CASE REPORT

Case Report: Corpus callosal apraxia following acute ischemic stroke [version 1; peer review: 1 not approved]

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Abstract
The corpus callosum is a compact structure that connects the right and left cerebral hemispheres. Here we report the case of a 50 year old woman who presented with features of corpus callosum apraxia, initially mistaken as psychiatric symptom by her relatives. Computed tomography and magnetic resonance of brain confirmed the diagnosis of acute ischemic infarct in the body of the corpus callosum. Isolated stroke involving the corpus callosum is rarely reported in literature and is a diagnostic challenge due to atypical clinical features.

Keywords
Corpus callosum, apraxia, disconnection syndrome, stroke

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Author roles: Narayanan S: Conceptualization, Data Curation, Formal Analysis, Writing – Original Draft Preparation, Writing – Review & Editing; Subramaniam G: Supervision, Visualization, Writing – Original Draft Preparation, Writing – Review & Editing

Competing interests: No competing interests were disclosed.

Grant information: The author(s) declared that no grants were involved in supporting this work.

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How to cite this article: Narayanan S and Subramaniam G. Case Report: Corpus callosal apraxia following acute ischemic stroke [version 1; peer review: 1 not approved] F1000Research 2018, 7:731 (https://doi.org/10.12688/f1000research.15059.1)

Introduction

Corpus callosum contains densely packed white matter tracts essential for coordination and integration of the two sides of brain. Pathology affecting the corpus callosum is very rare. Clinically it manifests with apraxia, cognitive dysfunction, disturbances of memory and may be mistaken for psychosis. Tumours (glioma, lymphoma, meningioma, metastasis), demyelinating diseases, trauma and congenital malformations can involve the corpus callosum. However, due to its rich collateral blood supply, isolated vascular lesions are extremely uncommon. Here we report the case of a 50 year old woman who presented with features of corpus callosum apraxia, initially mistaken as a psychiatric symptom.

Case report

A 50 year old woman presented with a history of abrupt onset of numbness on the left side of her body. She had no history suggestive of raised intracranial tension, cranial nerve dysfunction or motor involvement. The patient was admitted with a provisional diagnosis of sensory stroke considering the rapidity of onset. On the second day of hospitalisation, she found it hard to transfer objects from her left hand to the right hand, and found it hard to execute finer activities with left hand. Her family mistook these manifestations as psychiatric symptoms. The patient had a past medical history of systemic hypertension for 12 years and was on tab. amlodipine 5 mg once daily.

On examination on the second day of admission, the patient was conscious, oriented to time, place and person. She had a body mass index of 22.1 kg/m² and vital signs were stable. Tests for attention, registration, recall were normal. Her immediate and remote memory was intact. Cranial nerves were normal. She had grade 5 power in all limbs. Sensory system examination revealed apraxia of the left hand, which was diagnosed as ideomotor apraxia: the patient was unable to perform pantomime commands with her left hand and also had astereognosis of the left hand. The patient did not have any involuntary movements in the same limb and could perform all activities with her right hand, but was unable to do the same with the left. There were no cerebellar signs nor signs of meningeal irritation. These features were suggestive of callosal disconnection syndrome.

Computed tomography of brain revealed an acute infarct in the body of the corpus callosum (Figure 1). Magnetic resonance imaging (MRI) of the brain showed hyperintense signals in the body of corpus callosum in T2 weighted FLAIR images (Figure 2). Diffusion weighted images showed diffuse restriction confirming the presence of an acute infarct. Electroencephalogram was normal. Further workup was done to look for the cause of thrombosis. Echocardiography was normal. Arterial Doppler of the cerebral vessels did not reveal any abnormality. The patient’s complete blood count, erythrocyte sedimentation rate and peripheral smear were normal. Antinuclear antibody test and retroviral screening were negative.

The patient was treated with antiplatelets (tab. Ecosprin 150 mg once daily and tab. Clopidogrel 75 mg once daily), statins (tab. Atorvastatin 40 mg once daily), and antihypertensives (tab. Amlodipine 5mg once daily). She showed mild clinical improvement at 4 months of follow up (MRI showed resolution of edema and chronic infarct), although apraxia of the left hand persisted.

Discussion

The corpus callosum contains the white matter bundle that connects the interhemispheric neurons and serves to connect the cortical and subcortical structures of the brain. The corpus
The corpus callosum is present only in placental mammals and reflects the transition towards increased degree of myelination seen in higher animals. Anatomically it is divided into the rostrum, genu, body and splenium from anterior to posterior. The corpus callosum receives a blood supply from three arterial networks. The anterior communicating artery (medical callosal and subcallosal branches), anterior cerebral artery (through the pericallosal artery), posterior cerebral artery (through the posterior pericallosal branch); each supplies different parts of the corpus callosum.

Clinical manifestations of callosal infarcts include callosal disconnection syndrome (as in our case), neuropsychiatric symptoms, gait disorders and alien hand syndrome. Callosal disconnection manifests in different ways based on the part of the callosum affected. Lesions in the anterior part of callosum presents as unilateral unresponsiveness to touch (tactile anomia), difficulty in calculation, difficulty in copying drawings, and unilateral agraphia (inability to write with one hand). Posterior lesions cause visual unresponsiveness on one side (visual anomia) and a phenomenon called “alexia without agraphia” (able to write, but unable to read). Lesions in the body of the callosum results in unilateral ideomotor apraxia, difficulty to use objects with one hand, and inability to transfer objects from one hand to the other. This occurs in the left hand in majority of the cases, as the left is the dominant hemisphere in most individuals. MRI is the diagnostic test of choice for the localisation of a callosal infarct. Differential diagnoses for callosal infarcts include Marchiafava Bignami syndrome (callosal demyelination in chronic alcoholism) and multiple sclerosis.

Consent
Written informed consent for publication of their clinical details and clinical images was obtained from the patient.

Data availability
All data underlying the results are available as part of the article and no additional source data are required.

Competing interests
No competing interests were disclosed.

Grant information
The author(s) declared that no grants were involved in supporting this work.

References

Open Peer Review

Current Peer Review Status:  

Version 1

Reviewer Report 14 January 2019

https://doi.org/10.5256/f1000research.16394.r42656

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This paper focuses on a specific clinical sign of disconnection following corpus callosum damage, unilateral apraxia. The authors point out that focal damage to the corpus callosum is rare and may present a diagnostic challenge. I only partly agree, as damage to the corpus callosum is at least occasionally observed following ischemic stroke to one of the pericallosal arteries, though in this case the damage is often not confined to the corpus callosum, but also involves medial frontal cortex and cingulate cortex. I also think that such injury presents only a diagnostic challenge when the examiner does not specifically focus on symptoms of callosal disconnection. The body and genu of the corpus callosum carry fibers connecting pre-motor and parietal cortices of the two cerebral hemispheres which are necessary for the coordination of bilateral actions. When reading this case report I was surprised to notice that the authors observed apraxia of the left hand, but did not examine callosal transfer by asking the patient to execute bilateral actions. Besides alien hand syndrome, corpus callosum damage is known to result in diagnistic dyspraxia. This is a movement disorder of the left arm/hand characterized by movements that are antagonistic or symmetrical to the right hand, as well as the occasional inability to move the left arm (though force is typically preserved). Similarly to alien hand syndrome, this disorder suggests an impairment of voluntary control, and some patients report that their hand feels disobedient or has 'its own personality'. Such impairments appear sufficiently mysterious to be mistaken as psychiatric symptoms. The current case is interesting because of the isolated corpus callosum damage, but one would have wished a thorough examination of disconnection signs other than unilateral apraxia.

Is the background of the case's history and progression described in sufficient detail?
Partly

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?
No

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?
No

Is the case presented with sufficient detail to be useful for other practitioners?  
Partly

**Competing Interests:** No competing interests were disclosed.

**Reviewer Expertise:** Clinical neuropsychology

I have read this submission. I believe that I have an appropriate level of expertise to state that I do not consider it to be of an acceptable scientific standard, for reasons outlined above.

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