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Case Report

Delayed Diagnosis of Traumatic Vertebral Artery Dissection after La Tomatina Festival

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Abstract

We present the case of a 50-year-old patient who was repatriated to the UK following treatment for lateral medullary syndrome in Spain. On further imaging a vertebral artery dissection was discovered. After a review of the history, the patient revealed that on the day before his symptoms started, he attended La Tomatina, a tomato throwing festival. He reported that he was hit in the back of the neck several times by tomatoes. Previous injuries reported at La Tomatina do not include vertebral artery dissection.[1] The rules of the festival state that tomatoes should be crushed prior to throwing to prevent injury.[2] This case highlights the high index of suspicion required when patients present with posterior stroke symptoms. If the presentation is associated with an injury the onset can be delayed and the mechanism may not be immediately obvious. Further questioning may be required when a vertebral artery dissection diagnosis is suspected.

***Keywords:** Stroke, Neurology, Cranial Nerves*

Introduction

Vertebral artery dissection (VAD) is a rare cause of stroke, accounting for 2% of all ischaemic strokes but is an important cause of stroke in young patients.[3] Although vertebral artery dissections can be spontaneous, they are often caused when hyperextension or rotational stresses are applied to the neck.[4] The case reported here is of a 50-year-old male patient, who presented after being treated for an ischaemic stroke in Spain.

Case Presentation

A previously well 50-year-old male patient presented with sudden onset ataxia, slurred speech and right arm sensory changes whilst on holiday in Spain. He was a non-smoker. The neurology team in Spain diagnosed him with lateral medullary syndrome and treated him with 300mg aspirin.

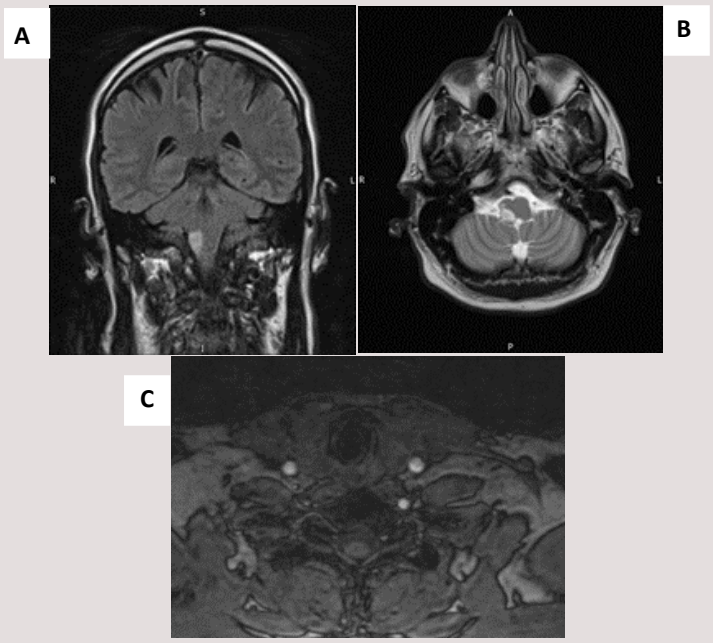
Ten days later, he was then repatriated to our centre in England. He reported ongoing symptoms of ataxia, dysphagia and double vision. On examination he had right sided hemifacial sensory loss. He had right sided ataxia and dysmetria with normal power. He had a left sided hemi-sensory deficit to pin prick. On examination of his cranial nerves he had horizontal nystagmus with fast phase to the right, right hemifacial sensory loss, left sided facial droop, dysphonia and his uvula was deviated to the left with no tongue deviation. This constellation of signs is in keeping with right lateral medullary syndrome. He underwent a flexible nasal endoscopy which confirmed right vocal cord palsy.

On further review of the history, the patient revealed that on the day before his symptoms started, he went to La Tomatina, a tomato throwing festival in Spain. He reported that he was hit in the back of the neck several times by tomatoes, some of which were quite firm, resulting in local discomfort in that area.

Investigations

In Spain, the patient had a CT head which showed no signs of acute intracranial haemorrhage and no acute ischaemia. He also had a CT angiogram which showed a replenishment defect in V4 segment of vertebral artery, an intact circle of Willis, and findings in keeping with a posterior circulation stroke. The translated reports of these images were only made available to us several days after repatriation.

On arriving at our centre in the UK, we requested an MRI head (Figure 1 & 2) and MRA neck (Figure 3) which showed an established right lateral medullary infarct with no acute ischaemia. There was poor flow within the right vertebral artery with a very narrow calibre from C6 to the skull base, in keeping with a vertebral artery dissection.



A: Fig:1 *MRI head (coronal flair): focal high T2 signal in the right lateral medulla in keeping with established right lateral medullary infarct*

B: Fig:2 *MRI head (T2): focal high T2 signal in the right lateral medulla in keeping with established right lateral medullary infarct*

C: Fig:3 *MRI head (T2): focal high T2 signal in the right lateral medulla in keeping with established right lateral medullary infarct*

Differential Diagnosis

Even though the diagnosis and apparent cause was obvious from history, examination and neuroimaging, we requested additional investigations to exclude other causes of stroke in the young. Unsurprisingly, these additional tests did not reveal any alternative aetiology or risk factor for this patient's stroke.

Based on this gentleman's presentation and examination findings, differential diagnoses would include anterior inferior cerebellar artery (AICA) occlusion and medial medullary syndrome. AICA infarcts are associated with hearing loss which this patient did not have.[5] Medial medullary syndrome is caused by a thrombotic or embolic occlusion of small perforating branches from vertebral or proximal basilar artery.[6] Medial medullary syndrome causes contralateral hemiparesis and hemi-sensory loss, associated with ipsilateral lingual palsy because of the involvement of cranial nerve XII. Medial medullary syndrome can also cause ipsilateral flaccid hemiparesis and can be associated with vertigo and nausea.[6]

Once a diagnosis of vertebral dissection is established with neuroimaging, differential diagnoses include spontaneous dissection and traumatic dissection. This may be established from the history. Underlying causes for dissection need to be considered including connective tissue disorders.

Treatment

This patient was managed according to the unit protocol for acute stroke. He was treated with tab aspirin 300mg for 2 weeks and then tab clopidogrel 75mg to continue lifelong. He also had significant input from the stroke therapy team including physiotherapists, occupational therapists and speech and language therapists. Initially, his swallow was poor which necessitated the insertion of a nasogastic tube for nutritional support. His swallow subsequently improved after a few days and the nasogastric tube was discontinued. Furthermore, he had ongoing issues with hiccups and was started on pregabalin, 150mg twice daily, to good effect.

Outcome

He continued to improve clinically and was discharged to a community hospital for ongoing stroke rehabilitation prior to discharge home after 26 days of admission. His Modified Rankin score was 3 at the point of discharge.

Results & Discussions

Vertebral artery dissection (VAD) is caused when the wall of the artery tears, allowing blood to collect within the arterial wall forming an intramural haematoma. Haemodynamic insufficiency, due to direct stenosis of the lumen or by thromboembolism, to areas of the brain supplied by this artery results in an ischaemic stroke.[7,8] Extension of the dissection can lead to subarachnoid haemorrhage; this is associated with intracranial VAD.[4] VAD can be spontaneous and associated with connective tissue disease but are often related to trauma. Traumatic VAD has been documented following various mechanisms of injury, including activities include hair washing at the hairdressers, yoga, sneezing, nose blowing and car accidents. Due to the relatively low impact trauma which can cause a dissection, identifying the preceding trauma can frequently be missed.[8]

Clinical manifestations of VAD include severe neck and head pain which can be followed by ischaemic symptoms (transient ischaemic attack or stroke).[4] The symptoms of pain can precede the neurological symptoms by hours to days and there have been case reports of delays between the trauma and the onset of the pain.[8] Lateral medullary and cerebellar infarctions are the most common type of strokes associated with VAD, presenting with vertigo, dizziness, double vision, ataxia and dysarthria. Diagnosis of dissection is made with MRI/MRA or CT angiography.

Treatment of vertebral artery dissection can include antiplatelet medication or anticoagulation. Cervical Artery Dissection in Stroke Study (CADISS) trial randomised patients with extracranial carotid or vertebral dissections to either antiplatelet treatment or anticoagulation with heparin and then warfarin. This study proved no difference in the rates of recurrent ischaemic stroke at 3 months or major bleeding between the 2 treatment arms.[9] Previous meta-analysis of these treatments also showed no difference in efficacy or complications between antiplatelet medication and anticoagulation.[10] Recommended treatment duration is 3-6 months.[9] There was insufficient evidence to assess the safety and efficacy of stenting or thrombolysis.[10]

La Tomatina is a festival that happens annually in Bunol, a village near Valencia, Spain. It is a one-day festival that began in 1945 and uses 160,000kg of red tomatoes.[11] Local residents and tourists throw tomatoes at each other over the course of the day. The rules of the festival state that tomatoes should be ripe and crushed prior to throwing them to prevent injury.[2] Previously documented injuries obtained during La Tomatina include eye irritation, minor trauma, heat stroke and alcohol poisoning.[12]

Conclusion

This case highlights the high index of suspicion required when patients present with posterior stroke symptoms and pain. Vertebral artery dissection (VAD) is an important cause of stroke in young patients. If VAD is associated with an injury the onset can be delayed and the mechanism of injury may not be immediately obvious on first review.

Further questioning may be required when a diagnosis of vertebral artery dissection is made.

Conflict of Interest

The authors declare no conflict of interest.

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