

## An unusual case of paraplegia in a middle-aged male

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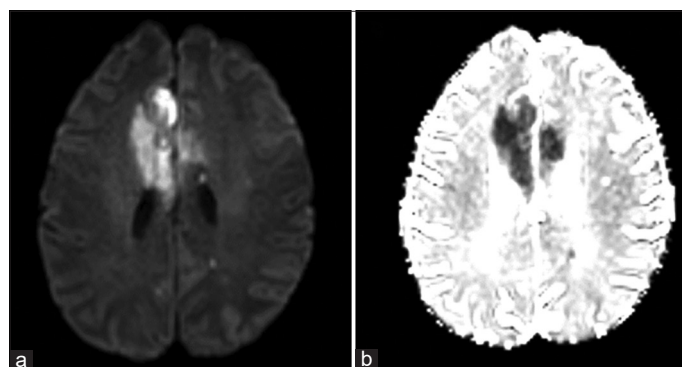
Infarcts involving the territory of the anterior cerebral artery (ACA) are rare and account for a smaller number of ischemic strokes [1]. The clinical presentation is however very varied and unusual presentations are known to occur. Hence, the diagnosis of these strokes is often made late or missed completely. We present here a case of a middle-aged male patient who presented with sudden onset of lower limb weakness and was found to have ACA territory involvement.

This 49-year-old male patient was a known case of hypertension and diabetes mellitus for more than 5 years on regular treatment. He presented to the emergency with a 2-day history of sudden onset of weakness of both lower limbs along with paresthesia. He also had urinary retention on the day of the presentation. The weakness progressed rapidly to involve both lower limbs and he was unable to walk without support. There was no upper limb weakness.

General examination was unremarkable. Neurological examination revealed complete flaccid paraplegia of both lower limbs (power=0/5) with absent deep tendon reflexes. Upper limb power was within normal limits. Sensory examination was normal and plantars were extensor bilaterally. Furthermore, the patient had behavioral disturbances in the form of reduced speech output and apathetic phenotype. There were no meningeal signs. He was catheterized due to urinary retention. The clinical picture of acute onset paraplegia with behavioral disturbances and normal sensory examination led us to suspect a cortical involvement in the form of bilateral ACA infarcts. A second possibility of a spinal thoracic cord infarction or demyelination was also kept in the differentials.

Routine blood investigations did not reveal anything significant. Magnetic resonance imaging (MRI) of the brain showed multiple bilateral infarcts in the ACA territory with magnetic resonance angiogram showing complete occlusion of both the pericallosal arteries (Figs. 1 and 2). MRI of the spine did not show any abnormalities. Cardiac workup was normal and vasculitic markers were negative. A final diagnosis of an ACA infarction with paraplegia was made.

He was managed with low molecular weight heparin, dual antiplatelet agents, and statins. Blood pressure and diabetic control



**Figure 1: (a and b) Diffusion-weighted imaging/apparent diffusion coefficient mapping showing bilateral infarcts in anterior cerebral artery territory**




**Figure 2: Magnetic resonance angiogram showing thrombosed and absent right anterior cerebral artery distal branches (circled)**

were also done. He was also started on intensive exercises and physiotherapy. Antidepressants were started for abulic symptoms. He had good motor power improvement and was able to walk at discharge with support. He is on regular follow-up and treatment.

ACA supplies many areas of the brain including the medial frontal lobes, the corpus callosum, and the basal ganglia [2]. The symptoms of the same depend on the site of occlusion of the vessels. Atherosclerosis is one of the most important causes of ischemic stroke [3]. The mechanisms of atherosclerosis include

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local branch occlusion by ulcerating plaque, artery-to-artery embolism, and vessel thrombosis [4]. Vessel thrombosis is one of the commonest causes of ACA infarction especially in Indian patients. Patients presenting with an acute stroke of the ACA will have different clinical features depending on whether the main vessel or its branches or involved. Clinical symptoms also depend on infarct size. The most common presentation seen in >90% of patients is weakness of the contralateral lower extremity [5]. Recurrent artery of Huebner infarcts are associated with contralateral face and arm weakness. Behavioral disturbances seen include abulia, agitation, motor perseveration, memory impairments, emotional lability, and anosognosia [6]. Delirium and language disorders are frequently seen. Bilateral involvement is relatively rare and presents with acute onset of lower limb weakness and behavioral disturbances including abulia, memory impairment, and agitation. The syndrome is recognized late due to misdiagnosis as spinal cord involvement. The absence of a clear sensory level and the presence of behavioral changes should lead one to suspect a cerebral involvement. Investigations should include prompt imaging using an MRI with an associated angiogram to look at the vessel patency. Diffusion-weighted imaging is crucial to making the diagnosis of these infarcts. Early diagnosis is important for therapeutic options including intravenous thrombolysis which can lead to better outcomes. Routine treatment with dual antiplatelet agents and statins is given in all cases. Physiotherapy, occupational therapy, and also psychosocial support will be needed in most patients.

In all cases of sudden onset paraplegia with bladder involvement, the presence of behavioral changes should lead one to suspect bilateral ACA involvement. A high index of suspicion should be kept in all such cases. Early diagnosis and treatment can lead to better outcomes.

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