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Lesion of the anterior cingulate cortex presenting as apathy in a patient with treatment-resistant depression

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Abstract

A 63-year-old female with past medical history of treatment-resistant depression and ischemic stroke with residual left leg weakness and wheelchair-bound, presented to our institution with acute onset of right-sided weakness, right facial droop and slurred speech. Neurological exam revealed sensory loss, right facial droop and dysarthria. National Institute of Health Stroke Scale (NIHSS) score was 19. The initial BP was 190/100. CT head was unremarkable. Magnetic Resonance Imaging (MRI) of the brain was negative for acute stroke. However, hyperintensity in the periventricular white matter and centrum semiovale, a lacunar infarct of genu of the corpus callosum especially on the right side and an extensive area of encephalomalacia in the corpus callosum involving the mid-body extending into the right Anterior Cingulate Cortex (ACC) with sclerotic margins were noted. MRI findings were consistent with cerebral Small Vessel Disease (SVD). Patient's symptom resolved after 18 hours. Given her cerebral SVD, patient underwent complete neuropsychological evaluation for her depressive symptoms. Scores evaluation scale were more in favor for apathy than depression.

Keywords

Apathy; small vessel disease; anterior cingulate cortex.

Introduction

Small Vessel Disease (SVD) is caused by a disruption of white matter cortical-sub-cortical pathways especially prefrontal and Anterior Cingulate Cortex (ACC) which are important for executive function and decision making, resulting in neuropsychiatric symptoms of apathy and depression [1]. Apathy is a reduction in goal-directed behavior that manifests as decreased initiative and interest whereas depression

is a negative emotional state that is completely distinguishable and dissociable from apathy [2]. Though depression was commonly found to be associated with SVD; apathy, rather than depression, has been suggested as the major neuropsychiatric symptom in patients with SVD and specifically associated with cognitive decline and executive dysfunction [1,3]. However, the symptoms of apathy are often misdiagnosed as depression as some of the scales used to measure depression include items relating to both apathy and depression [3]. The importance of separating these entities depends on the fact that depression and apathy have different types of management and prognosis.

Case Report

A 63-year-old female with past medical history of treatment-resistant depression and ischemic stroke with residual left leg weakness and wheelchair-bound, presented to the emergency room with acute onset of right-sided weakness, right facial droop, and slurred speech. Neurological examination revealed right arm and leg weakness, sensory loss, right facial droop, mild aphasia, and dysarthria. NIHSS score was 19. The initial BP recorded was 190/100. CT head was unremarkable. MRI of the brain was negative for acute stroke. However, hyperintensity in periventricular white matter and centrum semiovale in FLAIR sequence was noted demonstrating extensive micro vascular disease. A lacunar lesion was seen involving the genu of the corpus callosum, especially on the right side. There was also an extensive area of encephalomalacia in the corpus callosum involving the mid-body extending to the Anterior Cingulate Cortex (ACC) on the right side with sclerotic margins (Figure 1). Patient´s symptoms were completely resolved after 18 hours. She was discharged on the third day in stable condition and resume home medications. Given her treatment-resistant depression and based on the imaging findings of severe SVD on MRI, patients underwent a complete neuropsychological evaluation to diagnose apathy versus depression. Scores on the Apathy evaluation scale were more in favor for apathy than depression.

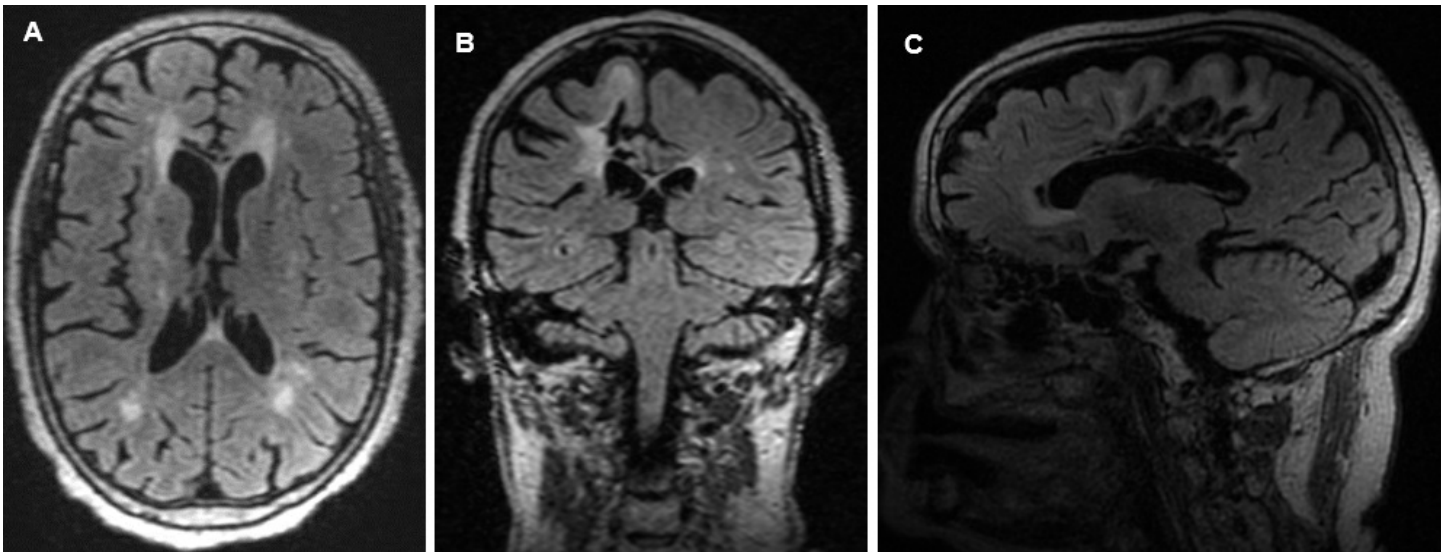

Figure 1: Brain MR, FLAIR sequence, **(A)** axial, **(B)** coronal and **(C)** sagittal planes showing lacunar lesion involving the genu of the corpus callosum with right side predominance and an extensive area of encephalomalacia in the corpus callosum involving the mid-body extending to the anterior cingulate cortex on the right side with sclerotic margins.

Discussion

We described a patient with severe SVD clinically causing apathy which was confused with depression. Patient's MRI revealed extensive damage to the white matter tracts manifested as hyperintensity in periventricular white matter and centrum semiovale in FLAIR sequence. A lacunar lesion involving the genu of the corpus callosum especially on the right side and an extensive area of encephalomalacia in the corpus callosum involving the mid-body extending to the ACC on the right side with sclerotic margins were also demonstrated.

Apathy and depression are the two entities related to a neuronal disruption in subcortical white matter structures of the prefrontal cortex and ACC. However, diagnosing apathy in SVD is a challenge because of considerable overlap with symptoms of depression, resulting in difficult differentiation of the two conditions clinically [1]. Several depression scales include both apathetic and depressive symptoms, and it may be the failure to differentiate these two conditions that have led to an over-diagnosis of depression in SVD and an under-diagnosis of apathy. Hence, multiple studies obtained a high incidence of depression than apathy by 10% and 12% respectively [1,2].

The cingulum is a large white matter tract underlying the ACC which has been linked to both affect regulation and cognitive control, and has been associated with apathy in both structural and functional MRI studies [4,5]. The structural damage corresponding to subcortical white matter, especially the ACC, is associated more with apathy rather than depression [1-3].

A study conducted by Hollock et, al analyzed which regions of white matter microstructure were specifically related to either apathy or depression using diffuse tensor imaging (DTI) sequencing [1]. A reduced median fractional anisotropy, especially in the ACC was associated with apathy ($p=\leq 0.001$), but not with depression ($p=0.09$).

Similarly, a study conducted by Tay J et, al. has proven that apathy, not depression, was associated with a neuronal disruption in the regions of the prefrontal cortex and ACC in patients with SVD [2]. The sub-networks delineated suggest that apathy is driven by damage to white matter networks underlying executive function decision making.

Another study conducted by Lohner et, al, showed that the presence of apathy was more commonly seen in patients with SVD involving anterior sub-cortical structures and was associated with impaired global cognition and executive function [3]. Whereas the presence of depression was not related to global cognition, impaired executive functioning/processing speed or memory/orientation.

Along with SVD, neural disruption in the region of ACC demonstrated in apathy was observed in other clinical entities like stroke, traumatic brain injury, and dementia [6-9]. Occasionally, apathy is masked by symptoms of depression making the diagnosis difficult [6].

Based on the available evidence, it is not appropriate to state that patients with SVD do not suffer from symptoms of depression. Rather, the depression in SVD may be secondary to apathy which is the di-

rect consequence of white matter damage. Also, the association between white matter disruption and depression previously described in SVD may be attributable to apathy; in part because scales used to assess depression in these studies included apathy related items.

Apathy, not depression, is associated with damage to subcortical white matter tracts, especially the prefrontal cortex and ACC and is often overlooked clinically. This highlights the importance of clinically assessing apathy along with depression because it has both immediate and long-term consequences for patients.

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