



Received: 26-01-2023  
Accepted: 03-02-2023

ISSN: 2583-049X

Letter to the Editor

## **Diagnosing cerebral vasculitis requires demonstration of inflammation of the vessel wall on biopsy or appropriate imaging modalities**

<sup>1</sup>Josef Finsterer, <sup>2</sup>Sounira Mehri

<sup>1</sup>Neurology & Neurophysiology Center, Vienna, Austria

<sup>2</sup>Biochemistry Laboratory, LR12ES05 "Nutrition-Functional Foods and Vascular Health", Faculty of Medicine, Monastir, Tunisia

Corresponding Author: **Josef Finsterer**

We read with interest the article by Rodriguez-Pla *et al.* on an 82-years-old female with visual impairment with bilateral edema and hemorrhages of the optic discs being attributed to Varicella Zoster virus (VZV) infection complicated by cerebral vasculitis with secondary optic neuropathy<sup>[1]</sup>. The patient benefited from intravenous acyclovir. The study is excellent but has limitations that raise concerns and should be discussed.

The main limitation of the study is that neither a digital subtraction angiography (DSA) nor a computed tomography angiography (CTA) or magnetic resonance vessel wall imaging with black blood sequences had been done to confirm cerebral vasculitis. These investigations are more sensitive to the evidence of cerebral vasculitis than the MRA. We should know why no biopsy of cerebral arteries was carried out.

Another limitation is that venous sinus thrombosis (VST) was not appropriately ruled out. Bilateral disc edema with peripapillary bleeding requires exclusion of this differential diagnosis<sup>[2]</sup>. There was no magnetic resonance venography (MRV) with contrast medium and no determination of the D-dimer.

We disagree with the diagnosis VZV encephalitis as mentioned in the abstract<sup>[1]</sup>. The patient did not present with symptoms or signs of encephalitis, there was no pleocytosis and magnetic resonance imaging (MRI) with contrast medium did not show any cerebral hyperintensity<sup>[1]</sup>.

We disagree with the diagnosis aortic root dilation<sup>[1]</sup>. A diameter of 40 mm in an 82-years-old female is within reference limits.

There is a discrepancy between the leg edema on clinical exam and the only moderately reduced glomerular filtration rate<sup>[1]</sup>. Is it conceivable that leg edema were due to heart failure? Chest X-ray revealed mild cardiomegaly. We should know whether the patient complained about exertional dyspnoea and whether the pro-brain natriuretic peptide (pro-BNP) was elevated or normal. It is not understandable why PCR for varicella zoster virus (VZV) was positive in the cerebrospinal fluid (CSF) and that encephalitis was diagnosed although there was no headache, cognitive impairment, impaired consciousness, or pleocytosis. This discrepancy should be solved.

It is also unimaginable that the basilar artery stenosis resolved within two months<sup>[1]</sup>. We should know how the authors ruled out vasospasm of the basilar artery. Basilar artery stenosis due to vasculitis of the basilar artery that resolves upon acyclovir has not been reported<sup>[3]</sup>. Furthermore, basilar artery stenosis should be confirmed by DSA or CTA as both have the higher diagnostic yield compared to MRA.

There is a discrepancy between the statement that "the patient had a respiratory infection with cough for past seven days" prior to admission and the statement in the next sentence "that she had no cough or shortness of breath"<sup>[1]</sup>. This discrepancy should be solved.

Several data are missing. The virus panel in the serum and CSF was not provided. Results of tests for tuberculosis (e.g., quantiferon test) were not reported. No reference limits were provided. The current medications were not reported. An acute SARS-CoV-2 infection was not ruled out. The anti-SARS-CoV-2 vaccination status was not mentioned. SARS-CoV-2 vaccination can be complicated by non-arteritic, anterior, ischemic optic neuropathy (AION)<sup>[4]</sup>. Overall, the interesting study has limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Diagnosing cerebral vasculitis requires demonstration of inflammation of the vessel wall on biopsy or appropriate imaging modalities.

**Acknowledgements**

**Funding:** No funding was received.

**Data access statement:** All data are available from the corresponding author.

**Ethics statement:** Not applicable.

**Author contribution:** JF: design, literature search, discussion, first draft, critical comments, final approval.

**Disclosures:** The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

**Data access statement:** Not applicable.

**Compliance with Ethics Guidelines:** This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

**Keywords:** Vasculitis, Disc Edema, Varicella Zoster Virus, Acyclovir, Retinal Bleeding

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