My Husband's Eye Looks Funny Doctor

R Sankaranarayanan

Citation

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Abstract

We report the case of a young man who presented with headache and Horner's syndrome. Investigations confirmed spontaneous carotid dissection. This case reminds of a rare but significant cause of stroke in the young.

CASE REPORT

A 38 year old man presented with a 1 week history of right sided throbbing headache, gradual in onset and not associated with visual symptoms. The headache had no diurnal variation, was not associated with premonitory symptoms, fever or vomiting. There were no known precipitating factors and no history of trauma. His wife also mentioned that his right eye looked funny. There was no associated limb weakness or sensory symptoms. He was started on Sumatriptan by the general practitioner as he was diagnosed to have migraine in the past. There was no other past medical history.

On examination, he was afebrile, his blood pressure. was 130/80 mm Hg and pulse was 72 per minute, regular. He had partial ptosis of his right eye, conjunctival congestion and meiosis. His fundoscopy, visual acuity and eye movements were normal The rest of his neurological examination was normal. There was no carotid bruit. There were no signs of meningism nor a rash. Examination of his cardiovascular, respiratory gastrointestinal systems was entirely normal. Blood tests showed a normal haemogram, renal profile, CRP and coagulation studies. His electrocardiogram and chest X ray were within normal limits.

CT Brain showed a small peripheral area of low attenuation in the right temporal lobe which raised the possibility of ischaemia (Figure 1). An urgent MRI brain showed a crescent shaped area (arrowed) of high signal representing a clot within false lumen of right internal carotid artery. (Figure 2)

Figure 1

Figure 1: CT Brain – low attenuation area in right temporal lobe

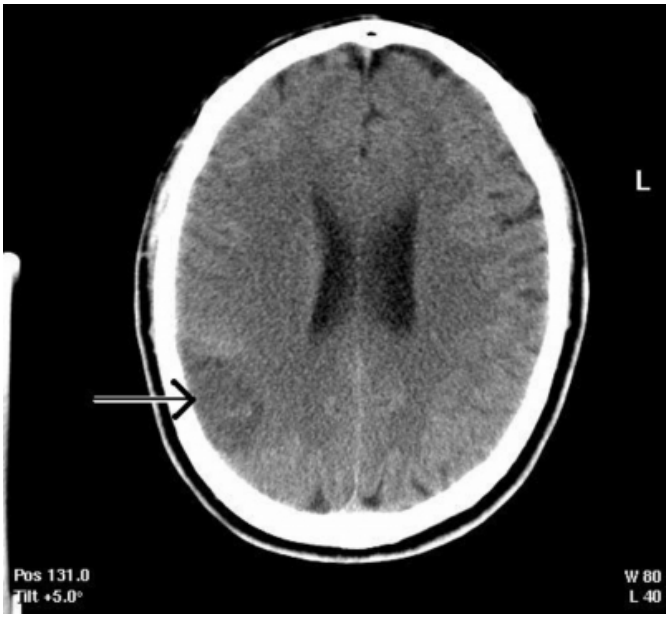
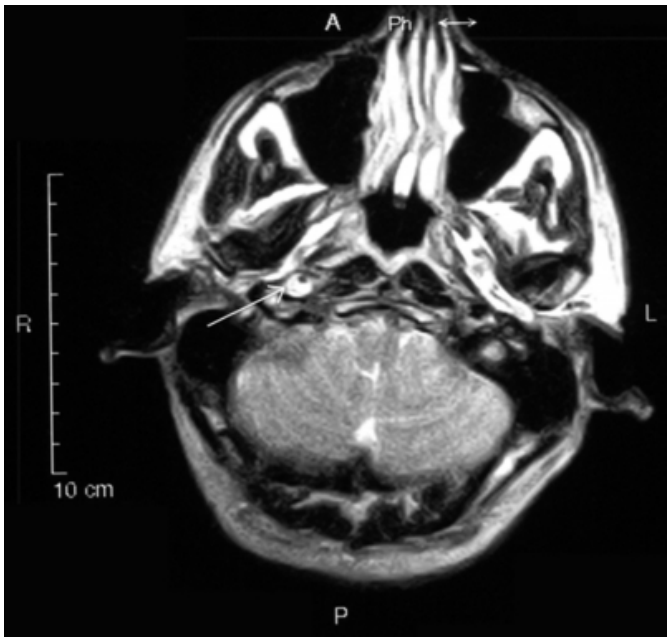


Figure 2

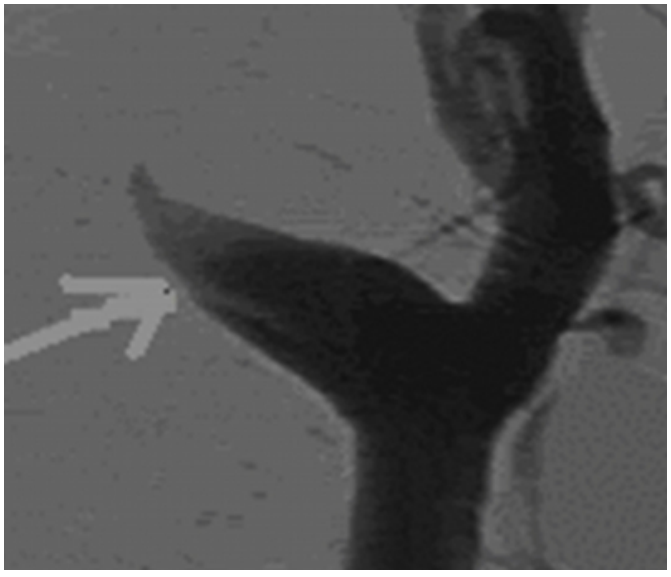
Figure 2: MRI showing right internal carotid dissection



The low signal area anterior to this is the signal void from the true lumen. There was normal void in the left internal carotid artery. This raised the strong possibility of carotid dissection which was confirmed by MR angiography. (Figure 3)

Figure 3

Figure 3: MRA



He was anticoagulated with warfarin and a repeat MR Angiogram in 6 months showed patent right internal carotid artery with normal flow.

DISCUSSION

Though spontaneous dissection of the carotid artery accounts for only about 2 percent of all ischemic strokes [1,2,3] but it is an important cause of ischemic stroke in young and middle-aged patients and account for 10 to 25 percent of such cases [3].

Historically it was thought that internal carotid artery dissection is associated with a clear history of neck trauma with resultant head and neck pain [4]. As seen from the above case, this is unfortunately not always true. A history of a minor precipitating event may be elicited in such patients and these instances have sometimes been colourfully called “bottoms-up dissection”[5] and “beauty-parlour stroke”[6]. Other precipitating events associated with hyperextension or rotation of the neck include practicing yoga, painting a ceiling, coughing, vomiting, sneezing, the receipt of anaesthesia or the act of resuscitation. It was also earlier thought that spontaneous dissection is usually associated with a connective tissue disorder such as Ehlers Danlos Syndrome Type 4, Marfan's Syndrome, Autosomal dominant polycystic kidney disease, Osteogenesis Imperfecta type 1 etc [7, 8]. In our patient nothing suggested underlying connective tissue disorder. One case control study also showed migraine to be a risk factor for carotid dissection [9].

The classical symptoms expected with carotid-artery dissection are unilateral head, face or neck pain accompanied by a partial Horner's syndrome (oculosympathetic palsy) and followed hours or days later by cerebral or retinal ischemia. This classic triad is found in less than 33% of patients, but if any 2 of the above triad are present, strong suspicions for carotid dissection should be aroused [4]. 50 – 95% of patients could have cerebral or retinal ischemic symptoms [10, 11]. Only about 20% of patients have an ischemic stroke without any warning signs [11].

Pain is usually the first manifestation of carotid-artery dissection and the other symptoms can take 4 days to appear [10]. About 25% of patients with a history of migraine mistake the headache to resemble a migraine, but most patients consider it to be unlike any other such pain [11, 12]. Oculosympathetic palsy though considered typical is only associated in less than half the cases [13,14,15]. About 12 % of patients with carotid-artery dissection may have cranial nerve palsies [11]. A quarter of patients complain of pulsatile tinnitus and a bruit may be present on auscultation[10,13,14,15].

The other differential diagnosis of painful Horner's syndrome includes cluster headache, tumours and Raeder's paratrigeminal syndrome.

Magnetic resonance techniques are replacing conventional angiography as the gold standard in the diagnosis of carotid artery dissection because the resolution of MRA can show the intramural haematoma itself [16, 17]. A combination of Doppler colour-flow imaging and transcranial ultrasonography provides the most useful information in the detection and follow-up of carotid-artery dissections [18]. An abnormal pattern of flow is identified in more than 90 percent of patients [18].

The prognosis is related to the extent of collateral circulation and the severity of the initial ischemic insult [4]. The reported rate of death is less than 5 percent, and about three quarters of patients who have had a stroke make a good functional recovery [13,15,19]. The associated headache resolves within a week in about 90 % of patients, but in some it can persist for many years.

The risk of a recurrent dissection in an initially unaffected artery is about 2 % during the first month but then decreases to a rate of only about 1 % per year [1]. However, the increased risk persists for at least a decade and possibly longer [20]. The risk of a recurrence is higher in young patients with a heritable arteriopathy [21]. Only rarely do dissections recur in the same artery [2,3,7].

To prevent thromboembolic complications, anticoagulation with intravenous heparin followed by oral warfarin has been recommended for all patients with acute dissection of the carotid artery, regardless of the type of symptoms, unless there are contraindications such as intracranial extension of the dissection [4]. Although antithrombotic treatment has been advocated since the 1970s, [13] no randomized trials have been reported, and the validity of such treatment has never been proved [22].

Most dissections of the carotid artery heal spontaneously. Surgical or endovascular treatment should be reserved for patients who have persistent symptoms of ischemia despite adequate anticoagulation [4]. Endovascular treatment, consisting of percutaneous balloon angioplasty and placement of one or more metallic stents, entails a lower risk than surgical treatment and, in most instances, has supplanted surgery as the initial therapy of choice once medical therapy fails [23,24]. However, the long-term results of carotid stenting are unknown, and the treatment of stent-related complications can be complex [25].

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Department of Medical Illustration - University Hospital of Hartlepool

CORRESPONDENCE TO

Dr Rajiv Sankaranarayanan MRCP University Hospital of Aintree Lower Lane, Liverpool L9 7AL United Kingdom
Email: rajiv-s@doctors.net.uk

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Author Information

Rajiv Sankaranarayanan, MRCP
Department of Internal Medicine, University Hospital of Hartlepool