

## Case Report

# Isolated oculomotor nerve palsy with pupillary dilatation: A rare presentation of internal carotid artery dissection

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**Key words:** oculomotor nerve palsy, internal carotid artery, pupillary dilatation, dissection of internal carotid artery, surgical third nerve palsy

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## Introduction

Causes of third cranial nerve palsy are medical (non-compressive) and surgical (compressive). Generally, medical pathologies are pupil sparing whereas compression of the nerve causes dilatation of the pupil [1]. Aneurysms, uncal herniation and tumours that compress the nerve will involve the pupillomotor parasympathetic fibres on the surface of the nerve trunk and their blood supply resulting in an early presentation with a dilated pupil. On the other hand, medical conditions, such as diabetes mellitus or hypertensive microangiopathy will affect the vasa vasorum and thus cause ischaemia of the core of the nerve and usually spare the surface pupillary fibres [2].

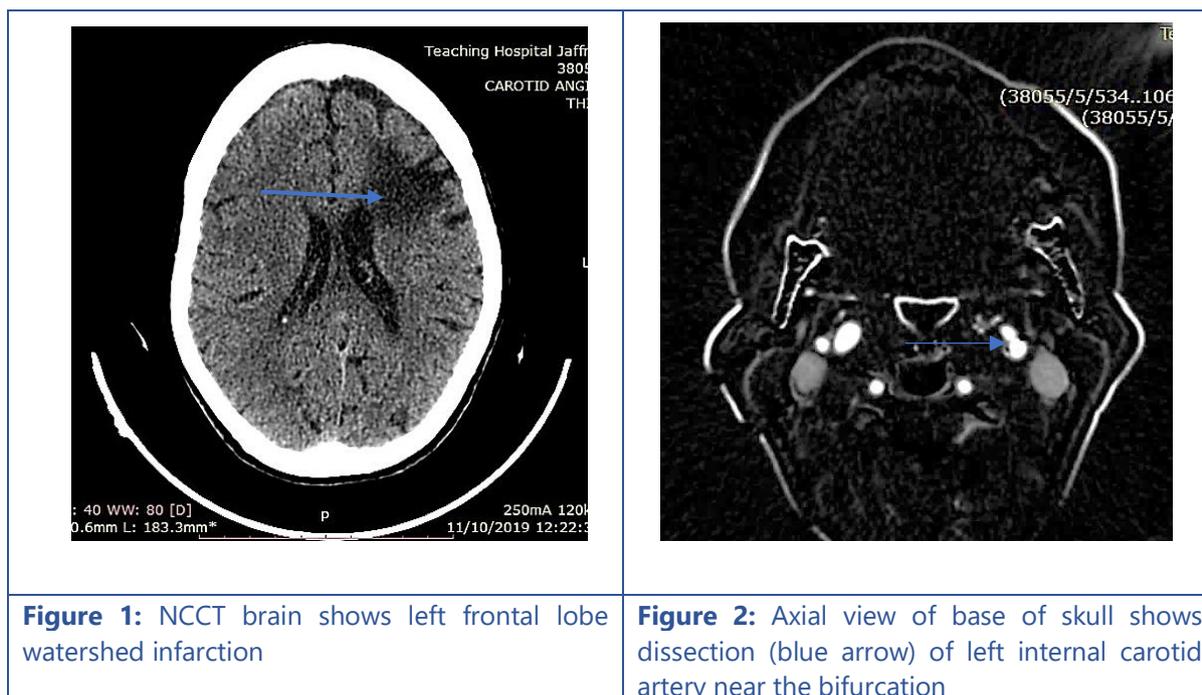
Internal carotid artery (ICA) dissection is an important cause of ischaemic stroke in young and middle-aged patients, accounting for 14–20% of all cases. Trauma and connective tissue disorders are well-known predisposing factors but the majority of cases are spontaneous or related to minor trauma [3]. The classical clinical picture is that of a patient who presents with unilateral pain in the head, face or neck and Horner's syndrome with contralateral motor and sensory manifestations. Cranial nerves may be affected in 6% to 12% of patients with carotid artery dissection. The lower cranial nerves are the most commonly affected. Ocular motor dysfunction, due to involvement of the third, fourth or sixth cranial nerve, is found only occasionally and isolated oculomotor nerve palsy is rarely seen [4].

## Case presentation

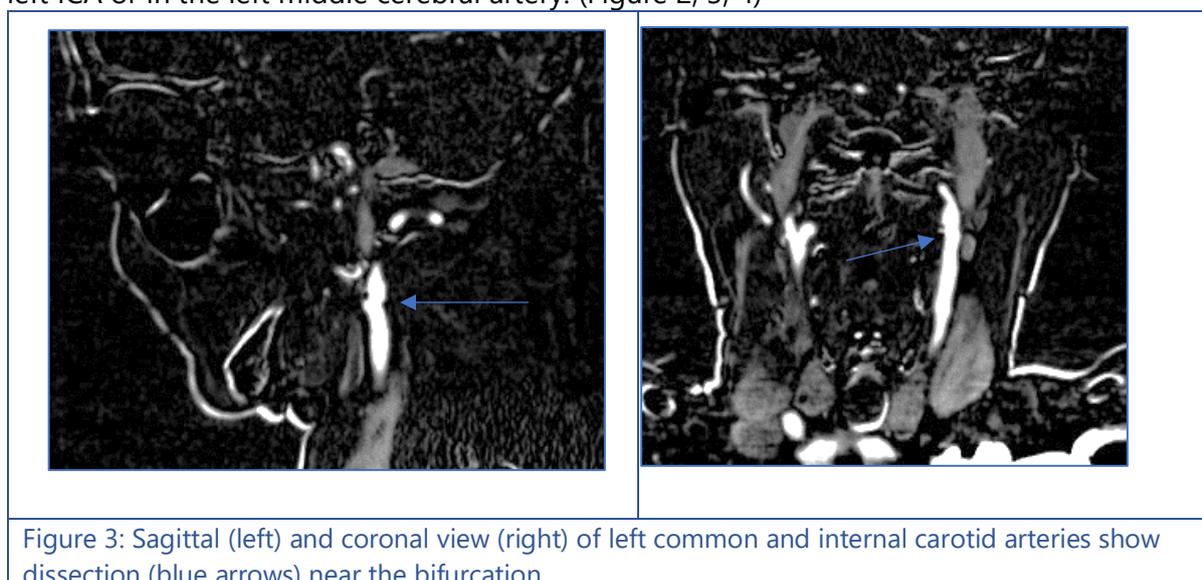
A 72-year-old woman with a history of type 2 diabetes mellitus (DM) and hypertension (HTN) was admitted to hospital with complaints of complete ptosis of the left eye and left side headache of three days' duration. She had double vision when looking to the left. There was no history of recent falls or trauma. She denied any visual, motor or sensory symptoms.

Her blood pressure was 150/80mmHg in both arms. Neurological examination revealed ptosis of the left eye with impaired adduction and pupillary dilatation with poor reaction to bright

light. Abduction and internal rotation of the orbit were preserved. Fundi were normal and all other cranial nerves were intact. She denied any symptoms suggestive of micro or macro vascular complications of DM and HTN. History and clinical examination were negative for any underlying connective tissue diseases including vasculitis. The patient did not have any limb, trunk or facial weakness during this presentation. Routine haematological investigations were normal. Erythrocyte sedimentation rate was 09mm (0-30) /first hour and C-reactive protein 6.6mg/L (0-3). Blood sugar measurements were satisfactory. Two-dimensional echocardiogram was normal with no intramural thrombi. CT Brain showed an area of watershed infarction in the left frontal lobe. (Figure 1)



CT Angiogram (CTA) showed dissection of the left ICA, originating from the bifurcation of the left common carotid artery. No blood flow was seen in the distal cavernous part of the left ICA or in the left middle cerebral artery. (Figure 2, 3, 4)



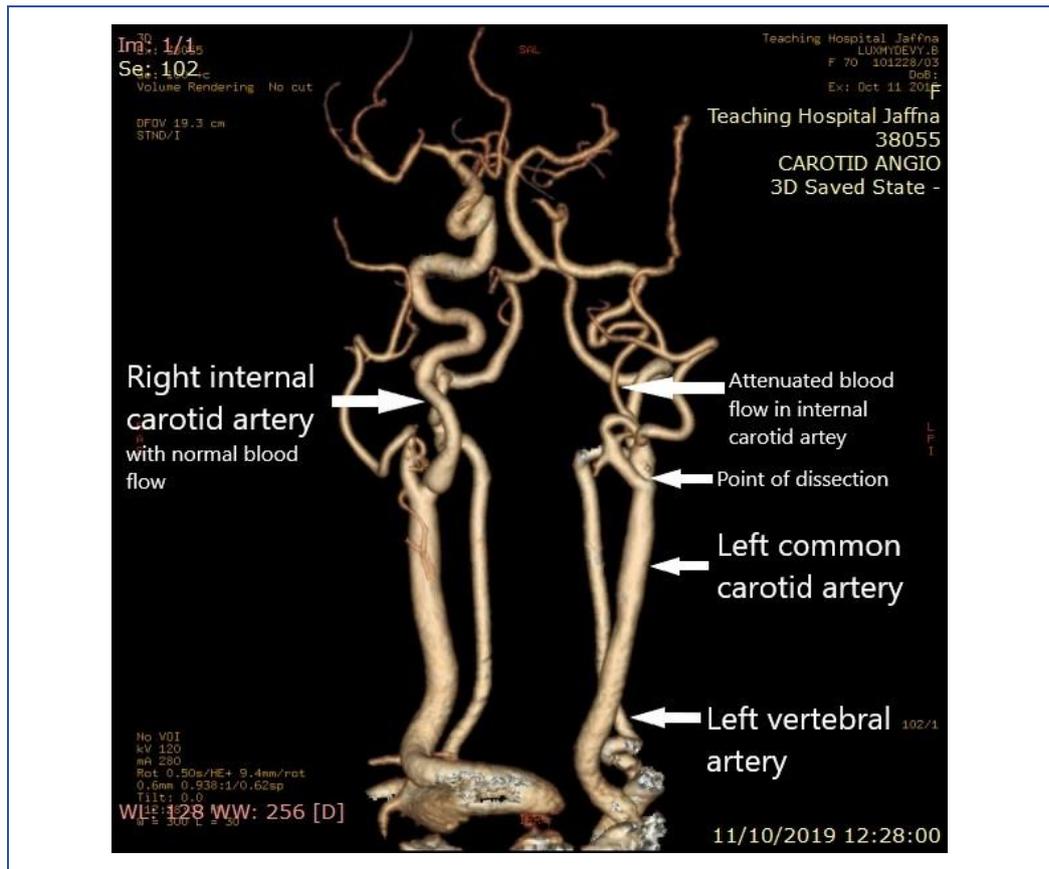


Figure 4: CTA showing dissection of the left ICA originating from the bifurcation of the left common carotid artery with attenuation of blood flow. There was no blood flow in the distal cavernous part of the left ICA and in the left middle cerebral artery.

Management options were discussed with the ophthalmologist, neurologist and neurosurgeon. The condition, the modes of treatment and their outcomes were explained to the patient. She was discharged from hospital and prescribed dual antiplatelet therapy, atorvastatin, antihypertensives and oral hypoglycaemic agents. On subsequent follow up it was noted that the oculomotor palsy had completely recovered, and the pupil had become normal in size after two months.

### Discussion

Angiographic evaluation is warranted in patients with oculomotor nerve palsy and pupillary dilatation to exclude a posterior communicating artery aneurysm. (3) We considered a posterior communicating artery aneurysm as the first differential diagnosis, but CTA did not reveal any intracranial aneurysm. Instead, dissection of ICA was found.

Isolated oculomotor nerve palsy is rare in ICA dissection and can easily be overlooked [3]. NCCT brain showed a watershed infarction probably due to proximal ICA occlusion which can be asymptomatic.

The most common ocular signs of carotid dissection are Horner's syndrome and transient monocular blindness. Involvement of the internal part of the pericarotid sympathetic plexus results in Horner's syndrome while thromboembolic events cause the blindness. Cranial nerve

palsies are not common in carotid artery dissection and isolated oculomotor nerve deficit is rare.

Proximally, the oculomotor nerve receives blood supply from the thalamoperforating arteries supplemented by other arterioles from brainstem vessels. The middle part of the nerve does not receive a blood supply. The distal part of the nerve is supplied by branches of the inferior cavernous sinus artery supplemented by a tentorial branch of the meningo-hypophyseal trunk which are branches of the ICA [5].

The ICA lies near to the oculomotor nerve within the cavernous sinus. If the dissection of the ICA extends into this site, the resulting compression or stretching of the oculomotor nerve could cause the nerve palsy. However, diminished blood supply to the third nerve due to dissection is another possible mechanism [4].

This patient's presentation was unusual with involvement of the oculomotor nerve with a dilated pupil rather than Horner's syndrome. NCCT brain and CTA did not reveal any aneurysm or masses adjacent to the course of the oculomotor nerve. The only abnormality detected in the CTA was the dissection of ICA near its origin not extending to the cavernous part. It should also be noted that no blood flow was seen in the distal cavernous part of the left ICA and in the left middle cerebral artery. So, mechanical compression was unlikely to be the cause for this presentation. Impairment of blood supply to the oculomotor nerve could be a plausible explanation.

Watershed stroke in the left frontal lobe may be caused by dissection of the left ICA as these types of infarctions are usually due to global hypoperfusion (e.g.: cardiac arrest) or local hypoperfusion due to stenosis of the carotid artery or other major vessels [6].

There are rare case reports of dilated pupils in ischaemic third-nerve palsy due to compromised blood flow [7,8]. The complete recovery of our patient in two months supports this pathology as most patients with ischaemic oculomotor nerve palsy improve within one month with complete recovery in three months [**Error! Reference source not found.**].

## Conclusion

ICA dissection should be considered as a cause of isolated oculomotor palsy with pupillary dilatation, if no other cause is found.

## Acknowledgements

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