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Case Report

Swollen and bloodshot eye following headache

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ABSTRACT

A ruptured cavernous carotid aneurysm (CCA) with carotid cavernous fistula can appear as a benign headache but progress to a swollen and bloodshot eye overnight. A 66-year-old woman visited emergency department with sudden onset of pain behind her left forehead and vomiting. She was treated for a migraine-like headache and discharged. She presented again on the next day with a persistent headache and a swollen left eye with blurred vision. An ophthalmologic examination revealed erythema of the left lid and chemosis at the temporal and lower bulbar conjunctiva. A cranial nonenhanced computed tomography (CT) scan had been performed at her previous visit. The scan exhibited a nodular mass lesion involving the left cavernous sinus. CT angiography was subsequently used to determine that the lesion was a giant aneurysm in the left cavernous internal carotid artery, causing enlargement of the left ophthalmic veins. The symptoms of her left eye rapidly progressed to severe chemosis, edematous change over periocular region, and limited movements after 8 h. The patient received emergent lateral canthotomy and inferior cantholysis to avoid acute orbital compartment syndrome and was subsequently treated with stent-assisted coil embolization. A ruptured CCA is an urgent condition that requires rapid assessment of both cranial vascular and ocular lesions. A history of sudden onset headache with a nonpainful acute unilateral red eye may serve as a clue to prompt additional diagnostic studies and ophthalmologist evaluation. Adequate radiological studies and early endovascular intervention can reduce the likelihood of permanent ocular injury and vision impairment.

1. Case report

Undiagnosed intracavernous aneurysm is often asymptomatic or presents insidiously as a nonspecific headache. We present a case of sudden onset headache that mimicked a migraine and was later confirmed to be ruptured intracavernous aneurysm causing carotid cavernous fistula complicated with chemosis and proptosis of the left eye.

A 66-year-old woman visited the emergency department because of the sudden onset of pain behind her left forehead and vomiting. She was treated for a migraine-like headache and discharged. She presented again on the next day with a persistent headache and a swollen left eye with blurred vision. She was afebrile with blood pressure of 172/92 mm Hg, a pulse rate of 74 beats per min, and a breathing rate of 22 breaths per min. Results for her complete blood cell counts, hemocoagulation tests, and biochemistry examinations were normal. An ophthalmologic examination revealed erythema of the left lid and chemosis at the temporal and lower bulbar conjunctiva. Her intraocular pressure was 12.7 mm Hg in the right eye and 23.7 mm Hg in the left eye, and her visual acuity was 6/8.6

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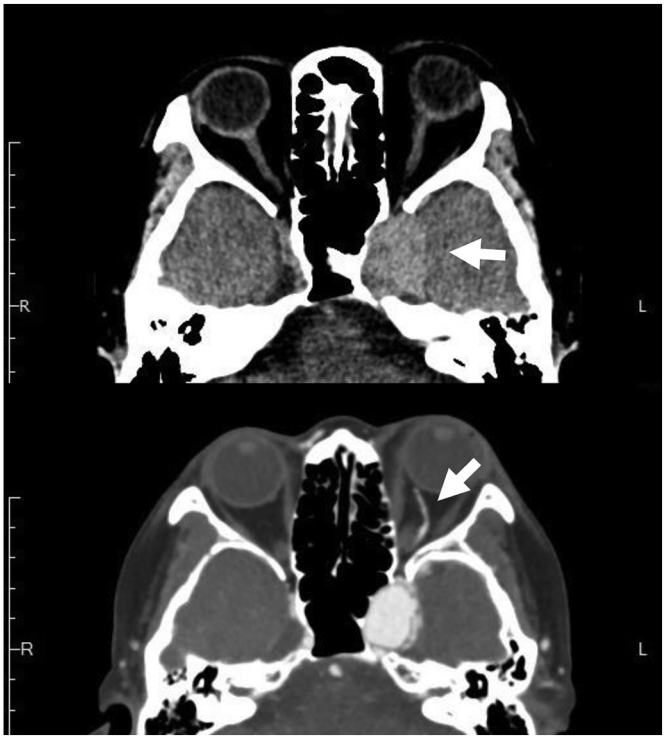

Fig. 1. Cranial non-enhanced computed tomography (CT) and CT angiography.

for the right eye and 6/10 for the left eye. Diplopia was noted when she gazed to the right, up, and down. A cranial nonenhanced computed tomography (CT) scan had been performed at her previous visit. The scan exhibited a nodular mass lesion involving the left cavernous sinus that measured approximately 2.6 cm × 1.9 cm. CT angiography was subsequently used to determine that the lesion was a giant aneurysm in the left cavernous internal carotid artery (ICA) and was causing enlargement of the left ophthalmic veins (Fig. 1). The symptoms of her left eye progressed to severe chemosis and proptosis after 8 h. Repeated ophthalmological examinations indicated edematous change without tenderness over the periocular region and limited movement of the left eye in all directions. Visual acuity was reduced to 6/30, and intraocular pressure increased to 54.0 mm Hg for the left eye. Emergency lateral canthotomy and inferior cantholysis were performed to avoid acute orbital compartment syndrome (Fig. 2). The patient was subsequently treated with stent-assisted coil embolization (Fig. 3).

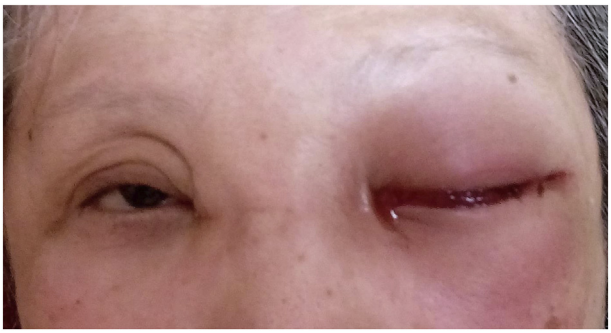

Fig. 2. Proptosis of left eye status post lateral canthotomy and inferior cantholysis.

2. Discussion

Cavernous carotid aneurysms (CCA) account for 2%–9% of all intracranial aneurysms [1]. The etiology of CCA can be idiopathic and traumatic. CCA often remain asymptomatic and may be detected incidentally or from a progressive increase of mass effects that results in compression of the cavernous sinus. Intracavernous rupture occasionally results in a direct carotid cavernous fistula through which arterialized blood is shunted into the venous system, causing venous reflux and ocular symptoms. The risk of rupture within 5 years is typically associated with aneurysm size, with 3% for aneurysms 13–24 mm and 6.4% for aneurysms >5 mm [2].

Carotid cavernous fistulas with direct connections between the ICA and the cavernous sinus are usually “high flow” lesions, contrasting with “low flow” lesions, which are connected by the meningeal or arterial branches and are characterized by more insidious symptoms. Patients with direct fistulas classically present with sudden onset of the clinical triad of chemosis, proptosis, and bruit. Prior to these manifestations, patients tend to experience a nonspecific headache, which is sometimes diagnosed as a migraine- or cluster-like headache upon initial presentation [3,4]. However, a headache with a painless red eye should not be confused with a painful red eye that may indicate another etiology such as conjunctivitis or acute angle-closure glaucoma [5]. Continuous venous reflux into ophthalmic veins and hemorrhagic mass effects in the cavernous sinus can lead to exophthalmos; oculomotor palsy of the third, fourth, and sixth cranial nerves; and ocular hypertension.

Diagnosis using routine nonenhanced cranial CT can be challenging; the imaging resolution is limited, and thus clear delineation of the vascular lesion at the cavernous sinus is difficult. Moreover, a ruptured CCA may only involve the cavernous sinus proper without hemorrhaging into the surrounding areas or subarachnoid space. CT angiography is therefore the modality of choice for determining initial diagnosis because it can be used to clearly identify vascular characteristics and anatomic information. Cerebral angiography is the standard for imaging vascular malformations because it enables specific vascular anatomical characterization as well as relatively accurate image reconstruction.

Endovascular intervention is the treatment of choice for ruptured CCA [6,7]. Endovascular coil embolization with or without the use of balloons or intracranial stents is currently the most effective modality because of the following advantages: preservation of the parent artery, reduced hospital stay, and lower rates of morbidity and mortality. Surgical intervention is reserved for lesions with significant aneurysmal mass effects, projection into subarachnoid space, and cases in which endovascular therapy has failed.

A ruptured CCA with the direct type of cavernous fistula is an urgent condition that requires rapid assessment of both cranial vascular and ocular lesions. A history of sudden onset headache with a nonpainful acute unilateral red eye may serve as a clue to prompt additional diagnostic studies and ophthalmological evaluation. Adequate radiological studies and early endovascular intervention can reduce the likelihood of permanent ocular injury and vision impairment.

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None.

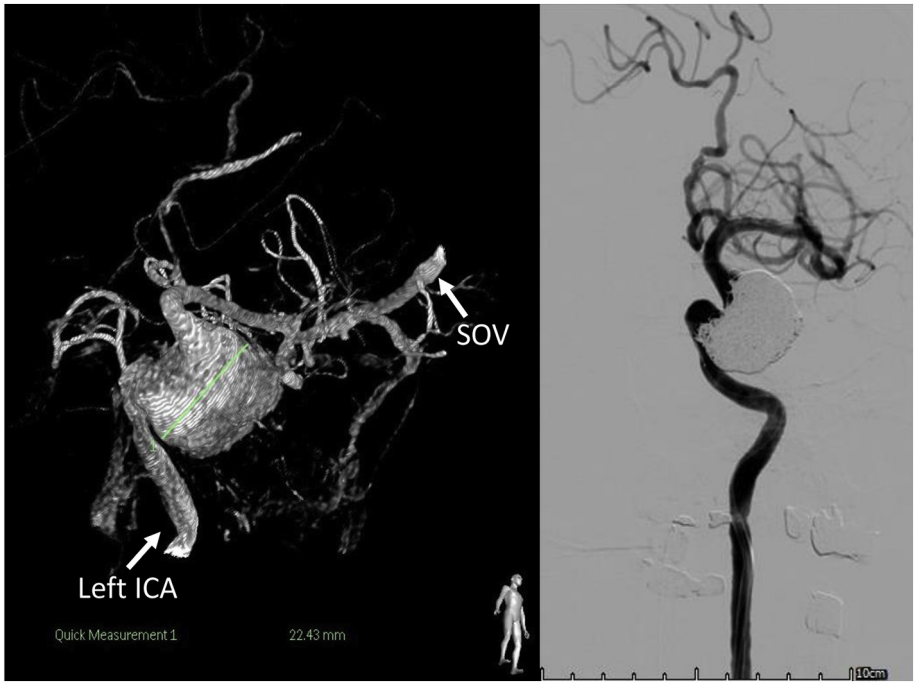

Fig. 3. Three-dimensional digital subtraction angiography shows the cavernous aneurysm (left; ICA, internal carotid artery; SOV, superior ophthalmic vein) and angiogram after stent-assisted coil embolization (right).

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