CASE REPORT

Symptomatic Vertebral Artery Occlusion in a Young Nigerian Male: A Case Report with Review of Current Literature

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ABSTRACT
Although vertebral artery occlusion is a recognized cause of posterior circulation stroke especially in the young adult population, this is the first report of a magnetic resonance angiography proven case of the disease in a Nigerian. Our case report illustrates the challenges that clinicians are faced with in the diagnosis of this condition and provides a synthesis of the current treatment nuances through a review of relevant literature.

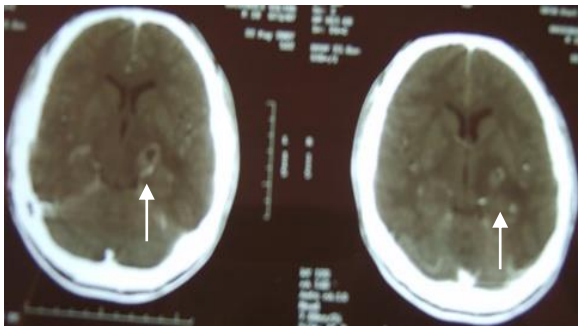
CASE REPORT
A 19 -year old right handed Nigerian male was admitted with recurrent left occipital headache of 2weeks' duration, associated with right hemi-body weakness and slurred speech. Symptoms started during a football game practice with a nodding of the ball. Few hours prior to admission, he was, also, involved in a passenger car crash but sustained no visible injuries. There was no history of vertigo, vomiting, or numbness in the extremities.

Physical examination revealed a drowsy young adult male in, otherwise, good general state of health. His Blood Pressure was 120/70mmHg, pulse rate was 70/min, respiratory rate was 22/min, and temperature was 36.5^{0}C. Glasgow Coma Score was 14 (*E3, V5, M6*), and he had orofacial apraxia and slurred speech. The right pupil was 2mm in

size, while the left was 4mm and both pupils reacted sluggishly to light. There was right homonymous field extinction, and ophthalmoscopy revealed normal fundi. Other cranial nerve examinations were unremarkable and there were no signs of meningism. He had a spastic quadriparesis which was worse on the right, motor power was (Medical Research Council, MRC), grade 3 in the muscle groups of the right extremity and grade 4 on the muscle groups on the left. He had a left appendicular ataxia and fine rotary nystagmus. No other cerebellar signs were found. Systemic evaluation revealed normal findings.

Full blood count was normal, erythrocyte sedimentation rate was 14mm/1st Hr (*Westergreen method*), platelet count, coagulation profile and lipid profile were in the normal range, and International Normalized Ratio (*INR*) was 1.16. Screening for vasculitis showed Rh factor negativity and absent LE cells. He had an oligoclonal band (OB) negative cerebrospinal fluid assay, and brain CT scan showed a ring enhancing left thalamocapsular hypodense lesion (Figure 1).

Figure 1. Cranial CT showing acute thalamocapsular infarct



Brain MRI scan showed a hyperintense left thalamocapsular lesion with perilesional hypointensity on T1WI imaging (Figure 2), which was hypointense on T2WI, with peri-lesional hyperintensity (Figure 3).

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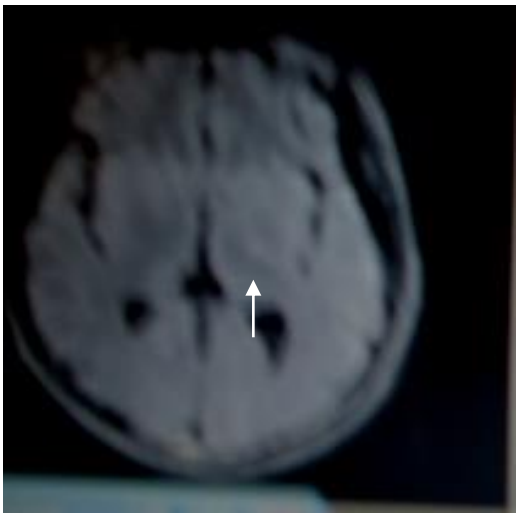
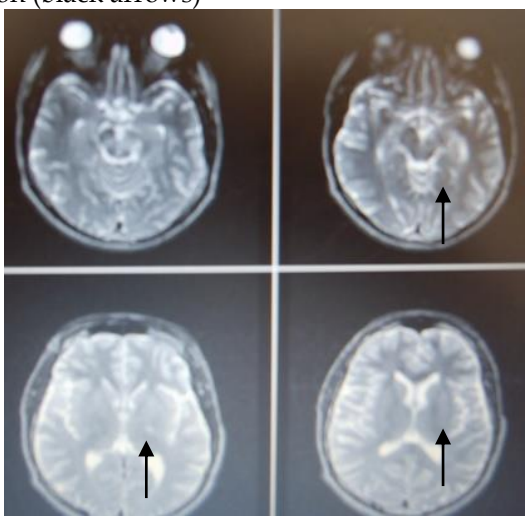
Figure 2. Cranial MRI (TIWI) showing thalamocapsular infarct



Carotid and vertebral Doppler ultrasonography showed normal right vertebral and bilateral carotid artery flow signals. However, the left vertebral artery flow signal was absent.

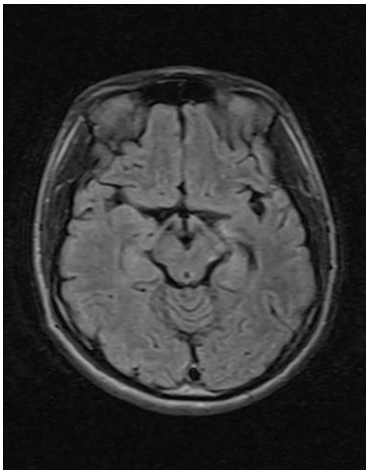
3. Cranial MRI (T2WI) shows the thalamocapsular lesion (black arrows)



The patient was treated non-operatively with a blend antiplatelet therapy using aspirin and dipyridamole, as well as speech and limb physiotherapy. There was a good clinical response with resolution of headaches, recovery of consciousness, resolution of visual field deficits and improvement in limb

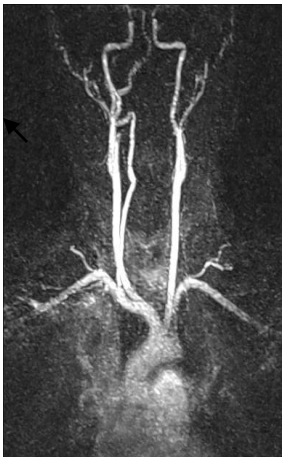
motor power. He progressed from inability to walk on admission to ambulation with a frame after one week therapy. He was discharged following a 2-week admission period when he became ambulant using a cane and regained full unaided ambulation at 3 months' follow-up.

Figure 4. Cranial MRI (T1WI) shows a left thalamopeduncular infarct



A follow-up brain MRI (Figure 4), at 3 months, showed a complete resolution of the left thalamocapsular lesion. Magnetic resonance angiography, MRA, showed the absence of the left vertebral artery (Figure 5).

Figure 5. An MRA showing the absence of left vertebral artery



DISCUSSION

Our index case suffered a left vertebral artery occlusion (VAO) after nodding the ball during a soccer game with resultant cerebellar, brain stem and left thalamic signs. Good clinical judgement aided by a high index of suspicion was combined with cranial CT, MRI and MRA, as well as Doppler ultrasonography in confirming the diagnosis of extracranial VAO with distal thromboembolism. With multi-disciplinary care, anti-platelets therapy for 6months and physiotherapy, our patient experienced a good neurological outcome. Most cases of VAO that were previously reported have occurred secondary to vertebral artery dissection (VAD).[1] Although vertebral artery dissection (VAD) is a rare cause of stroke accounting for only 0.5-2.0% of all cerebrovascular accidents generally, it is currently recognized as a common cause of vertebrobasilar stroke in the young adult population as typified by our 19-year old index patient.[2] It can occur spontaneously or following trauma and is, also, the most common cause of cerebellar infarcts in the age group below 40years.[1,2]

The VAO of our index patient is classified as spontaneous because it occurred following an, otherwise, innocuous event or activity of daily living such as turning the head to nod the ball during a sport. Other innocuous events associated with spontaneous VAO include driving, painting, nose blowing, yoga, etc. [3, 4]

Most reported cases of traumatic dissection have, however, occurred following chiropractic manipulations, while other causes include falls, road traffic accidents and penetrating injury.[4]

Vertebral dissections are typically of 4 types (V_1-V_4).[6] Extracranial dissection includes:

V_1 - from origin to entry into the transverse foramen;
V_2 - within the foramen from C6-C2;
V_3 - after exit from the foramen;

Intracranial or V_4 dissection occurs after the vessel has pierced the dura.[4]

The occurrence of cerebellar, brainstem and thalamic signs in our index patient, when correlated with absence of the left vertebral signal on Doppler ultrasound and non-visualization of the left vertebral artery on magnetic resonance angiogram highly

suggested an extracranial dissection with associated distal thromboembolism.

An expanding intramural haematoma is the underlying lesion in the evolution of VAD.[5, 6] It arises as bleeding from the *vasa vasorum* within the media of the vessel wall, either spontaneously or following trauma. The haematoma may seal off and, if it is small, may remain asymptomatic. If the dissection is sub-intimal, it may partially or completely occlude the vertebral artery or one of its branches, or may become a trigger for distal thromboembolism with brain stem and thalamic infarctions, as occurred in our index case. Rarely, extracranial dissections may extend intracranially to involve the basilar artery.[5, 6]

Diagnosis of VAD requires a high index of clinical suspicion triggered by a suggestive clinical presentation.[6] Occipital headaches, as noted in our patient, and neck pain are the most common symptoms occurring in 60-70% of cases.[4, 6] There may, as well, be a history of neck manipulation or trauma. A disabling neurologic syndrome - the lateral medullary syndrome, which is also referred to as Wallenberg's syndrome - occurs typically with involvement of the postero-inferior cerebellar artery. Patients may also present with medial medullary syndrome, cerebellar /upper brainstem deficits, or, rarely, thalamic stroke syndrome from distal embolism, as was noted in our patient. [7]

Diagnosis of VAD is confirmed by vertebral angiography. Findings include luminal narrowing, occlusion, double lumen, pseudoaneurysm formation or a characteristic 'string sign'.[6, 7]

Cranial CT scan may show sequelae of vertebral dissection such as subarachnoid hemorrhage or infarction. Color Doppler/Duplex ultrasonography may show absence or reduction or abnormal flow signals as observed in the index case. Transcranial Doppler may detect High Intensity Transient signals (HITS), which represent distally propagating emboli. [8] Cranial MRI/MRA provides a non-invasive imaging modality and has become the imaging facility of choice.[3,5] Findings on MRI may include intimal flap and intramural haematoma, whereas MRA may, in addition, show an abrupt cut-off of flow signal at the site of occlusion.

With cranial MRI features, patients with VAD can be classified into three groups:

the first group involves symptomatic cases with radiologic evidence of ischaemia as was the case in the index patient;

the second group involves symptomatic cases without radiological evidence of cerebrovascular ischaemia;

the third group belongs to incidental findings on MRI for unrelated presentation.

Whereas treatment is indicated in the first group, decision on treatment for the remaining groups is based on the presence and age of the clot on one hand and severity and duration of clinical symptoms on the other.[3,5, 9,10, 11]

Treatment for VAD is mainly non-operative. For extracranial VAD, the options include:

1. Antiplatelets - aspirin and dipyridamole. Aspirin is currently the standard treatment for acute stroke.
2. Anticoagulants are not associated with reduction in early mortality or recurrence and may increase the risk of haemorrhagic transformation. However in spontaneous extracranial VAD without SAH, anticoagulants may be beneficial in reducing recurrent ischaemic events.[11]

Anticoagulant and antiplatelet therapy are usually continued for ½-2years during which recanalisation is expected to occur.[11]
Surgery for VAD includes open procedures such as vertebral clipping or ligation with or without bypass, resection with autogenous interposition vascular graft and wrapping especially with repeated ischaemic events or endovascular treatment with stent placement,

coiling, clips or balloons.[10,11] Previous experience has shown that with appropriate diagnosis and surgical correction of carefully selected cases, complete resolution of haemodynamic and embolic symptoms can occur predictably.[10, 11, 12, 13]

The main option for surgical treatment of proximal vertebral artery occlusion (V_1 segment) is transposition of the vertebral artery to the common carotid artery.[9,10, 11] Vertebral artery transposition has been reported to offer an effective and safe emergent treatment for post-traumatic VAD following penetrating head trauma.[10]

Most cases of VAD generally run a benign course with recanalisation of the vessel in the ensuing weeks and months. However, extension into the vertebral artery and recurrent ischaemic events are risk factors for a poorer outcome.[13, 14, 15] Vertebral dissection is rarely reported among children and there are currently no guidelines on the treatment of vertebral occlusion in children. Recommendations by the American Heart Association (AHA) for treatment of children with cervicocephalic arterial dissections are Classes IIa and IIb (level of evidence C) deriving mainly from adult studies.[11, 12, 13, 14, 15] Nonetheless, a multi-disciplinary team approach to care has been suggested as key to attainment of the best outcomes in children with VAD from the time of admission to the end of follow-up therapy.[11, 14, 15]

CONCLUSION

We have in this report highlighted the role of good clinical judgement aided by neuroimaging in the prompt diagnosis of vertebral artery occlusion. Although there are a few recommendations, there are currently no clear guidelines based on scientific evidence for treatment of VAO in children.[11, 15] All existing guidelines are based on the adult series.

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