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Case Report

A Rarity in a Rare Case of Acute Stroke

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Abstract

Anterior circulation strokes involving the anterior cerebral artery territory is rarer compared to middle cerebral artery territory and especially bilateral anterior cerebral artery (ACA) territory involvement is still rarer. A middle aged chronic alcoholic and smoker male non-diabetic, non-hypertensive was brought to us for acute onset of decreased responsiveness of 8-10 hours duration. On examination, he was quadriplegic, mute and had preserved eye movements and intact brain stem reflexes. Brain imaging revealed acute infarct in bilateral anterior cerebral artery territory. Patient was treated with medical measures and the patient sensorium improved with disability in walking.

Keywords: Akinetic Mute; Quadriplegia; Acute Stroke; Anterior Cerebral Artery territory; Bilateral ACA

Abbreviations

ACA: Anterior Cerebral Artery; GCS: Glasgow Coma Scale; CT: Computed Tomography

Introduction

ACA territory infarcts are rare accounting for about 0.3% to 4.4% of total cerebral infarctions, of which bilateral ACA infarcts are still much rarer. In the stroke registry of E Kumaral., *et al*, 1.3% of 3705 patients of ischemic stroke represent ACA infarction. Here we present a rare case of quadriplegia due to bilateral anterior cerebral artery infarction.

Case Report

A 48 year old man, chronic smoker and alcoholic, nondiabetic, non-hypertensive was brought to the casualty with acute onset of decreased responsiveness by his wife in the evening on the same day. He went to bed normally the previous night, got up in the morning and got his daily chores done normally including breakfast. There was no history of headache, vomiting, fever, seizures, diarrhea, vomiting or trauma. There was no history of bowel or bladder incontinence. There was no history of prior hospitalization or recent alcohol binge. On examination, the patient had BP 140/100 mm Hg, pulse rate 90/ min, was not in respiratory distress and was drowsy, easily arousable, responding to painful stimuli but not to calls or commands, GCS score was E4V1M4 (9/15). Spontaneous eye opening was preserved with Dolls eye movement manoeurable in both horizontal and vertical planes. Bilateral pupils were 3 mm equal and reacting to light. Bilateral corneal and conjunctival reflexes were intact. There was no facial weakness. Spinomotor system examination showed hypotonia in all four limbs. MRC grading of power was 1/5 in all four limbs. Deep tendon reflexes on both sides were just present. Abdominal reflex was absent and plantar reflex was extensor on both sides. Sensory system examination could not be done as he was drowsy. Apraxia could not be tested because of weakness. Primitive reflexes were not present. There was no spinal tenderness or deformity. The Fundus examination was normal. The

cardiovascular system examination was clinically normal. All basic investigations including complete blood count, serum electrolytes, liver function tests, renal functions and lipid profile were normal. Carotid – vertebral Doppler and cardiac evaluation was within normal limits. The patient was started on Inj. Thiamine and his sensorium improved partially. CT Brain showed acute infarct in bilateral anterior cerebral artery territory. MRI Brain showed acute infarct in bilateral para median fronto parietal cortex and entire corpus callosum with hemorrhage in right putamen and left central pons. MRA showed thrombosis in bilateral anterior cerebral arteries.

Patient was treated with antiplatelets, statins, physiotherapy and other supportive measures.

Discussion

At the initial presentation, for a middle aged male, chronic alcoholic and smoker, presenting with acute onset quadriplegia with mutism and preserved eye movements and intact brainstem reflexes, the differential diagnosis considered were Wernicke's Encephalopathy, Osmotic demyelination syndrome, Basilar artery occlusion, non-convulsive status epilepticus, spinal dural arterio-venous fistula, acute transverse myelitis, atlanto-axial dislocation, cervical epidural hematoma and fracture of upper cervical vertebra. Our patient being an alcoholic, with background malnutrition can present less commonly with spastic paresis due to thiamine deficiency and hence Wernicke's Encephalopathy was considered here. Osmotic demyelination syndrome is typically present with paraparesis or quadriparesis and rarely despite correction of serum sodium according to proposed guidelines. Our patient however, did not have recent history of hospitalization as well as serum electrolytes were within normal limits, hence osmotic demyelination syndrome was unlikely. Basilar artery occlusion causes damage to bilateral ventral pons causing quadriplegia, inability to speak or swallow and with impaired horizontal eye movements but with preserved vertical eye movements and blink response. In this patient, since the horizontal eye movements were preserved, basilar artery occlusion was considered to be less likely. Non convulsive status epilepticus manifests primarily as altered mental status as opposed to dramatic convulsions seen in generalized tonic clonic status epilepticus and thus was considered here. Upper spinal cord lesions like spinal dural AV fistula presents as chronic myelopathy unless there is an hemorrhage. However, the presence of altered sensorium and mutism in this patient ruled out this entity. Acute transverse myelitis in this patient was ruled out because of hyperacute presentation, mutism and unresponsiveness. Although atlanto-axial dislocation occurs in almost all age groups, it is more common in adolescents and presents with pyramidal signs, numbness, neck pain and neck movement restriction. We considered this diagnosis to be least possible because of atypical age, no neck pain and no neck movement restriction in our patient [1-5].

Further, investigating with CT Brain showed hypodensity in bilateral paramedian frontal and parietal regions. MRI Brain showed diffusion restricting T2 FLAIR hyperintensity in bilateral paramedian fronto parietal cortex, and entire corpus callosum. MRA showed no flow in bilateral anterior cerebral artery. Patient was treated with aspirin, statins, physiotherapy and other supportive measures. Patient improved clinically when followed up after about 3 months. The territory supplied by anterior cerebral artery includes rostrum, genu, trunk and splenium of corpus callosum, cingulate gyrus, frontal pole, medial aspect of superior frontal gyrus, supplementary motor area, paracentral lobule and precuneus. The presumed etiology for anterior cerebral artery disease includes anterior cerebral artery atherosclerosis with or without internal carotid artery occlusion and cardiogenic embolism. According to a study by Suk Y Kang et al, the presentations of ACA infarct included motor dysfunction, acute confusion, apathy, amnesia, urinary incontinence, aphasia, ideomotor apraxia, sensory deficits, parkinsonian features, anosognosia. Inspite of motor dysfunction being the most common presentation, majority presented with hemiparesis followed by monoparesis and paraparesis, with none presenting as quadriparesis. The manifestations of bilateral ACA infarct are akinetic mutism, abulia and utilization behaviour due to involvement of frontal pole and cingulated gyrus, urinary and fecal incontinence due to paracentral lobule, quadriparesis due to supplementary motor area and paracentral lobule and also with personality and depressive mood changes.

Conclusion

MCA territory infarction and ACA territory infarction can present with similar clinical features and may have the same etiological spectrum, but frontal lobe dysfunction features and collosal dysfunction can make a difference in the diagnosis. Bilateral ACA territory infarction patients are dependant on the activities of daily living for a longer duration of time.

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None.

Conflict of Interest

Nil.

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