Monocular Blindness as a Rare Presentation of Aortic Dissection Complicated by Carotid Artery Dissection: A Case Report and Literature Review

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The clinical manifestations of aortic dissection are highly variable. Although the majority of patients manifest typically with chest pain and radiation to the back or congestive cardiac failure, atypical presentations have also been documented that can be easily overlooked. We report a 47-year-old man who developed monocular blindness and visited our emergency department. Computed tomography of the chest at a later time revealed aortic dissection complicated by common carotid artery dissection. After successful surgical treatment, the patient was discharged uneventfully 29 days after admission. A high level of vigilance is required in order to diagnose carotid artery dissection when a patient presents with painless acute monocular visual loss.

Key words: acute visual loss, monocular blindness, aortic dissection, carotid artery dissection

Introduction

Acute visual loss is not an unusual complaint in the emergency department. The etiologies include vitreous hemorrhage, retinal vascular occlusion, glaucoma, optic neuritis, temporal arteritis, and trauma. Aortic dissection is a clinical chameleon that can present without any classical features such as chest pain radiating to the back, or a pulse deficit between the upper and lower extremities (1). Nonetheless, about 90% of cases present with chest pain, stroke, or congestive cardiac failure (2). Central retinal artery (CRA) occlusion due to internal carotid artery dissection is uncommon and is usually associated with other neurological deficits (3). Clinical presentations of common carotid artery dissection include neck pain, unilateral facial or orbital pain and oculosympathetic palsy (3). Monocular blindness can be a result of an interruption of the blood flow to the retina (1). We report a middle-aged man who developed monocular blindness secondary to aortic dissection that extended to the carotid artery.

Case Report

A 47-year-old man without any previously known systemic disease initially presented with a sudden onset of visual loss involving his right eye since the night before his visit to our emergency department. Five days before the episode, he had suffered from severe back pain after lifting a weight...
of less than one kilogram. Bilateral numbness of the feet and right lower limb claudication were noted afterwards. His body temperature on arrival at our emergency department was 36.4 degree with a respiratory rate and a pulse rate of 20 and 80 per minute, respectively, and a blood pressure of 160/55 mmHg. The visual loss was transient and showed slight recovery. On examination, the visual acuity of his right eye was 0.6 with free and full eye movement. His right pupil had been iatrogenically dilated during his visit to a local ophthalmology clinic, while his left pupil was light-reactive and 3.0 mm in size. No remarkable visual field defect was noted in either eye. Examination of the fundus of right eye showed localized disc swelling without a thrombus or ischemic change over macula; this contrasted with a lack of any noticeable anomaly in his left eye. There was no evidence of cranial nerve involvement on neurological examination, including diplopia, facial numbness, dysphagia, tongue deviation, or a decrease in muscle power of the extremities. However, severe back pain and hypotension were noted minutes later. Because of these symptoms and the finding of a widened mediastinum on chest radiography, the patient underwent emergency computed tomography. This revealed aortic dissection type A from ascending aorta down to bilateral iliac arteries, with involvement of innominate trunk and right proximal common carotid artery (CCA) as well as occlusion of right proximal CCA (Fig. 1 & 2 & 3). An emergency operation was performed by the cardiovascular surgeon. The findings during the operation included: (1) an aortic root aneurysm
with acute/subacute dissection, involving the whole aortobi-iliac artery and the whole of the branchiocephalic artery, which were complicated by total occlusion of right CCA; and (2) marfanoid with annuloaortic ectasia and severe aortic regurgitation (AR). A 20 mm Hemashiled tube graft (reversed sleeve) was used to replace the ascending aorta. Right common carotid artery was replaced by a 8mm Gore-Tex graft connected to the aortic Hemashield graft. Anticoagulation and antiplatelet regimens, namely warfarin and clopidogrel, respectively, were started five days after operation. The patient was discharged without complication after hospitalization for 29 days. However, there had been no significant improvement in the visual acuity of his right eye by the time the patient had been discharge. Oral warfarin was prescribed for 5 months, which was followed by a maintenance dose of aspirin. He has been regularly followed at our outpatient clinic for over 26 months and over this time there has still been no improvement in the visual acuity of the right eye.

**Discussion**

Among the various causes of painless visual loss, central retinal artery occlusion (CRAO), central retinal vein occlusion, and temporal arteritis are the most common\(^{(4,6)}\). In this context, the leading causes of CRAO are embolus, thrombosis, vasculitis, sickle cell disease, and trauma\(^{(5,7,9)}\). However, in rare cases, malperfusion of the affected eye caused by ipsilateral carotid artery dissection may also lead to monocular blindness. The clinical presentation has been reported to be severe, sudden, painless, central or paracentral visual loss\(^{(4)}\).

The incidence of internal carotid artery dissection has been estimated to be 2.6 cases/100000 population\(^{(10)}\) but the percentage of aortic dissection involving internal carotid artery has not been documented. The reported common clinical manifestations of internal carotid artery dissection include headache or facial pain (76%), focal cerebral ischemic symptoms (62%) and oculosympathetic palsy (36%)\(^{(11)}\). Indeed, there are a number of previous reports of isolated

![Computed tomography of the chest showing total occlusion of right common carotid artery (arrow)](image)
central retinal artery occlusion due to internal carotid artery dissection in the literature\(^3,11-14\). The neurological presentations of the above cases, when combined with visual loss, are tell-tale signs of a compromised cerebral blood flow. Under these circumstances, an imaging study of the brain ought to be the immediate diagnostic tool of choice. The situation presented in the present report is unique in that there was no pain, no history of trauma and no neurological defect, all of which would be suggestive of the presence of a potentially life-threatening vascular lesion such as arterial dissection. In such a case such as this, delayed diagnosis is likely without a high level of suspicion.

Anticoagulant or antiplatelet medication may prevent arterial thrombosis when treating extracranial internal carotid artery dissection\(^15\). In addition, there was no significant difference in the odds of death when antiplatelet drugs compared to anticoagulants\(^15\). Nonetheless, a low incidence of intracranial hemorrhages (0.5%) has reported among patients on anticoagulants, while none was documented among patients on an antiplatelet regimen\(^15\). Although antiplatelet therapy is still the treatment of choice for carotid artery dissection, carotid artery stenting is the preferred therapeutic alternative for patients with progressive infarcts. This is despite the possibility of antiplatelet or anticoagulation therapy, the fact that some patients may have contraindications \textit{re} anticoagulation, and the fact that some patients may have iatrogenic dissections due to the surgical procedures\(^16\). The morbidity rate of carotid artery stenting has been reported to be 6\%\(^17\).

In our patient, it is proposed that his visual loss was caused by central retinal artery occlusion due to common carotid artery dissection, with the dissection occurring without any focal neurological signs such as local headache or Horner syndrome. The precipitating factor for dissection in our patient may have been lifting of the 1kg weight, which then induced a marfanoid vessel injury that presented as monocular blindness.

**Conclusion**

The majority of carotid artery dissection are attributable to trauma leading to vessel damage; however, especially in patients with connective tissue diseases, a history of trauma or an underlying systemic disease may be absent. The present report highlights the importance of a high level of vigilance when a patient presents with painless acute monocular visual loss. The relevant parts of a patient's history, including any weight-bearing and any neurological defect, as well as the presence of pain of apparently unrelated origin, should be also carefully scrutinized and carotid artery dissection need to be included in the list of differential diagnoses.

**References**

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以單眼視力喪失為表現的主動脈剝離合併頸總動脈剝離：病例報告

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主動脈剝離的臨床表現是很多樣性的。雖然大多數的病人典型表現為胸痛並轉移至背部或心衰竭，但非典型的表現卻容易被忽略。我們報告一個47歲男性因為單眼視力喪失至急診就診。電腦斷層顯示主動脈剝離合併頸總動脈剝離。經開刀修補29天後順利出院。若病人以無痛性單眼視力喪失求診時，頸總動脈剝離也是須考慮的診斷之一。

關鍵詞：急性視力喪失，單眼視力喪失，主動脈剝離，頸總動脈剝離

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