not recall how long the facial paralysis lasted, but it did recover spontaneously. Neurological examination and magnetic resonance imaging (MRI) were within normal limits. Shortly afterwards she had another, severe, attack, which rendered her unconscious until the following day. When she did regain consciousness she was disorientated for a few hours. Neurological examination showed no neurological deficit, and she was discharged with a diagnosis of migraine with aura.

As the years progressed the aura became worse, and 2 years after the first attack she started becoming completely paralyzed on her left side during attacks. When the pain lifted, so too did the paralysis. During her pregnancy she experienced no migraines, but after the birth of her son the migraines returned and became steadily worse. She had constant headaches, but she also had 2 or 3 migraines/week. As the pain got worse, she would develop a complete scotoma of the left eye, she was unable to speak, and the left hand and left foot became paralyzed. After a particularly severe episode she was prescribed Maxalt (rizatriptan), which she used daily to prevent the migraines.

The patient had been prescribed Maxalt (rizatriptan) elsewhere, despite the fact that the triptans are contraindicated in sporadic hemiplegic migraine.[11] She used it regularly because it was the only medication that relieved her pain. As the triptans are potent constrictors of the extracranial vessels,[12] we made a clinical examination of the terminal branches of the external carotid artery during an attack to find out whether the pain was indeed extracranial. Digital compression of the superficial temporal, frontal, occipital, and internal maxillary arteries all reduced the pain. As we know that the pain of migraine that originates in the extracranial terminal branches responds well to cautery of the involved vessels,[9] and as the patient was aware that digital compression of the extracranial arteries relieved her pain, she elected to have cautery. The involved vessels were cauterized on 26 September 2011, and she has had no further attacks 2 years later.

Case 2

A 35-year-old woman had her first attack of migraine as a teenager. She had also had several isolated episodes of visual aura. The first attack of sporadic hemiplegic migraine occurred when she was in her early 20 s. She described it as follows: “the first hemiplegic migraine attack happened when I was at work on the ‘phone. I had been feeling odd all morning but at the time did not recognize this as aura. I was finding it difficult to find the right words whilst on the ‘phone, and then the ‘phone fell out of my (left) hand. Shortly after that I lost the right side of my visual field”. She was taken to hospital, but during the journey the paralysis in the arm stopped and the headache began. The next attack was a few months later, and then a few weeks after that a continuous cycle established itself of a day of aura, a day or more of pain, a day of exhaustion, and then possibly a day without symptoms, and then back to the aura. Despite drug treatment by her consultant neurologists her condition worsened, and she also developed chronic facial pain.

She experienced multiple sensory disturbances. Her visual disturbances she described as follows – “things looking too bright/sharp, violet spots, pin point white flashes in the periphery of my vision, partial loss of visual field either completely or to a broken glass or patterned distortion, Alice in Wonderland syndrome”. She also experienced sensitivity to multiple sounds: “I get very disorientated if, for example, the radio is on in the kitchen and the TV is on in the living room. If you add people having a conversation to the mix I find it unbearable”; to smell: “nausea from strong smells – in particular coffee, cigarette smoke, synthetic floral smells like air