SLOW TO RESPOND:
BILATERAL CORPUS CALLOSUM INFARCTION

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INTRODUCTION

Acute corpus callosum infarction is generally rare and it is easily misdiagnosed in the early stage due to its complex clinical manifestation. We herein report a case with a rare presentation of stroke which requires much workup and imaging to reach a conclusive diagnosis. The management of this patient is analysed and discussed in this case study.

CASE REPORT

A 59-year-old lady, previously ADL independent, with underlying diabetes mellitus, hypertension and dyslipidemia presented the emergency department with a chief complaint of an altered mental state for the past 6 days prior to presentation. Her key clinical features included slowness in response (which included slowness in thinking/responding, however with no lapse in judgement or comprehension) and poverty of speech, sometimes refusing to speak at all to her family members - only smiling in response. She also had involuntary movements of bilateral upper limbs, as well as weakness of bilateral lower limbs, progressively becoming unable to ambulate. This included developing urinary incontinence.

She had no signs or symptoms of an infection, and vision was intact. Her symptoms were progressive and debilitating. Plain CT brain of this patient revealed hypodensity over the corpus callosum involving the genu and splenium (Figure 1). Because of the non-specific symptoms and non-classical signs, she was initially thought to have some sort of space-occupying lesion e.g. CNS lymphoma.

However, MRI revealed she had diffuse atherosclerotic narrowing of the intracranial arteries with non flow detected within both anterior communicating arteries (ACAs) resuting in acute corpus callosum (genu and body) infarction (Figure 2, Figure 3). She was subsequently treated for bilateral ACA territory infarction, and was started on double antiplatelet therapy. She was given physiotherapy and stroke rehabilitation, and was discharged home after 5 days in hospital.

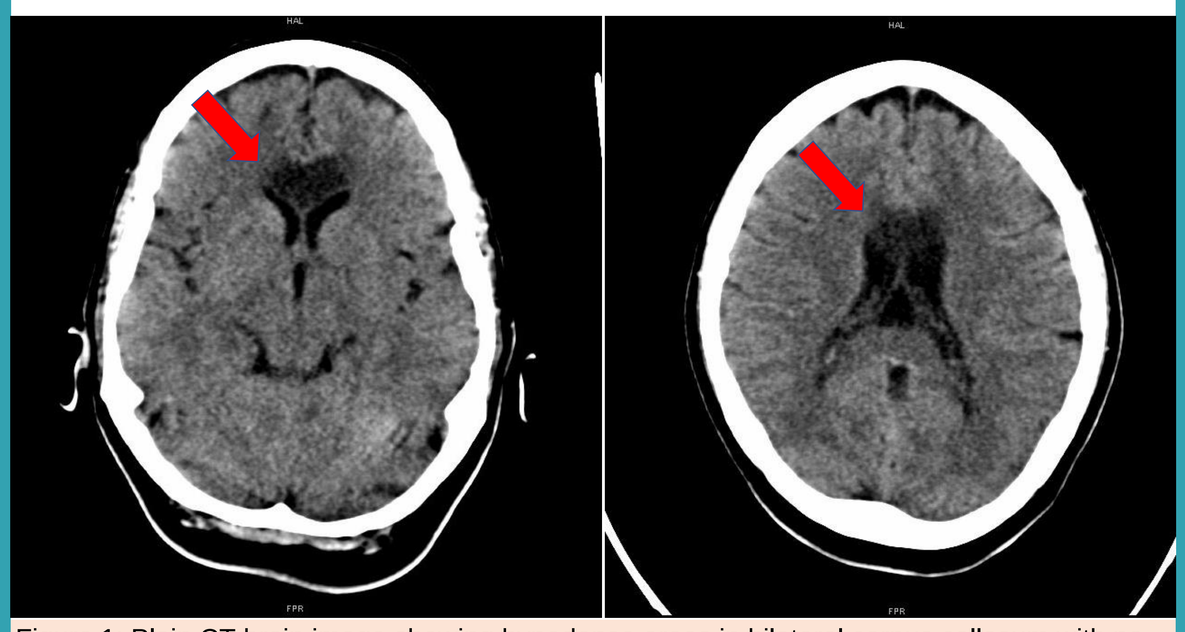

Figure 1: Plain CT brain image showing hypodense areas in bilateral corpus callosum with no mass effect.

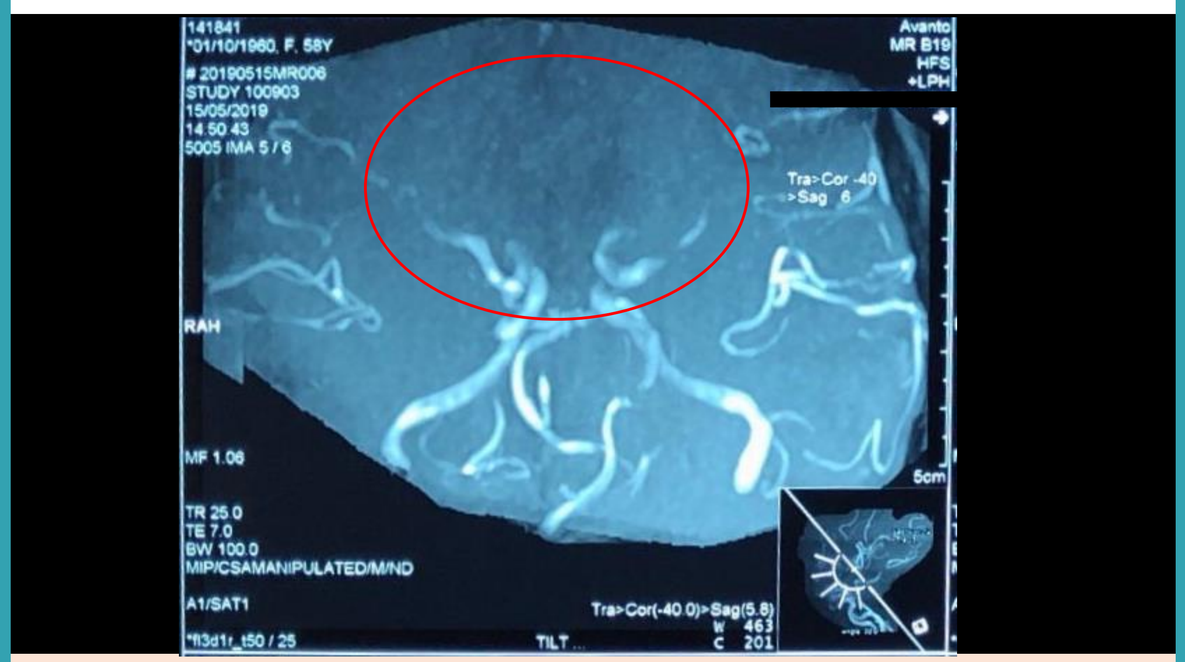

Figure 2: MRA showing no flow in both anterior communicating arteries (ACAs). No occlusion of the MCAs and posterior circulation.

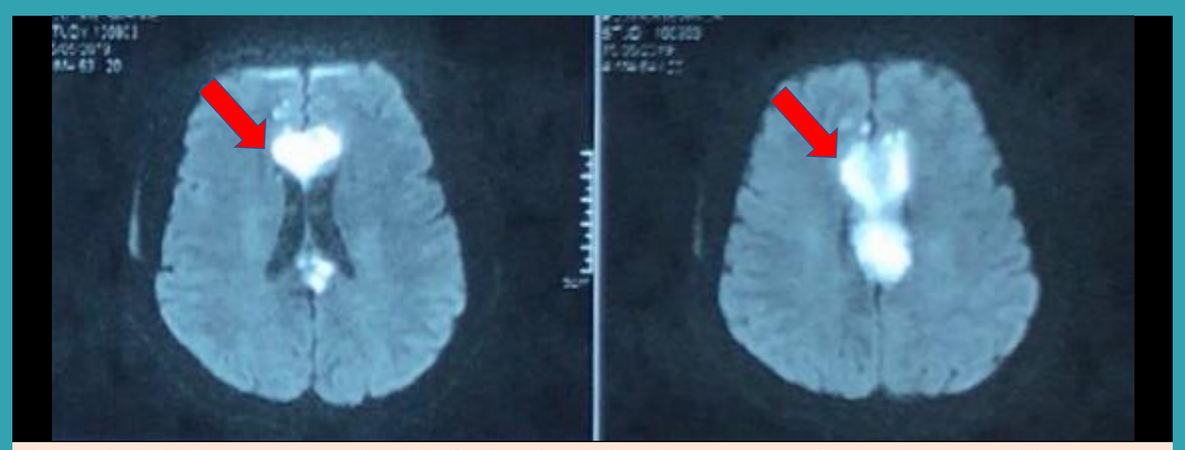

Figure 3: DWI images - restricted diffusion involving the corpus callosum (genu and body, sparing the splenium).

DISCUSSION

The corpus callosum is the largest subcortical commisural fibre, and is vital in cerebral functions. It usually has a rich blood supply from bilateral anterior and posterior cerebral circulation. Due to this rich blood supply, the infarction of the corpus callosum (especially bilateral) is uncommon (as low as 2.9%) and have not been well characterized in recent literature.[1] The appearance of bilateral corpus callosum infarctions can be confounding for the managing physician, and can suggest a more malignant aetiology. In addition, the patients may present with atypical clinical and radiological findings, which was seen in our patient.

Because infarctions of the corpus callosum may present with non-localizing neurologic signs, and radiologic features that may suggest a neoplasm e.g. mass-like enhancement or extension across the midline, some clinicians may order brain biopsies to reach a definitive diagnosis.[2] This is because neoplastic lesions are relatively more common than acute stroke (53% tumour vs. 33% ischaemic origin)[3]. However, the advances of neuroimaging have done away with invasive procedures such as biopsies to reach a more definitive diagnosis.

We now have access to radiological modalities such as diffusion-weighted imaging (DWI), perfusion imaging, or magnetic resonance angiography (MRA) to enable us to better delineate these lesions.

In this case, the cerebral magnetic resonance imaging (MRI) revealed that the infarction was confined to the genu and body of the corpus callosum (this region is supplied by anterior pericallosal artery, a branch of the anterior cerebral artery). The splenium of the corpus callosum was preserved, which explains why the patient did not have classic callosal disconnection syndrome (this region is supplied by the posterior pericallosal artery, a branch of the posterior cerebral artery).

CONCLUSION

By understanding the varied clinical and radiologic features of corpus callosum infarction, clinicians can avoid unnecessary invasive procedures like biopsy by utilising more advanced imaging such as diffusion-weighted imaging and MR angiography, now available in our healthcare setting.

REFERENCES

1. Zhiyong Zhang, Xiufeng Meng, Wei Liu, Zunjing Liu. “Clinical Features, Etiology, and 6-Month Prognosis of Isolated Corpus Callosum Infarction,” BioMed Research International, vol. 2019, Article ID 9458039

2. Kasow DL, Destian S, Braun C, et al. Corpus callosum infarcts with atypical clinical and radiologic presentations. AJNR Am J Neuroradiol. 2000;21:1876-1880.

3. Wilson CA, Mullen MT, Jackson BP, Ishida K, Messé R. Etiology of Corpus Callosum Lesions with Restricted Diffusion. Clin Neuroradiol 2017 Mar;27(1):31-37. doi: 10.1007/s00062-015-0409-8.