Journal of Heart and Stroke

Case Report
Published: 26 Jun, 2020

Quadriparesis and Seizure from Bilateral Anterior Cerebral Artery Infarction: A Case Report

Samson Erkabu[1], Biniam Barega[1], Hailu Dessallegn[2] and Eyoel Negash[2]*

[1]*Department of Internal Medicine, Ras Desta Damtew Memorial Hospital, Ethiopia*

[2]*Department of Epidemiology and Biostatistics, Ras Desta Damtew Memorial Hospital, Ethiopia*

Abstract

Bilateral Anterior Cerebral Artery (ACA) territory infarction is not a common phenomenon. It is commonly a result of vasospasm of vessels following subarachnoid hemorrhage in the territory of ACA or it could be because of thrombosis. Patients with bilateral anterior infarction have different clinical presentations. We report a patient who comes with quadriparesis and seizure after bilateral ACA infarction.

Keywords: Bilateral cerebral infarction; Anterior cerebral artery; Quadriparesis and seizure

Introduction

Anterior Cerebral Artery (ACA) territory infarcts are much less common compared to either middle or posterior cerebral artery territory infarcts. Infarctions of the ACA and its branches account for 0.3% to 4.4% of stroke cases reported in different series [1]. The low rate of infarcts in this vascular distribution could be partly a result of significant vascular anastomosis [2].

The incidence of Bilateral ACA infarction is even rare. Among the 27 patients with ACA infarction in the Lausanne Stroke Registry, only two of them had bilateral ACA territory infarction [3]. One study involving 48 patients with ACA infarction had also only two cases of bilateral ACA infarctions [4]. Here we report a man who presents with seizure and quadriparesis after he suffered bilateral anterior cerebral artery infarction.

OPEN ACCESS

Correspondence:
Samson Erkabu, Department of Internal Medicine, Ras Desta Damtew Memorial Hospital, A Community Hospital at Addis Ababa, Ethiopia,
E-mail: samigetch@yahoo.com

Received Date: *02 Jun 2020*
Accepted Date: *17 Jun 2020*
Published Date: *26 Jun 2020*

Citation:
Erkabu S, Barega B, Dessallegn H, Negash E. Quadriparesis and Seizure from Bilateral Anterior Cerebral Artery Infarction: A Case Report. J Heart Stroke. 2020; 5(1): 1059.

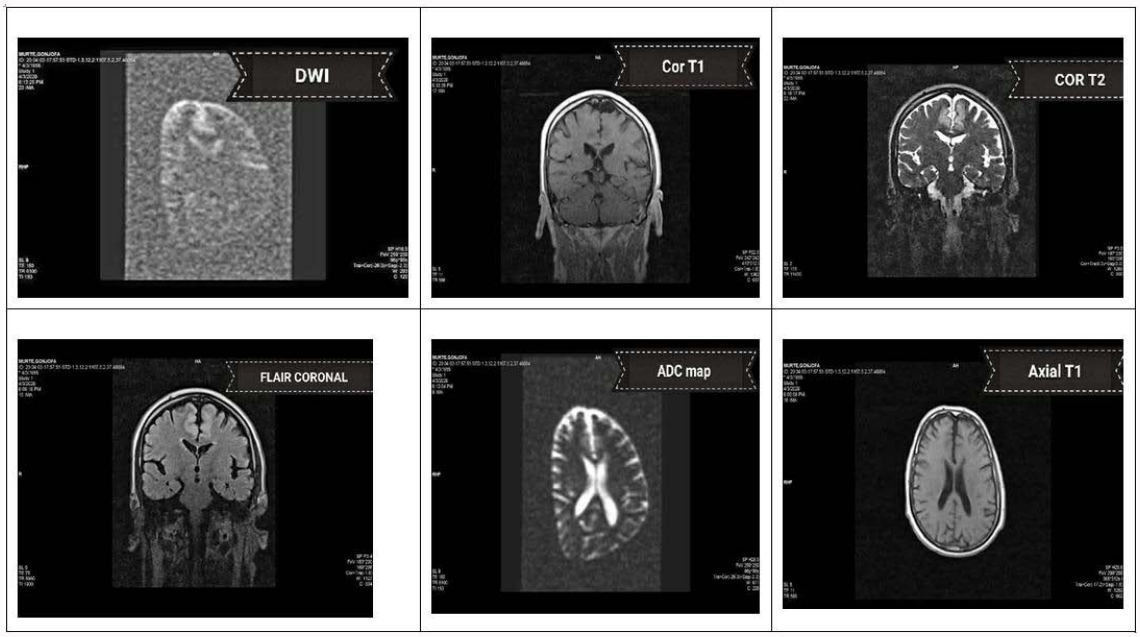
ISSN: 2475-5702

Case Presentation

A 65 years old male diabetic and hypertensive patient on intermediate insulin, Enalapril, Aspirin, and Atorvastatin comes to a health center with sudden onset of vomiting that is followed by a generalized tonic-clonic seizure. The seizure occurs three times and the patient didn't regain his consciousness between the episodes. His vital signs at the health centers were BP 200/100 mmHg, tachycardic, and tachypneic with random blood glucose of 176 mg/dl. The patient was transferred to Ras Desta Damtew Memorial Hospital with Ambulance. At the emergency room of the hospital, he was loaded with Phenytoin and phenobarbital, after the airway was secured. With the impression of stroke computed tomography was done. The CT finding was unremarkable. The second day MRI was done and the patient has bilateral ACA infarction. The seizure was controlled but the patient was lethargic and he was transferred to the adult intensive care unit. At the ICU he maintained his oxygen saturation with a face mask of oxygen; he also received empiric antibiotics and his routine medications.

The blood works including complete blood count, renal function test, and electrolytes and coagulation profiles were normal. His Doppler ultrasound of the extracranial carotid arteries was unremarkable. His echocardiography showed a moderate concentric left ventricular hypertrophy with grade one diastolic dysfunction. Below is magnetic resonance imaging of his brain that showed bilateral parafalcine region of frontal lobe flair and T2W hyperintensity which shows restricted diffusion on DWI sequences. Interpreted as bilateral ACA territory infarctions (Figure 1). Subsequent days at ICU he started to regain his conscious but this time he was having jerking movement of the head. The neurologic examination revealed a fully awake individual with no language disorder but was slow and had a delayed response for a stimulus with a lack of spontaneity and a quadriparesis. The following was his motor examination findings.

Clonazepam was given for the jerking movement and it improved gradually. After a week in general medical ward patient showed an improvement, the strengths of his extremities increased



Figur 1: MRI of the brain.

Table 1: Motor examination of the extremities.

	Upper right extremity	Lower right extremity	Upper left extremity	Lower left extremity
Strength	1/5	1/5	1/5	1/5
Reflex	1/4	2/4	1/4	2/4
Tone	Decreased	Decreased	Decreased	Decreased
Fasciculation	None	None	None	None
Plantar reflex		Equivocal		Equivocal

and there was no seizure. The slowness and response to stimuli were not much improved. The patient was discharged home with his routine drugs and anti-epileptics (Table 1).

Discussion

The mechanisms of ACA infarctions are similar to other vascular territory infarctions. A review on the occurrence and mechanism of occlusion of ACA infarction revealed that infarction in this vascular territory could be a result of emboli; propagation of thrombotic material from an occluded internal artery into the intracranial branches and Spasm [5]. Other Reports showed 63% of ACA infarctions result from cardiogenic emboli or artery-to-artery emboli [3]. Kang SY and Kim JS evaluated 100 patients with ACA infarctions and reported atherosclerosis as the most common cause of ACA stroke [6-13].

Bilateral ACA territory infarction could usually be due to vasospasm that occurs as complication of subarachnoid hemorrhage caused by the rupture of one or more aneurysms of the anterior communicating arteries or distal ACAs [6]. Another cause is thrombosis of the proximal part of one anterior cerebral artery when the contralateral proximal branch is rudimental or absent.

In our patient, intracranial atherosclerosis (either intracranial internal carotid or ACA) is the more likely etiology given his underlying hypertension and diabetes. We found no evidence of bleed to suggest SAH that could lead to vasospasm. We also believe magnetic resonance arteriography should have been taken to see any vessel anomaly; but it was not readily available at the hospital.

The symptoms that accompany bilateral infarction of the anterior cerebral artery territory differ from those caused by unilateral infarction, particularly the profound mental changes [8,9]. The clinical features of patients with bilateral ACA infarction are variable. The most consistent findings were frontal disinhibition signs such as enhanced glabellar tap, utilization behavior, forced grasping, snout, and other primitive reflexes [7]. Paraparesis and akinetic mutism were also documented as a result of bilateral ACA stroke [4]. In 2004, Yamaguchi et al. [12] reported a similar case with a patient presenting with lower limb weakness and magnetic resonance angiography demonstrating bilateral anaplastic ACAs [12]. A case with simultaneous bilateral cerebral infarction was reported with features of a space-occupying lesion [11].

Anterior circulation stroke can cause quadriplegia similar to infarcts of the posterior circulation. It may even mimic a basilar artery occlusion [10]. When it results in quadriparesis the infarction usually involves a significant portion of the upper cerebral convexity. Cases of quadriplegia due to ACA territory infarct of unpaired Azygous ACA were reported [14,15].

Conclusion

Despite the rarity patient with bilateral ACA infarction, may present with seizure and quadriparesis and bilateral ACA infarction could be a differential diagnosis for sudden onset of quadriparesis.

Ethical Issues

Permission to report the case was given by the patient care takers after discussing and consent was made.

Acknowledgement

We would like to acknowledge Mehari G/yohannis (MD, Associate Professor of Neurology at University of Texas Southwestern Medical Center) for his support in the writing of the case report.

References

1. Matos Casano HA, Tadi P, Ciofoaia GA. Anterior cerebral artery stroke. StatPearls Publishing. 2020.
2. Kubis N, Guichard JP, Woimant F. Isolated anterior cerebral artery infarcts: A series of 16 patients. Cerebrovasc Dis. 1999;9(3):185-7.
3. Bogousslavsky J, Regli F. Anterior cerebral artery territory infarction in the Lausanne Stroke Registry. Clinical and etiologic patterns. Arch Neurol. 1990;47(2):144-50.
4. Kumral E, Bayulkem G, Evyapan D, Yunten N. Spectrum of anterior cerebral artery territory infarction: Clinical and MRI findings. Eur J Neurol. 2002;9(6):615-24.
5. Gacs G, Fox AF, Barnett HJ, Vinuela F. Occurrence and mechanisms of occlusion of the anterior cerebral artery. Stroke. 1983;14(6):952-9.
6. Orlandi G, Moretti P, Fioretti C, Puglioli M, Collavoli P, Murri L. Bilateral medial frontal infarction in a case of azygous anterior cerebral artery stenosis. Ital J Neurol Sci. 1998;19(2):106-8.
7. Kobayashi S, Maki T, Kunimoto M. Clinical symptoms of bilateral anterior cerebral artery territory infarction. J Clin Neurosci. 2011;18(2):218-22.
8. Wood JH. Cerebral Blood Flow: Physiological and Clinical Aspects. 1987;24-5.
9. Freedom FR. Akinetic mutism and bilateral anterior cerebral artery occlusion. J Neurol Neurosurg Psychiatry. 1971;34(6):693-8.
10. Ferbert A, Thron A. Bilateral anterior cerebral artery territory infarction in the differential diagnosis of basilar artery occlusion. J Neurol. 1992;239(3):162-4.
11. Menezes BF, Cheserem B, Kandasamy J, O'Brien D. Acute bilateral anterior circulation stroke due to anomalous cerebral vasculature: A case report. J Med Case Reports. 2008;2:188.
12. Yamaguchi K, Uchino A, Sawada A, Takase Y, Kuroda Y, Kudo S. Bilateral anterior cerebral artery territory infarction associated with unilateral hypoplasia of the A1 segment: Report of two cases. Radiat Med. 2004;22(6):422-5.
13. Kang SY, Kim JS. Anterior cerebral artery infarction: Stroke mechanism and clinical-imaging study in 100 patients. Neurology. 2008;70(24 Pt 2):2386-93.
14. Saleh C, Michel-de Cazotte E, Hund-Georgiadis M. Tetraparesis following an anterior circulation stroke: A case report. Case Rep Neurol. 2018;10(3):342-5.
15. Rajasekharan C, Deepak M. Acute onset quadriplegia. BMJ Case Rep. 2012;2012:bcr0720114472.