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Diffuse corpus callosum infarction — Rare vascular entity with differing etiology

Rohan Mahale *, Anish Mehta, Kiran Buddaraju, Aju Abraham John, Mahendra Javali, Rangasetty Srinivasa

Department of Neurology, MS Ramaiah Medical College & Hospital, Bangalore 560054, Karnataka, India

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ABSTRACT

Introduction: Infarctions of the corpus callosum are rare vascular events. It is relatively immune to vascular insult because of its rich vascular supply from anterior and posterior circulations of brain.
Objective: Report of 3 patients with largely diffuse acute corpus callosum infarction.
Methods: 3 patients with largely diffuse acute corpus callosum infarction were studied and each of these 3 patients had 3 different aetiologies.
Results: The 3 different aetiologies of largely diffuse acute corpus callosum infarction were cardioembolism, tuberculous arteritis and takayasu arteritis.
Conclusion: Diffuse corpus callosum infarcts are rare events. This case series narrates the three different aetiologies of diffuse acute corpus callosum infarction which is a rare vascular event.

1. Introduction

The corpus callosum (CC) is the largest white matter tract interconnecting homologous association areas of both hemispheres with approximately 180 million callosal fibers traversing through it [1]. It has abundant blood supply from both the anterior and posterior cerebral circulation. The ACA–PCA anastomoses and the perpendicular orientation of the callosal branches to the parent artery reduce vascular insults to the CC [2]. Diffuse CC infarcts are rare events. Hereby, we describe three patients with acute diffuse CC infarction and the etiology was different in each of these 3 patients.

2. Case report

2.1. Patient 1

A 45-year-old right-handed Asian man presented with acute onset right lower limb weakness of 2 days duration. It was sudden in onset. There was no speech disturbance or weakness in upper limbs. The next day after admission, he developed left lower limb weakness with urinary incontinence. He became apathetic with decreased speech output. He was diabetic and hypertensive on medications. On examination, he was conscious but withdrawn. Comprehension to commands was present but speech output was reduced. Motor examination showed hypotonia of both lower limbs with weakness of grade 2/5. Sensation was preserved with mute plantar response. Brain magnetic resonance imaging (MRI) revealed diffusion restriction in the genu, body and splenium of the CC. Magnetic resonance angiogram (MRA) showed an azygous anterior cerebral artery (ACA), with non-visualization of right ACA (Fig. 1). Two-dimensional echocardiogram (2D-echo) showed global hypokinesia with ejection fraction of 25%. There was intracardiac thrombus. A diagnosis of acute diffuse CC infarct secondary to cardioembolism was made. He was treated with heparin followed by oral anti-coagulants. At the 3-month follow-up, his speech output had improved and was ambulatory with support.

2.2. Patient 2

A 44-year-old right-handed Asian man presented with gait disturbances in the form of short stepped, apractic gait with fear of falling of 4 days duration. This was associated with vague sensation in the head. On second day of admission, there was worsening of symptoms as he became apathetic, not indicating needs with decreased speech output and lower limbs movement with incontinence. He was a pure vegetarian, smoker and hypertensive on medications. His vitals were stable but absent radial pulse in the right upper limb. Neurologically, he was conscious, looking around but was mute. He had decreased movement of both lower limbs and right upper limb with hypotonia and hyporeflexia. Plantar responses were mute. Brain MRI showed diffusion restriction in the genu and entire body of the CC. MRA showed non-visualization of both ICA with thinning of right vertebral artery (Fig. 2). Digital subtraction angiography (DSA) showed occlusion of right subclavian artery, decreased flow in right vertebral artery, non-visualization of both ICA with cut-off at the proximal common carotid arteries. 2D-echo showed concentric left ventricular hypertrophy. Complete

* Corresponding author.
E-mail address: rohanmahale83@gmail.com (R. Mahale).

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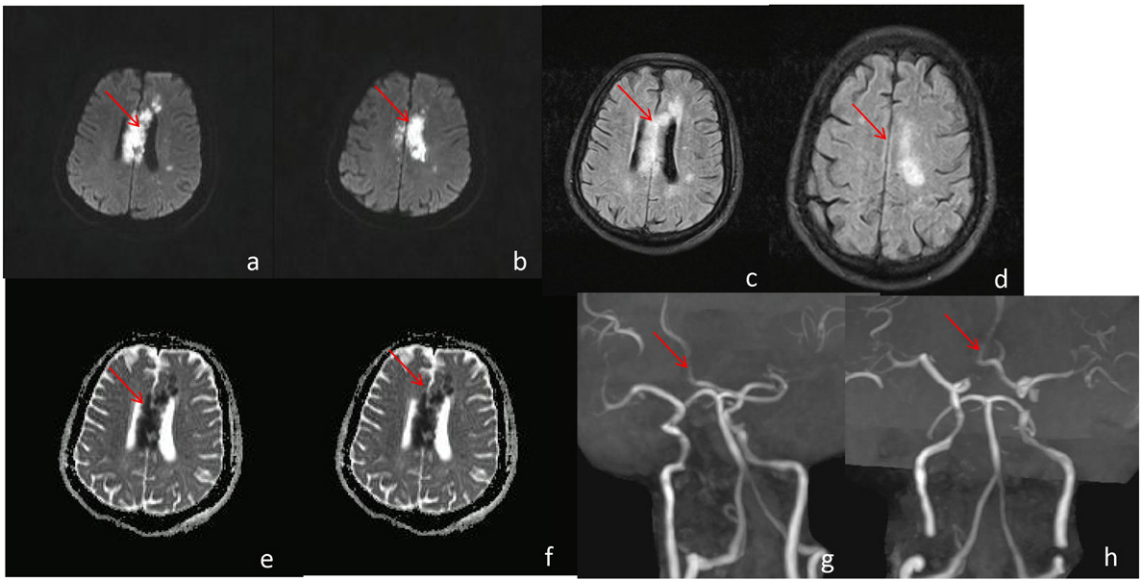

Fig. 1. Brain MRI diffusion-weighted imaging (DWI) (a) & (b) showing diffusion restriction in genu, body and splenium of CC (red arrows); fluid attenuated inversion recovery (FLAIR) axial view (c) & (d) showing hyperintensity in genu, body and splenium of CC (red arrows); apparent diffusion co-efficient (ADC) (e) & (f) showing dark signal in genu, body and splenium of CC (red arrows); brain MRA (g) and (h) showing an azygous anterior cerebral artery (ACA), with non-visualization of right ACA (red arrows).

hemogram showed raised erythrocyte sedimentation rate (62 mm h). Renal, hepatic and thyroid functions were normal. Serological tests for human immunodeficiency virus, hepatitis-B surface antigen and syphilis were negative. Anti-nuclear antibody (ANA) and ANA profile including anti-nuclear cytoplasmic antibodies were negative. A diagnosis of acute diffuse CC infarct due to takayasu arteritis (TA) was made. He was treated with high dose methyl prednisolone, anti-platelets and

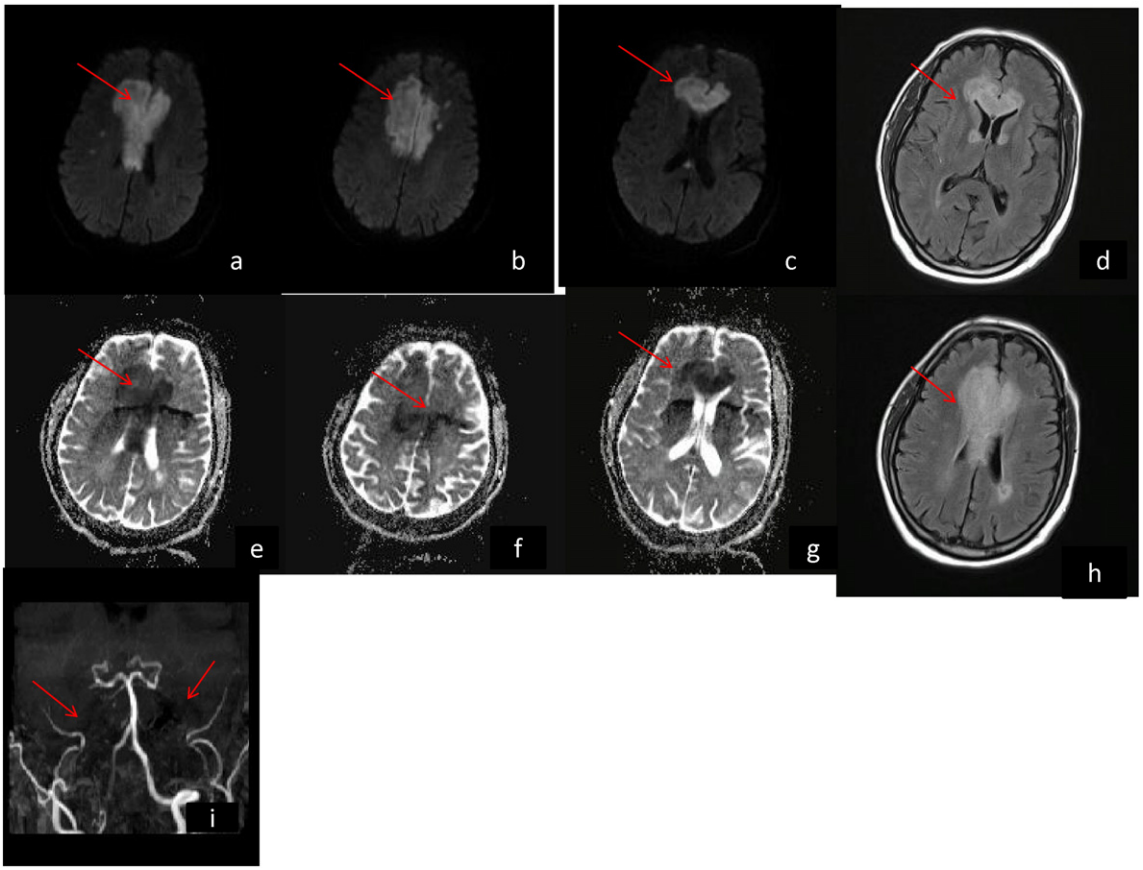

Fig. 2. Brain MRI DWI (a), (b) & (c) showing diffusion restriction in genu, body and splenium of CC (red arrows); FLAIR axial view (d) & (h) showing hyperintensity in genu, body and splenium of CC (red arrows); ADC (e), (f) & (g) showing dark signal in genu, body and splenium of CC (red arrows); brain MRA showing non-visualization of both ICA with thinning of right vertebral artery (red arrow).