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Unilateral basal ganglia infarction presenting as sudden onset daytime sleepiness

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Abstract

Basal ganglia (BG) are involved in motor coordination. BG strokes usually present with problems controlling speech, movements, mood and posture leading to abulia which is a prominent feature. BG stroke presenting with daytime sleepiness is not well reported. We report a 63-year- old Asian woman with hypertension presenting with sudden onset daytime sleepiness due to basal ganglia infarction. This case highlights the importance of considering BG infarction as a differential diagnosis for sudden onset daytime sleepiness.

Keywords: basal ganglia infarction, apathy, lacunar infarcts, excessive daytime sleepiness, stroke

Introduction

Basal ganglia are a group of subcortical nuclei responsible primarily for motor control as well as motor learning, executive functions, behaviour, and emotions. It is the key part of the network of the brain that controls your voluntary movements.(1) BG lesions lead to abulia, which is a syndrome of "hypofunction," with lack of initiative, spontaneity, drive, apathy, slowness of thought (bradyphrenia), blunting of emotional responses and response to external stimuli.(2) The commonest cause of BG strokes is lacunar infarcts due to lenticulostriate artery ischaemia.(3) Damage to the basal ganglia causes problems in controlling speech, movement, and posture similar to the symptoms of parkinsonism. Sudden changes in behaviour and apathy have been reported as signs of basal ganglia stroke.(4,5) However, basal ganglia stroke, which presents as excessive sleepiness is not reported.

Case presentation

A 63-year-old active woman complained of excessive sleepiness for a day. She was otherwise well. She was on losartan 50 mg twice a day and atorvastatin 20 mg at night for the past 3 years. The sleepiness persisted for the next day as well and she accidentally dropped a glass bottle held in her left hand. She cut her left index finger while chopping vegetables, which was unusual for her. Her family noted some subtle changes in her behaviour with unusual lethargy and she was taken for medical advice.

On examination, her GCS was 15/15, and there were no focal neurological signs, including sensory disturbance or incoordination. Her blood pressure was 150/90 mmHg and the pulse was 88 beats per minute. All her basic haematological and biochemical studies, including serum electrolytes, random blood sugars and other investigations, were normal.

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Because of the sudden onset of symptoms, she had a magnetic resonance imaging (MRI) scan of the brain done following 48 hours of the index event. The MRI of the brain showed an acute infarction of the right-side basal ganglia region (figure 1).

She was managed for an acute infarction of the brain with Aspirin 300 mg stat and Clopidogrel 300 mg stat, followed by Aspirin 75mg and Clopidogrel 75 mg at night for 3 weeks. Furthermore, her blood pressure control was optimised by adding hydrochlorothiazide 25 mg in the morning to losartan 50 mg twice daily while the atorvastatin dose was increased to 40 mg a day. Her sleepiness improved over a month and her apathy improved gradually over one year, but she's still not as active as she was before, after two years from the event.

Discussion

This previously active woman with sudden onset sleepiness, apathy and incoordination was diagnosed to have a BG infarction based on MRI findings. She did not have prominent weakness of muscles but had bradykinesia and incoordination confirming basal ganglia involvement. She was treated for a cerebral infarction but was not thrombolysed, as she presented after 6 hours of the onset of symptoms and because she did not have an objective weakness or impairment.

BG are a group of subcortical nuclei primarily responsible for motor control, motor learning, executive functions like paying attention and staying focused, self-monitoring, organising, planning, behaviour, and emotions.(1,6,7) Our patient had an impairment of most of these functions which were difficult to be identified by an external person or the clinician, as they are difficult to assess objectively. However, her family who knew her usual behaviour, had noted some abnormality.

Basal ganglia infarction, presenting with impairment in speech, movement, and posture leading to abulia, akinesia, amnesia, dis-inhibition and hemi-neglect is reported in the literature.(8,9,10) Abulia is the commonest behavioural symptom that is observed in BG infarcts, with a prevalence of 13%.(8,11) Abulia and confusion have been noted in patients with right side anterior lenticulostriate artery infarcts, which were also present in our patient. Prominent motor deficits, neglect, frontal system dysfunctions and visual amnesia have been noted in relation to right side lateral lenticulostriate artery infarcts.(9) Daytime somnolence is not reported as a presenting symptom of BG strokes.

BG receives input from the suprachiasmatic nucleus (SCN), which is the master pacemaker of the circadian rhythm located in the hypothalamus. BG has a reciprocal connection with the SCN, which allows BG to influence the circadian rhythm. In addition, BG receives input from other sleep-regulating areas of the brain like the thalamus and brainstem. All these signals play a role in the regulation of sleep and wakefulness during higher level cognitive processes. Damage to the BG nucleus in an infarct can disrupt these actions.(14) BG are responsible for choosing actions that will lead to a positive consequence and facilitate desired movement while inhibiting unwanted movements that contradict the said action

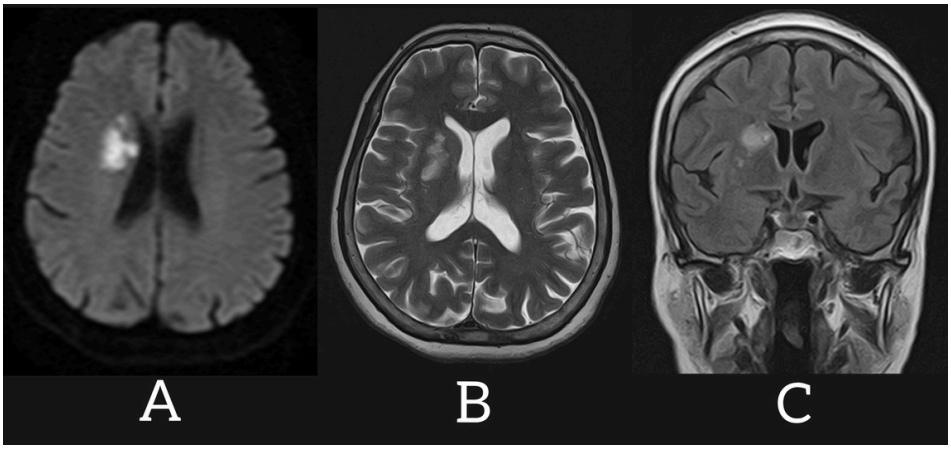

Figure 1 - Non-contract magnetic resonance image of the brain after 48 hours of the index event showing acute infarction of the right-side basal ganglia, **A;** Diffusion weighted image, **B;** Apparent diffusion coefficient image, **C;** T2 Flair imagest x-ray taken on admission(left) and after treatment(right) showed remarkable improvement

simultaneously.(8,11-13) "Rate model of basal ganglia" explains this by modulating firing rates of neurons in the BG by the balance of excitatory and inhibitory inputs received(15), explaining the maintenance of awake status.

Conclusion

This case highlights the importance of considering basal ganglia stroke as a differential diagnosis of acute onset daytime sleepiness. It also highlights the importance of taking a good history from the patient as well as from close family or eyewitnesses.

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