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Extracranial Internal Carotid Artery Vasospasm as a Cause of Vision Loss

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Abstract

Background: Although intracranial vasospasm causing cerebral ischemia is a well-known entity in neurosurgical practice, extracranial internal carotid artery vasospasm is an extremely rare cause of ischemia and concomitant vision loss.

Case Description: A 38-year-old man presented with loss of vision in the left eye. Magnetic resonance imaging and magnetic resonance angiography revealed a narrowing of the left internal carotid artery beyond the carotid bulb, and computed tomography angiography demonstrated significant stenosis of the left internal carotid artery at the level of C2. A plan for internal carotid artery stenting was aborted when an intraoperative angiogram performed 24 hours after initial conventional angiogram showed near complete resolution of the stenosis. Thus a diagnosis of extracranial internal carotid artery vasospasm was confirmed.

Conclusion: Extracranial internal carotid artery vasospasm is a rare cause of vision loss that can be identified and distinguished from other etiologies based on its characteristic location and timeframe of resolution.

Keywords

Extracranial vasospasm, Ischemic stroke, Internal carotid artery vasospasm

Introduction

Intracranial vasospasm is a fairly well-recognized cause of cerebral ischemia, and is most commonly seen as a complication of subarachnoid hemorrhage [1]. Less common, however, is the diagnosis of extracranial internal carotid artery (ICA) vasospasm as a cause of cerebral ischemia. To date, only a few such cases have been reported. Herein, we present a case of ICA stenosis attributable to extracranial vasospasm that resolved after 24 hours.

Case Report

A 38-year-old man presented after awakening with loss of vision in the left eye. He denied pain, photophobia, weakness, or sensory deficits. The patient had a history of intermittent supraventricular tachycardia, and a family history of diabetes, coronary artery disease, and peripheral vascular disease. He was not on antiplatelet or anticoagulation medication at the time of presentation. The patient presented

to Will's Eye Hospital in Philadelphia, where he was diagnosed with left branch retinal artery occlusion and referred to Jefferson Hospital for Neuroscience to be evaluated by the stroke service.

On examination, the patient was alert, oriented, with fluent speech and intact memory. His neurological exam was notable for a left medial upper visual field deficit present only in the left eye. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) of the head and neck was performed and demonstrated no acute infarct or mass effect (Figure A), but revealed a narrowing of approximately 50% in the left ICA beyond the carotid bulb (Figure B). No flow gap was appreciated to suggest a high-grade stenosis on MRA. Computed tomography angiography (CTA) was also performed and demonstrated a focal area of stenosis in the left ICA measuring 78% at the level of C2 with a mild prominence of soft tissue in this location. A conventional cerebral angiogram was then performed 48 hours after the onset of symptoms that confirmed a 70% stenosis and revealed a possible dissection flap of the left ICA (Figure C). The patient was given 600 mg clopidogrel and 650 mg aspirin in preparation for the placement of a left ICA stent on the following day. Upon beginning the stenting procedure, the follow-up angiogram performed 24 hours after the initial angiogram revealed a near-complete resolution of the stenosis with only 10% residual stenosis (Figure D), therefore a stent was not placed.

The patient was discharged on clopidogrel, aspirin, and rosuvastatin and instructed to follow-up with his primary care physician and stroke clinic. At 3 months clinical follow-up he remains at his neurologic baseline without any recurrence of symptoms. The patients will have another MRA of the neck for long-term follow-up to assess for maintenance of resolution of any carotid stenosis.

Discussion

Given this patient's age, location of stenosis, and lack of atherosclerotic disease, a diagnosis of acute dissection that had resolved after the administration of antiplatelet therapy was suggested. However, given the timeframe over which the stenosis resolved, in addition to the absence of blood in the arterial vessel wall on MRI, this scenario was deemed unlikely. Therefore, a diagnosis was made of extracranial ICA vasospasm that resolved within 24 hours.

In young patients with ischemic stroke, arterial dissection can be confused with extracranial ICA vasospasm [2]. The length of time over which the stenosis resolves can be used to differentiate the two diagnoses. Spontaneous dissections of the ICA resolving after antiplatelet or anticoagulation therapy have been examined in recent studies. Nedeltchev et al. followed 249 patients with spontaneous ICA dissection who were treated with antiplatelet or anticoagulation therapy. They found that complete recanalization is most likely to occur within the first 6 months after the initial onset of symptoms [3]. In similar studies, Rao et al. reported 80% luminal recovery at a mean interval of 11.2 months [4], Steinke et al. reported 68% luminal recovery at a mean interval of 51 days [5], and Sturzenegger et al. reported 63% luminal recovery at a mean interval of 15 months [6]. Since our patient's stenosis resolved within 24 hours, dissection of the artery as a potential etiology was excluded.

Intracranial artery vasospasm is a common cause of cerebral ischemia. It is encountered as a complication of subarachnoid hemorrhage [1], and is also associated with reversible cerebral vasoconstriction syndrome (RCVS) or cerebral vasculitis [7]. Vasospasm of extracranial vessels is less commonly reported, though it has been associated with migrainous headaches [8], intraoperative mechanical manipulation [9], and ergot poisoning [10]. Furthermore, vasospasm that occurs secondary

to vessel wall manipulation during conventional angiography is a common occurrence. Mechanically-induced vasospasm has been found to occur in approximately 40% of cases where a guiding catheter was advanced during diagnostic angiography [11]. It has been suggested, however, that extracranial carotid artery vasospasm could occur spontaneously without a mechanical trigger [12]. Due to the relatively short window of time with which to observe extracranial vasospasm, it is perhaps under-diagnosed as a cause of cerebral ischemia [13]. However, recognition of extracranial vasospasm as an important cause of ischemic disease has been demonstrated in many recent cases.

While an association with migrainous headaches has been noted in previous cases, our case is one of non-migrainous vasospasm. Other such cases have been reported [2, 12-17]. It is important to note that the recurrent extracranial ICA vasospasm seen in these cases occurs in the intermediate portion of the ICA, which is also the case in our patient. Though the reason for this pattern is unknown, it has been postulated that the preferential occurrence of extracranial vasospasm in this location is due to a distinct pattern of sympathetic vasomotor innervation. As such, sympathetic blockade as a treatment of recurrent extracranial ICA vasospasm has been used with success [16]. However, to date, a standardized treatment is not available.

Our patient represents a case of rapidly resolving stenosis that first appeared without an apparent trigger. A similar case to that of our patient has been reported. Yoshimoto et al. described a 39-year-old female who experienced multiple episodes of amaurosis fugax over the course of 2-3 years. The patient had no predisposing factors or history of headaches. Coronary angiography showed the presence of vasospastic angina. MRA revealed an area of stenosis in the cervical segment of the left ICA. On cerebral digital subtraction angiography performed the next day, the stenosis was no longer seen. Follow-up MRA confirmed the absence of stenosis. Given the timeframe of the resolution of the stenosis, conditions such as arterial dissection, fibromuscular dysplasia, and atherosclerosis were ruled out as potential causes of the stenosis. In that case, the patient was diagnosed with idiopathic vasospasm of the extracranial ICA [2].

Figures

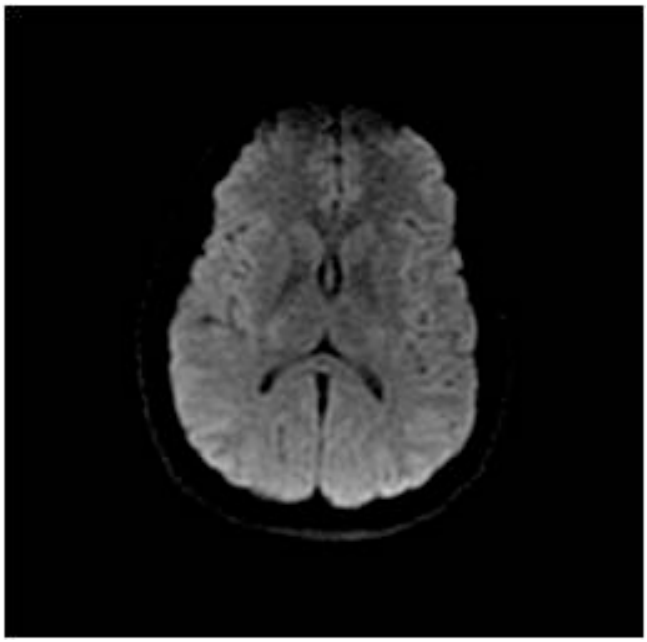


Figure A: Initial MRI (DWI sequence) showing no cerebral infarct.

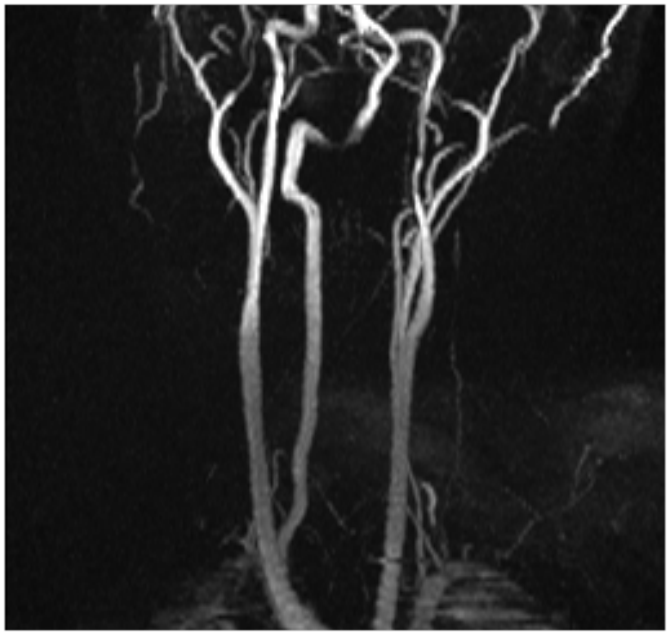


Figure B: Initial MRA showing significant Left ICA stenosis

In summary, we have described a case of extracranial ICA stenosis resolving within a 24 hour period. Identification of the seemingly characteristic location of extracranial vasospasm in combination with information regarding the timeframe of the resolution of the stenosis can aid in distinguishing extracranial vasospasm from other etiologies as the cause of stenosis.

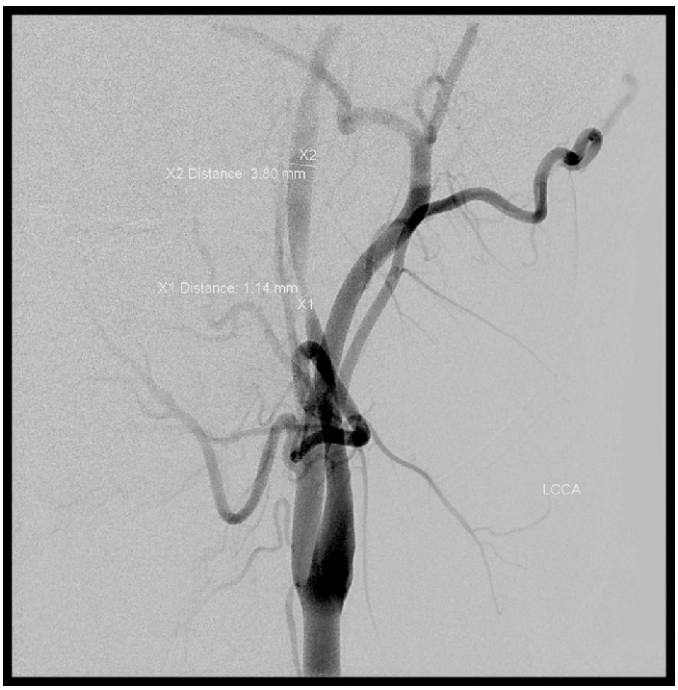


Figure C: Initial DSA (frontal projection) of Left ICA showing 70% stenosis.

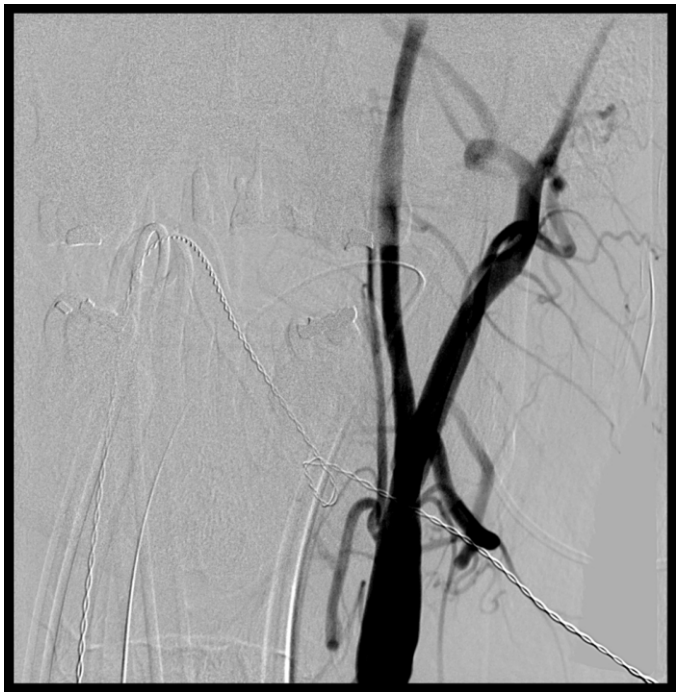
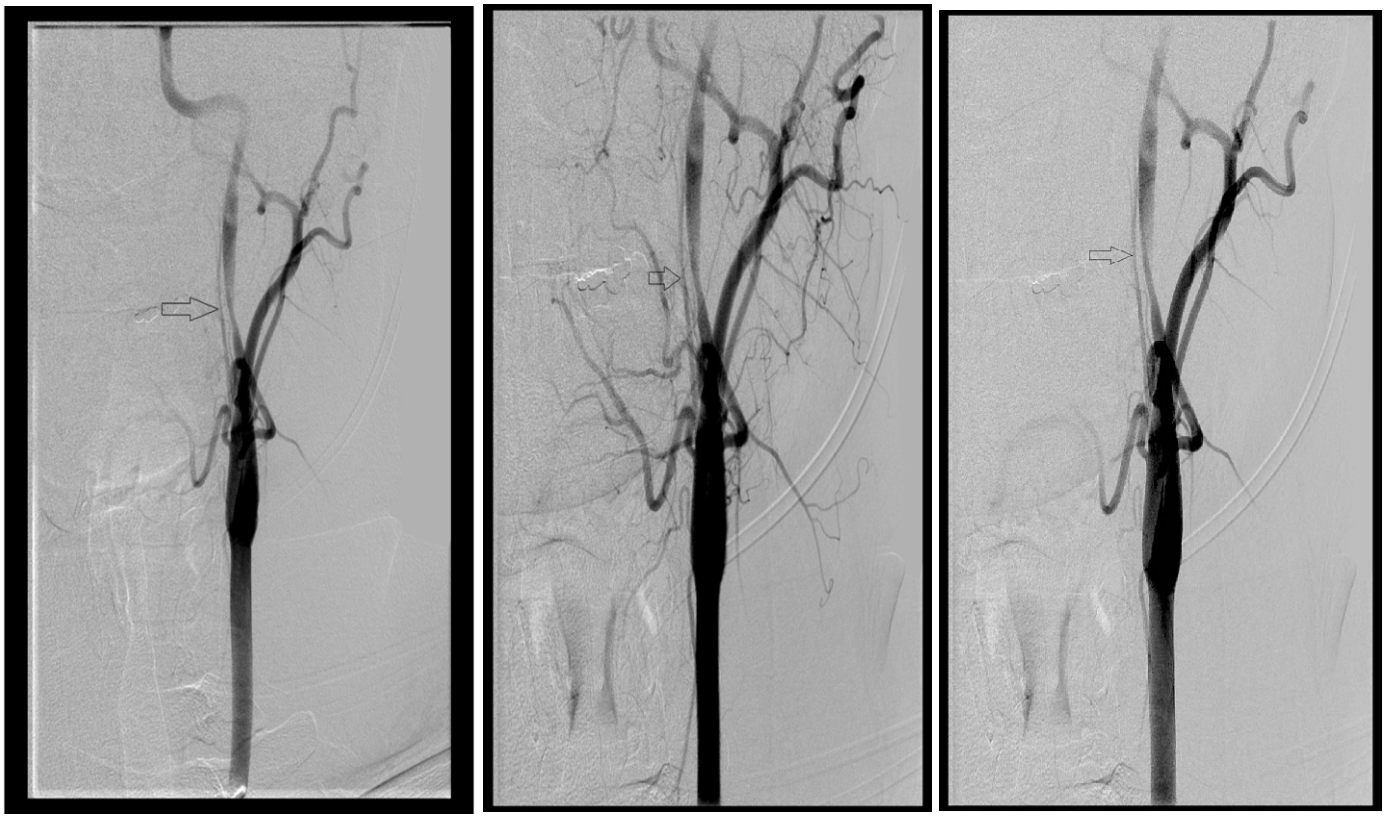
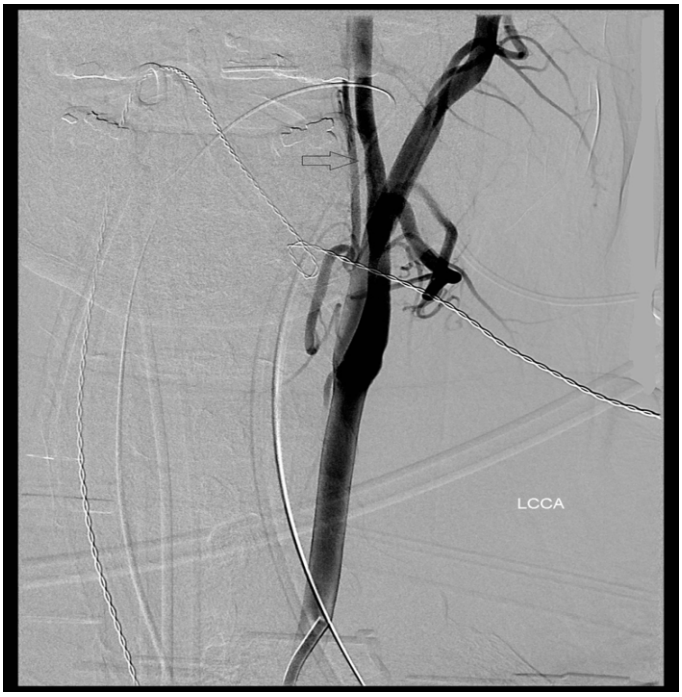
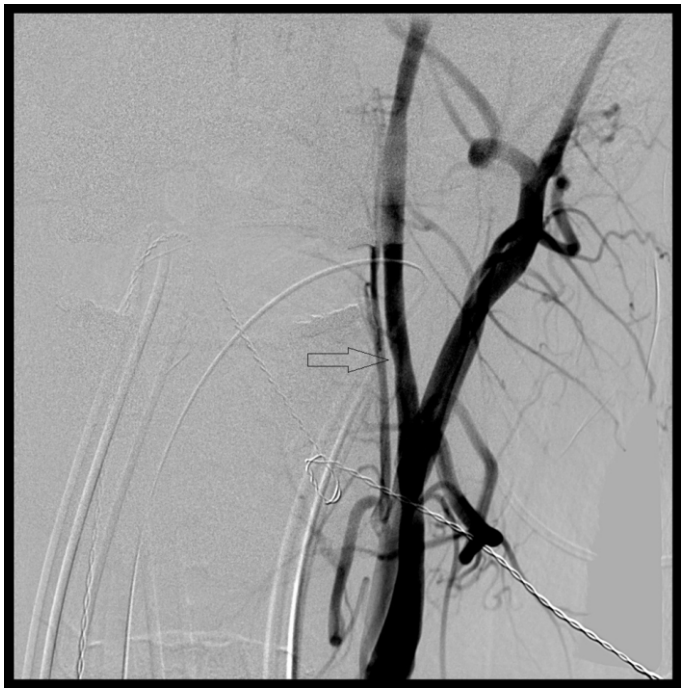


Figure D: Follow up DSA performed 24 hours later showing resolution of Left ICA stenosis.



Supplementary Figure 1: Initial DSA (frontal projection) of Left CCA showing area of focal stenosis (black arrow).





Supplementary Figure 2: 24 hour DSA (frontal projection) of left CCA showing resolution of focal area of stenosis (black arrow).

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