LETTER TO THE EDITOR

Status Epilepticus is Not Uncommon as the First Presentation of PRES the Diagnosis of Which Requires the Exclusion of All Differential Diagnoses

Josef Finsterer

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Dear Editor,

We read with interest the article by Prasad et al. on a retrospective study of 40 patients with posterior reversible encephalopathy syndrome (PRES) regarding the outcome of status epilepticus at the 3 and 6 months follow-up, conducted by telephone interview or on-site[1] Status epilepticus was found to be the initial presentation of PRES in eight patients.[1] It was concluded that the diagnosis of PRES with status epilepticus as the presenting manifestation is important since timely treatment of status epilepticus and its underlying cause can reduce the mortality and morbidity of these patients.[1] The study is compelling but has limitations that should be discussed.

The main limitation of the study is its retrospective design.[1] Disadvantages of the retrospective design are that missing data can no longer be supplemented, that patients can no longer be examined again, that open questions can only be answered on the basis of available data, that no new data can be generated, and that the correctness of the data and diagnosis can no longer be controlled.

A second limitation is that the number of patients with PRES and status epilepticus is too small to draw conclusions such as those presented.[1] To answer the question of whether early treatment leads to a more favorable outcome, a well-powered study comparing early versus late treatment needs to be conducted. Incidentally, status epilepticus is an emergency that should be treated early anyway, as no time should be lost to stop the seizure activity.

As the design was retrospective, it is surprising that all patients received a follow-up magnetic resonance imaging (MRI) 6 months after the diagnosis of PRES, which showed complete resolution of the initial cerebral abnormalities.[1] The availability of an MRI 6 months after the first event was adventitious or due to an internal protocol that required all patients with PRES to undergo a second imaging examination at 6 months? If the latter was the case, we need to know why the 6-month period was chosen. The patients included were neurologically examined after 3 and 6 months either on-site or telephone.[1] Did all 40 patients really come to the hospital for the MRI follow-up examination or was the MRI performed outside the hospital?

There is a discrepancy between the aims of the study ("to study the clinical spectrum and outcome of patients with status epilepticus as presenting feature of PRES"), and the results, which show 31 patients with seizures as the presenting symptom, and only 8 patients with status epilepticus as the presenting symptom. Why were the 32 patients without status epilepticus not excluded from further analysis? Status epilepticus as the manifestation of PRES is not a new finding. Status epilepticus has previously been described as the first manifestation of PRES.[2,3]

There are several differential diagnoses for PRES. These include ADEM, AHLE, AHNE, ANE, NMO-SD, MS, postictal state, infectious (e.g., herpes) and immune encephalitis, venous sinus thrombosis (VST), basilar artery thrombosis, posterior cerebral artery stroke, toxic or endocrine encephalopathy, and primary cerebral vasculitis.[4] We should know whether all of these differential diagnoses have been considered and adequately ruled out. Specifically, how did the authors delineate a postictal state and PRES in the 31 patients with seizures and the 8 patients with status epilepticus respectively? How was cerebral vasculitis excluded in the patient with lupus erythematosus? Was cerebral angiography available?

There is an inconsistency regarding the number of patients included in the abstract. There were 33 patients with eclampsia or preeclampsia, 3 patients with hypertensive encephalopathy, and 1 patient each with lupus, acute intermittent porphyria, and acute renal failure.[1] This accounts for 39 patients but included were 40.[1] What was the cause of PRES in the remaining patient? According to Table 1, the 40th patient had ADEM.[1] How was ADEM differentiated from PRES in this particular patient? What is the pathophysiological explanation that ADEM could trigger PRES?

Neurological deficits were observed in four patients. We should know in detail what neurological deficits were identified. We should also know how many were followed-up by telephone and how many in person.

In summary, the interesting study has limitations that put the results and their interpretation into perspective. Addressing these issues would strengthen the conclusions and could improve the status of the study.

Neurology and Neurophysiology Center, Vienna, Austria

Corresponding Author: Josef Finsterer, Neurology and Neurophysiology Center, Vienna, Austria, Phone: +4315861075, e-mail: fifigs1@yahoo.de

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Orcid

Josef Finsterer https://orcid.org/0000-0003-2839-7305

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