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Original Article

Temporal Lobe Epilepsy Caused by Dilatation of Deep Middle Cerebral Vein: Case Report

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Abstract:
Seizures and epilepsy are well-recognized manifestations of cerebrovascular malformations; however, epilepsy associated with venous dilation is uncommon. We report a patient who presented with temporal lobe seizures characterized by automatisms and no other neurological deficits. Magnetic resonance imaging (MRI) of the brain revealed marked dilation of the left deep middle cerebral vein. We suggest that this venous dilation may have contributed to the patient's temporal lobe epilepsy.
Keywords: epilepsy, cerebral vein dilatation, neurology, case report

1 Introduction

Epilepsy is one of the most common clinical manifestations of intracranial vascular malformations (Giakoumettis et al., 2017). Approximately 24–40% of patients with cerebral arteriovenous malformations (AVMs) experience seizures, particularly those with superficial venous drainage (Garcin et al., 2012). AVMs located in the temporal lobe are more frequently associated with epilepsy (Galletti et al., 2014). An MRI study of 10 patients with AVM-associated epilepsy demonstrated a strong association between venous congestion—secondary to impaired cerebrovascular reserve—and seizure occurrence (Fierstra et al., 2011).

Here, we report a case of temporal lobe epilepsy associated with dilatation of the deep middle cerebral vein, which may have contributed to seizure generation via a venous congestion-related mechanism.

Case Report

A 63-year-old man presented to the neurology department with a one-year history of recurrent episodes of unconsciousness, occurring five to six times per month. Each episode lasted approximately 3–4 minutes. The episodes were characterized by sudden cessation of ongoing activity, immobility, and inability to speak, sometimes accompanied by monotonic speech. They were observed during rest, walking, and conversation. Upon recovery, the patient exhibited retrograde amnesia. There was no history of substance abuse or family history of epilepsy. Neurological examination revealed no abnormal signs.

Magnetic resonance imaging (MRI) of the brain revealed dilation of the left deep middle cerebral vein (Fig. 1).

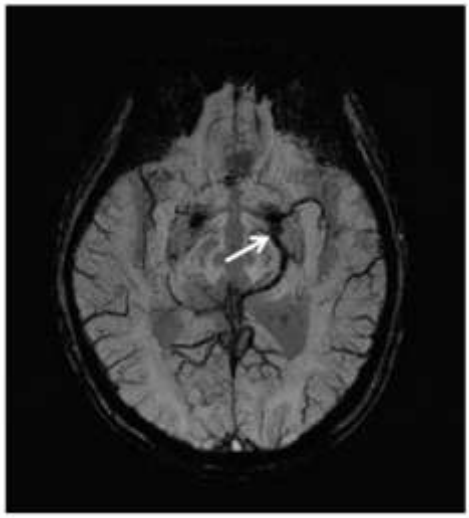

Fig.1 Susceptibility-weighted imaging showed dilatation of left deep middle cerebral vein.

Interictal 24-hour electroencephalogram (EEG) monitoring demonstrated sharp-and-slow-wave complexes in the left frontal and anterior temporal regions during sleep (Fig. 2).

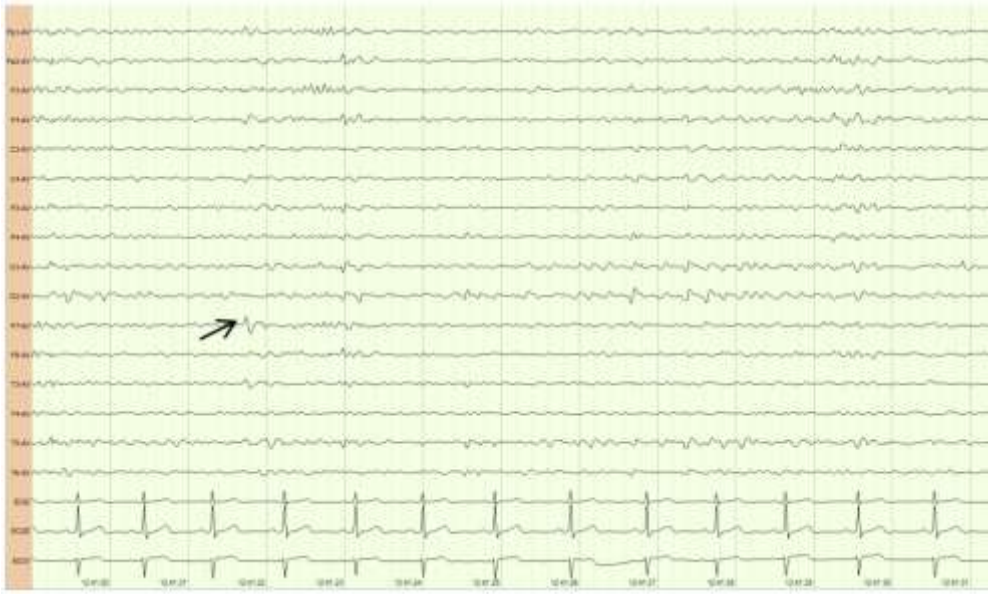

Fig.2 EEG shows sharp and slow wave complexes emission in left frontal and anterior temporal regions during sleep.

Serum lipid testing showed a mild increase in triglycerides (2.24 mmol/L; normal range: < 1.70 mmol/L) and total cholesterol (6.15 mmol/L; normal range: < 5.18 mmol/L). Other laboratory tests—including blood cell counts, electrolytes, renal and liver function, thyroid function, glycated hemoglobin, D-dimer, and antibodies to human immunodeficiency virus and syphilis—were within normal limits. The electrocardiogram was also normal.

A diagnosis of focal epilepsy characterized by automatisms was made. The patient's seizures were effectively controlled with oral oxcarbazepine at a dose of 300 mg twice per day.

Discussion

In this report, the absence of other known risk factors for epilepsy raises the possibility that dilation of the left deep middle cerebral vein contributed to the development of temporal lobe epilepsy. The anatomical location of the dilated vein in the left temporal region is consistent with the interictal EEG findings of sharp-and-slow-wave complexes in the left frontal and anterior temporal regions.

Dilatation of intracranial veins can impair local

circulation and cause cerebral ischemia and hypoxia, which subsequently increases neuronal excitability by enhancing the release of the excitatory neurotransmitter glutamate from presynaptic neurons and reducing the amount of the inhibitory neurotransmitter (γ-aminobutyric acid, GABA), and finally contributes to epileptogenesis. Moreover, oxidative stress induced by hypoxia can lead to lipid peroxidation, protein and DNA damage, and activation of inflammatory responses, which in turn exacerbate oxidative stress (Terrone et al., 2020). This vicious cycle is considered a key mechanism of epileptogenesis. Additionally, disruption of the blood-brain barrier caused by venous dilatation plays a crucial role in astrocyte activation via the albumin-mediated transforming growth factor β (TGFβ) pathway (Cacheaux et al., 2009; Ivens et al., 2007). This activation increases the expression of glial fibrillary acidic protein (GFAP) and is often associated with temporal lobe epilepsy (Kovács et al., 2012). Furthermore, blood-brain barrier disruption induces albumin and leukocyte spillover, leading to local neuroinflammatory processes, increased neuronal excitability, and reorganization of neuronal networks, all of which promote epileptogenesis (Profaci et al., 2020).

Antiepileptic drug therapy is the first-line treatment for epilepsy caused by venous malformations; surgical resection or other interventions may be effective in patients who respond poorly to medication (Andrea et al., 2015). Clinically, venous dilatation is a potential cause of epilepsy, and neuroimaging in patients with epilepsy of unknown etiology may aid diagnosis and guide clinical decision-making. However, further experimental and large-scale clinical studies are needed to verify the relationship between intracranial venous dilatation and epilepsy.

Conclusion

Dilatation of the deep middle cerebral vein may represent a rare cause of temporal lobe epilepsy. Careful neuroimaging evaluation in patients with unexplained seizures can help identify such vascular anomalies and guide targeted treatment.

Acknowledgement

Not applicable.

Conflict of Interest Statement

The authors declare no conflict of interest.

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