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Third nerve palsy following carotid artery dissection and posterior cerebral artery thrombectomy: Case report and review of the literature

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Abstract

Background: Common causes of oculomotor nerve palsy are diabetes, aneurysmal compression, and uncal herniation. A lesser-known cause of third nerve dysfunction is ischemia, often due to carotid artery dissection.

Case Description: An 80-year-old man presented with an acute ischemic stroke with a National Institutes of Health Stroke Scale score of >20 from a high cervical internal carotid artery (ICA) dissection and a tandem ICA terminus embolic occlusion with extension of clot into the adjacent fetal posterior cerebral artery (PCA). We used a stentriever to perform selective PCA thrombectomy, with immediate postthrombectomy development of ipsilateral anisocoria. The anisocoria progressed into complete oculomotor nerve palsy over 8 h after the procedure.

Conclusions: The clinical course described in this case is consistent with injury to the third nerve due to mechanical injury or occlusion of perforator supply to the nerve during thrombectomy. Oculomotor nerve palsy is a rare but known complication after ischemia; however, to our knowledge, this is the first case after thrombectomy for a PCA embolus.

Key Words: Carotid artery dissection, mechanical thrombectomy, oculomotor nerve palsy, stroke, third cranial nerve palsy

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INTRODUCTION

Common causes of third cranial nerve palsy classically involve medical and compressive (surgical) pathologies. Generally, medical pathologies are at first pupil sparing, whereas compression of the nerve first causes dilation and then subsequent motor dysfunction. In this report, we present a lesser-known cause of third nerve dysfunction due to carotid dissection and discuss the vascular anatomical nuances that need to be considered in the differential of intraprocedural development of oculomotor palsy.

CASE REPORT

An 80-year-old man with type II diabetes and known atrial fibrillation for which he was on warfarin presented

with confusion, aphasia, and right-sided hemiplegia 1.5 h after last seen normal. His level of anticoagulation was subtherapeutic with an international normalized ratio of 1.27. The National Institutes of Health Stroke Scale score was 21 with symmetric pupil reactivity and no gaze deviation. Computed tomography (CT) perfusion imaging suggested a large posterior cerebral artery (PCA) distribution, salvageable penumbra [Figure 1a]. CT angiography (CTA) revealed left internal carotid artery (ICA) occlusion extending from the carotid bulb up to the terminus with a luminal defect suspicious for a thrombus [Figure 1b]. 4D CTA reconstructions revealed delayed filling of the left anterior cerebral artery and middle cerebral artery (MCA) through the anterior communicating artery (ACOM), but there was no filling of the ICA terminus [Figure 1c and d]. The PCA was not visible on CTA, possibly due to the presence of a fetal PCA and a second embolus into the PCA. The patient was not considered a candidate for tissue plasminogen activator because of concurrent anticoagulation.

Secondary to severe neurologic dysfunction and inability to cooperate, the patient was emergently intubated and brought to the interventional suite for possible intraarterial thrombectomy. Catheter angiography confirmed cross-flow across the ACOM with an ICA-terminus occlusion. Additionally, the posterior circulation revealed no left P1 segment, confirming a fetal PCA. Left carotid angiography revealed left ICA dissection just distal to the origin of the ICA (flame-shaped ICA stump) and the external carotid artery filling the ophthalmic artery but not the clinoidal segment of the ICA. This suggested the presence of a dissection in the left ICA with an embolus in the ophthalmic, communicating, and terminal segments of the ICA [Figure 2a-d].

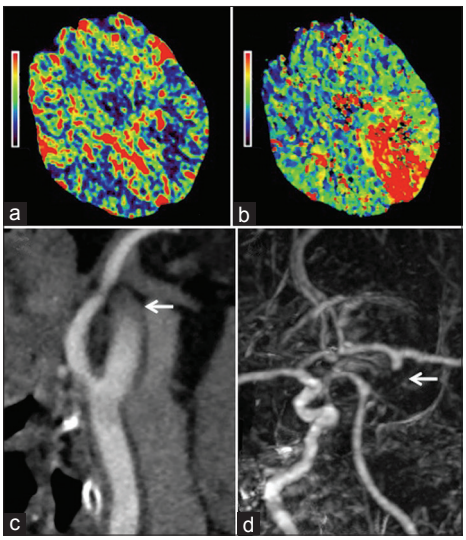

Figure 1: (a) Cerebral blood volume scan from a CT perfusion study obtained at the time of admission. A small area of volume deficit is visible in the left PCA territory. (b) Time-to-peak map from the CT perfusion study showing a large area of reduced perfusion in the left PCA, consistent with salvageable penumbra. (c) CT angiogram of the common carotid artery. Proximal occlusion consistent with a dissection flap is seen (*arrow*). (d) CT angiogram of cerebral vessels showing an early filling defect in the terminal ICA (*arrow*)

Direct aspiration with the 5 Max Aspiration catheter (Penumbra Inc., Alameda, California, USA) resulted in recanalization of the entire ICA with persistent fetal PCA occlusion [Figure 2e]. A 4 × 15 mm Solitaire stentriever (ev3-Covidien, Irvine, California, USA) was then deployed in the left PCA up to the P2 segments, and thrombectomy was performed. The vessel recanalized with residual occlusion beyond the P3 segment [Figure 2f]. Immediately after PCA thrombectomy, transient left pupillary mydriasis was observed. Further recanalization was abandoned, and a CT head scan was obtained immediately, which revealed no acute changes of concern.

During the 8 h after the thrombectomy procedure, the patient developed a progressive left mydriatic pupil, which ultimately became fixed at 7 mm in diameter to light stimulation [Figure 3]. A repeat head CT scan did not show any significant effacement of the basal cisterns [Figure 4a]. By 16 h postthrombectomy, the patient had developed complete oculomotor palsy with ptosis, a fixed mydriatic pupil, and eye deviation laterally and downwards, suggestive of an isolated third nerve palsy. Magnetic resonance imaging revealed infarction of the distal PCA territory with sparing of the midbrain and multiple embolic infarcts of the MCA territory [Figure 4b and c]. The oculomotor palsy remained, with resolution of the anisocoria. The patient failed to improve significantly; and, in line with preevent wishes, the family elected to withdraw care 10 days after the procedure and the patient expired.

DISCUSSION

Development of an intraprocedural third nerve palsy can be an ominous sign of impending herniation from an enlarging hematoma. In this case, we abandoned further attempts at recanalization to obtain an emergent CT scan to exclude the presence of such a hematoma, resulting potentially in poor outcome from lack of full revascularization. Newer generation angiography systems have the ability to provide close to CT quality images with fluoroscopic equipment,[1,7] obviating the need to abandon the procedure for transfer to a CT scanner. This report highlights the vascular anatomical nuances that need to be considered in the differential of intraprocedural development of oculomotor palsy.

The vascular anatomy of the third nerve has been described by Cahill *et al.*[2] In 11 cadaveric specimens, they demonstrated that the proximal, extracavernous portion of the third nerve was supplied by perforators arising from the PCA in all samples. The middle portion had no specific extraneural supply, whereas the distal, intracavernous portion was supplied by perforators from the cavernous ICA, namely, the inferior hypophyseal and inferior cavernous sinus arteries, in all specimens.

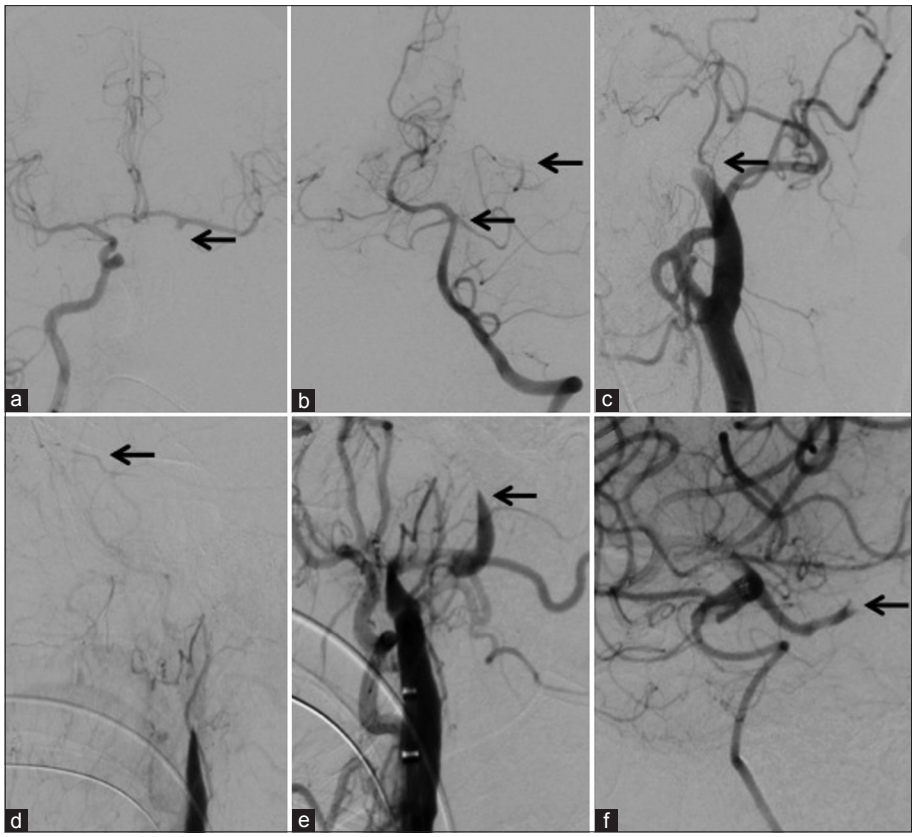

Figure 2: Angiograms preceding and following endovascular intervention. (a) Right ICA injection showing contralateral filling of the MCA with a defect suggestive of a dissection in the ICA terminus on the left (*arrow*). (b) Left vertebral injection showing filling of the right but not the left PCA. There is retrograde filling of the distal left P2 by superior cerebellar artery collaterals (*arrow* on far right). (c) Left carotid artery angiogram showing progressive narrowing of the ICA lumen (*arrow*), consistent with artery dissection. (d) Left ICA injection showing late filling of the left ophthalmic artery due to collateral circulation (*arrow*). No back filling of the ICA terminus was observed. (e) Left ICA injection is shown. The Penumbra catheter (Penumbra, Inc., Alameda, California, USA) visibly cleared the proximal ICA occlusion; however, a dissection flap is visible distally (*arrow*). (f) Angiogram obtained immediately after Solitaire (ev3-Covidien, Irvine, California, USA) thrombectomy shows good filling of the distal ICA; however, an occlusion persists in the left P2 segment of the PCA (*arrow*)

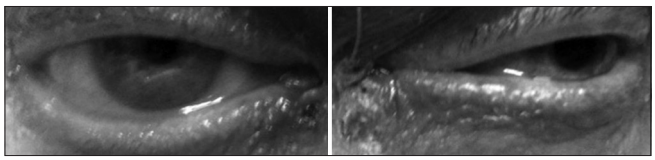

Figure 3: Picture taken on hospital day 4 of patient's right and left eyes, respectively. Mydriasis of the left eye is visible with deviation laterally and inferiorly

The anatomy of the ICA in relation to the third nerve makes it very plausible that a dissection of the cavernous ICA can compromise vascular supply of the distal nerve. Consequently, third, fourth, and sixth nerve palsies are known sequelae of ICA dissection, occurring with a frequency of 2.6%; however, fewer patients develop an isolated third nerve involvement.[6,10] In a recent case report and review, Nizam *et al*.[8] summarized 16 cases of partial or complete third nerve involvement, along with other symptoms, in the setting of carotid artery dissection that have been reported in the literature. Interestingly, a fetal PCA variant would allow for additional proximal involvement of the nerve as well. Consequently, it has been suggested previously that the presence of fetal PCA anatomy increases the likelihood

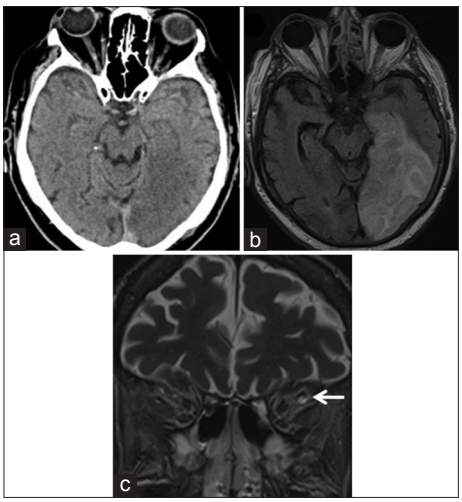

Figure 4: (a) CT scan of the head on hospital day 1, after left pupillary dilation was first observed. Hypodensity in the left PCA territory is seen; no effacement of cisterns was evident. (b) Axial T2 fluid-attenuated inversion recovery (FLAIR) magnetic resonance image on hospital day 5 showing evolving left PCA infarct. (c) Coronal T2 magnetic resonance image of head. A left-sided hyperintensity is visible in the retroglobar area (*arrow*), consistent with congestion of the superior ophthalmic vein

of third nerve ischemia during dissection,[8] and such palsies have been reported in other cases.[3,5]

Preganglionic parasympathetic blockade usually occurs in the context of oculomotor nerve palsies, and much has been written about the significance of pupil involvement or pupil "sparing." In general, if the oculomotor nerve palsy is incomplete, pupil involvement implies an extrinsic compressive lesion until proved otherwise.[9] The anatomical studies of Kerr and Hollowell,[4] conducted in several species of animals, help to explain this clinical finding: They observed that the pupillomotor fibers travel superficially throughout much of the subarachnoid part of the nerve, rendering them more susceptible to compressive lesions but less susceptible to ischemic injury, such as in diabetic neuropathy. Our patient's clinical course and symptoms are compatible with involvement of third nerve vasculature owing to ischemia in the region of the PCA and posterior communicating artery perforators; however, a direct mechanical effect of stentriever thrombectomy cannot be excluded.

Oculomotor nerve involvement in our patient may have been caused by three factors: Direct irritation (compression) of the oculomotor nerve during thrombectomy, compaction of emboli within the ostia of perforator branches to the third nerve, and extension of the dissection into the cavernous carotid artery after evacuation of the clot. Initial correspondence of the mydriasis to the mechanical removal of the PCA clot argues to a direct mechanical compression of the third nerve. However, because the symptoms waxed and waned and ultimately progressed to a full motor paralysis with ptosis, this explanation alone is less likely. Further, magnetic resonance imaging findings postoperatively did not demonstrate any midbrain stroke to explain the patient's examination findings [Figure 4b and c]. Finally, the clinical course after discovery of the pupil is certainly compatible with previous reports of ICA dissection-associated third nerve palsy[8] or ischemic injury from perforator loss in the recanalized PCA. Our hypothesis, therefore, is that the most likely cause of third nerve palsy in this case was the recanalization of the fetal PCA. This either allowed the dissection flap to spread to the areas of oculomotomotor nerve perforators in the vessel or released clot directly into these perforators. In either case, third nerve ischemia and mydriasis would be expected.

To our knowledge, this is the first report of an isolated third nerve palsy following mechanical thrombectomy of an ICA terminus/PCA embolus. Because of the association between third nerve palsy and uncal herniation from mass effect, this clinical finding warrants an urgent CT scan to exclude a potentially fatal enlarging hematoma. This report highlights nonexpansile causes of oculomotor palsy, which should be considered when faced with this finding during a procedure. We recommend obtaining an emergent intraprocedural fluoroscopic CT-like image to exclude a hematoma prior to abandoning further recanalization in order to acquire a formal CT scan.

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