SAR Journal of Psychiatry and Neuroscience

Abbreviated Key Title: *SAR J Psychiatry Neurosci*
Home page: https://sarpublication.com/journal/sarjpn/home
DOI: https://doi.org/10.36346/sarjpn.2026.v07i04.002

OPEN ACCESS

ISSN 2707-7764 (P)
ISSN 2709-6939 (O)

Letter to Editor

A Weird Presentation of Large Vessel Vasculitis and a Blinded Approach

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Article History: | Received: 16.05.2026 | Accepted: 04.07.2026 | Published: 06.07.2026 |

DEAR EDITOR

A 40-year-old woman with no known comorbidities experienced an acute onset of weakness in her left upper and lower limbs, along with facial weakness and mild speech impairment. At the emergency room, her vitals were stable, and the National Institutes of Health Stroke Scale was seven. The CT scan was unremarkable (Alberta Stroke Program Early CT Score: 10), so she was thrombolysed with tenecteplase at four hours from the onset of symptoms shortly after arrival. She had mild improvement of symptoms, and an immediate MRI of the brain post-thrombolysis revealed no stroke but significant stenosis of the cervical, petrous, supraclinoid internal carotid, and proximal middle cerebral arteries bilaterally. She gradually developed a confusional state and involuntary movements of limbs, so antiepileptic drugs were started (levetiracetam and sodium valproate), and she required artificial ventilatory support. An electroencephalogram confirmed ongoing status epilepticus and was further managed with fosphenytoin along with midazolam.

There was no history of fever, headache, drug, or toxin exposure. In view of diffuse intracranial and extracranial stenosis with non-convulsive status without background comorbidities, vasculitis was suspected. Methylprednisolone and intravenous immunoglobulin were both started on the day of admission; aspirin and a statin were added on the next day. CT angiography revealed similar stenosis in bilateral carotids with circumferential and symmetric thickening. However, the cerebrospinal fluid (CSF) study was unremarkable (cell count of three, all lymphocytes; protein of 30 mg/dl; glucose: 115 mg/dl; the comprehensive infection markers, including for tuberculosis, were normal in CSF). The routine blood tests and metabolic parameters were within normal limits. Common chronic infections, including HIV, hepatitis B and C, and syphilis, were ruled out. Her vasculitis and systemic autoimmune markers (ANA, antiphospholipid antibodies, anti-dsDNA, ANCA, cryoglobulin, and complement level) were normal. An echocardiogram and cardiac rhythm monitoring were unremarkable. She gradually improved to a GCS of E3M5VT after 48 hours but started having blood pressure surges and persistent tachycardia (autonomic dysfunction) without any evidence of infection, which settled gradually with symptomatic treatment. The family of the patient denied digital subtraction angiography or brain biopsy for a definitive diagnosis. A repeat MRI of the brain with vessel wall imaging on the 3rd day revealed vessel wall enhancements of bilateral petrous and cavernous internal carotid arteries with mild improvement in vessel stenosis. There were patchy infarcts in the bilateral corona radiata, centrum semiovale, and temporal lobes with mild hemorrhagic conversion strongly supporting the vasculitis etiology. (Figure 1)

Rituximab was started and continued as per protocol as the maintenance therapy. She was later tracheostomized and weaned off the ventilator on the seventh day. On follow-up after two weeks, she was in a minimally conscious state, and repeat imaging (Figure 2) revealed a considerable decrement of the lesions without any new vascular events.

Our patient had no background comorbidities and presented with an acute stroke. The delayed DWI restriction, as in our case is seen in acute strokes

secondary to vasculitis [1]. This phenomenon is because of the evolving nature of vascular inflammation and cumulative ischemic damage over time. The multi-territorial involvement with large vessel attenuation and concentric vessel wall enhancement supported our diagnosis. As there was no evidence of systemic involvement, we were dealing with a case of large vessel CNS vasculopathy. The diagnostic criteria for common large vessel vasculitis, giant cell arteritis, and Takayasu arteritis did not match in our case. The common noninflammatory diagnosis of vasculopathy, like atherosclerosis, moyamoya disease, fibromuscular dysplasia, or reversible cerebral vasoconstriction syndrome, were also inappropriate for her. Although we kept large vessel vasculitis as the likely possibility because of the close clinical features of acute stroke, encephalopathy, seizures, and radiological findings, in view of the extracranial large vessel involvement, including petrous ICA, we could not call this a large vessel-related primary angitis of CNS (LV-PACNS). Wang Y *et al*., found LV-PACNS had a higher incidence of cerebrovascular events (88.9%) compared to small vessel-PACNS, and almost all cases of LV-PACNS patients had cerebrovascular stenosis and circumferential vascular wall enhancements [2].

Thrombolysis helps treat acute stroke secondary to vasculitis by breaking down fibrin-rich clots within occluded cerebral vessels but carries with it a significant chance of hemorrhagic complications and edema by reperfusing inflamed blood vessel. We did only a CT scan and thrombolysed our patient and later did vascular imaging. We were lucky our patient did not have a symptomatic hemorrhage.

However, a patient especially young, without comorbidities, should probably undergo multiphasic CT angiography before proceeding with thrombolysis. This is extra five-minute process has the following advantages: 1. CTA source images are more sensitive than NCCT for detecting early ischemic changes. 2. We can determine the site of the clot, calculate the clot burden score, and understand how much the process of thrombolysis will be beneficial. 3. We will be able to rule out uncommon cases of CNS vasculitis, aortic dissection, and large aneurysms at critical sites. 4. The collateral scoring will help us understand the possibility of successful acute stroke intervention and the chance of hemorrhagic transformation.

In the context of CNS vasculitis, thrombolysis-related seizures are mostly caused by the iatrogenic reperfusion of severely injured, inflammatory blood vessels, which can result in severe edema or hemorrhagic transformation. We probably had this complication as the patient landed in status epilepticus immediately after the thrombolysis. For thrombolysis in CNS vasculitis, we need to balance carefully the risk of bleeding. The laboratory markers that may prophesy increased bleeding possibility, like high ESR, CRP (increased inflammatory state), von Willebrand factor, and fibrinogen, may be considered before the procedure.

Endovascular therapy is a "rescue" treatment for large-vessel central nervous system (CNS) vasculitis. However, there remains the risk of procedural complications, including vessel rupture, hemorrhage, and re-occlusion [3]. As our patient was responding to medical treatment we did not proceed with the same.

CNS vasculitis commonly presents with a subacute to chronic presentation of headache, cognitive impairment, psychiatric manifestations, encephalopathy, and later stroke. However, a first-time presentation with a devastating stroke, like in our case, is possibly the rarest, and the earliest possible introduction of immunomodulatory therapy saved her.

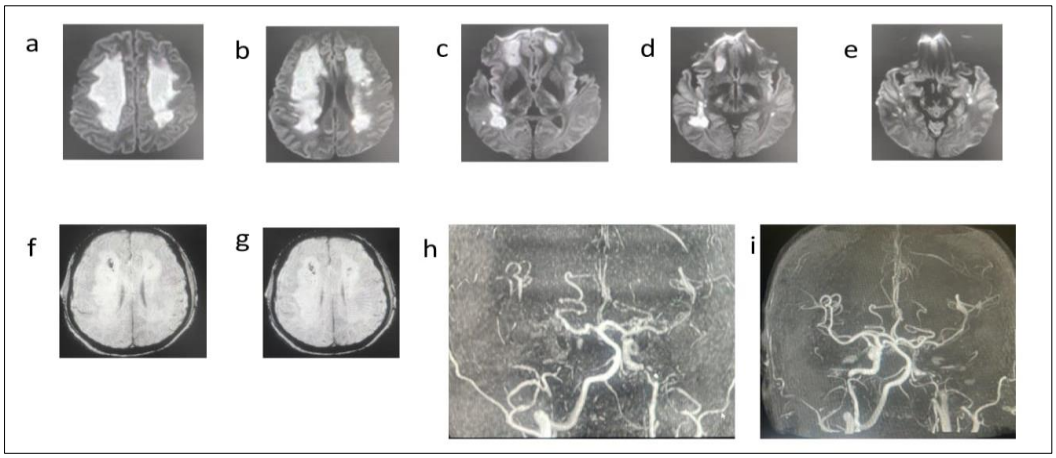

Figure 1: a,b,c,d,e showing DWI images: patchy infarcts in the bilateral corona radiata, centrum semiovale, and temporal lobes with mild hemorrhagic conversion notable in SWI (f and g). MRA revealing stenosis of the cervical, petrous, supraclinoid internal carotid, and proximal middle cerebral arteries bilaterally (h) and improvement of vessel stenosis post treatment (i)

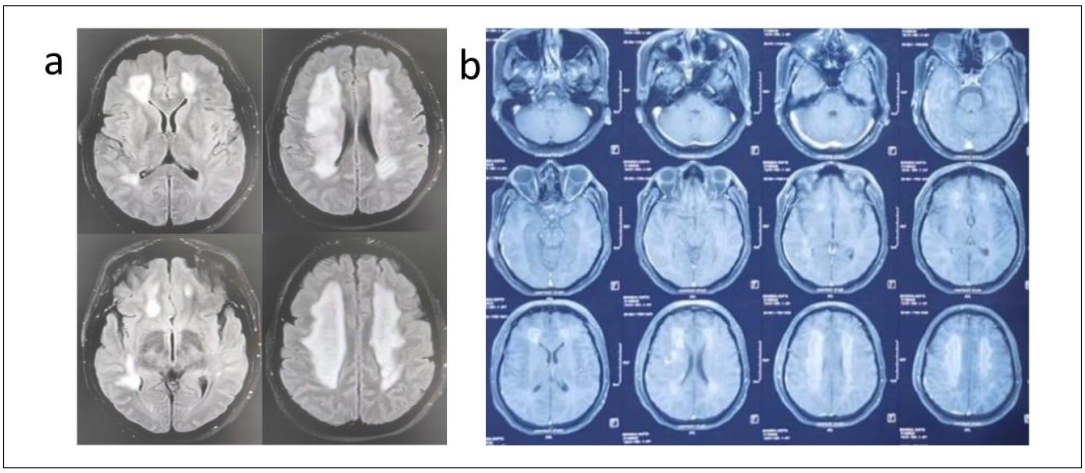

Figure 2: MRI T2FLAIR image (a) showing patchy infarcts in bilateral hemispheres with progressive vasogenic edema surrounding these regions and decrement of the lesions two weeks post-treatment (b)

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