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# Rasmussen Encephalitis: A Late CT Discovery within Partial Seizures in a Young Adult

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**Abstract** **Case Report**

Rasmussen encephalitis (RE) is a unilateral hemispheric encephalitis whose main clinical features include refractory focal epilepsy or epilepsia partialis continua, hemiparesis, and progressive cognitive decline. Despite theautoimmune pathogenesis of RE, the only definitive therapeutic option is currently represented by surgery. This is a case of a 23-year-old women, with a background of partial epilepsy resistant to carbamazepine occurring every 3 or 4 months, which first imaging investigations never shown any plausible cause. The patient had neither history of prenatal disease. She presented to us within a 48 hours of partial, reversible hemiparesis and loss of consciousness. A brain computed tomography (CT) was performed showing a cortical hemiatrophy.

**Keywords:** Rasmussen's encephalitis, partial seizures, cerebral hemiatrophy.



## INTRODUCTION

Rasmussen encephalitis (RE) is a cortical inflammatory condition which evolves in a chronic pattern. It involves children, characterized by drug resistant focal seizures, a progressive paralysis, a cerebral hemiatrophy as well as progressive cognitive decline in the late stages of disease [1] with a presumed immune-mediated pathophysiological basis. Unusual variant forms, including adolescent and adult-onset RE have been described.

## OBSERVATION

We present the case of 23-year-old women, with a background of partial epilepsy resistant to carbamazepine occurring every 3 or 4 months, which first imaging investigations never shown any plausible cause. The patient had neither history of prenatal disease, febrile convulsion, or head trauma nor a family history of epilepsy. She presented to us within a 48 hours of partial, reversible hemiparesis and loss of consciousness. A brain computed tomography (CT) was performed showing a cortical hemiatrophy with a subcortical non enhancing hypodensity, ill-defined, with a minimal cortical involvement. Adding to that is the discreet dilatation of the ipsilateral ventricle (Fig.1). The injection of iodine contrast media didn't show any evidence of enhancement.

Cerebrospinal fluid analysis was acellular without protein or glucose abnormalities and without IgG intrathecal neither synthesis nor oligoclonal bands.

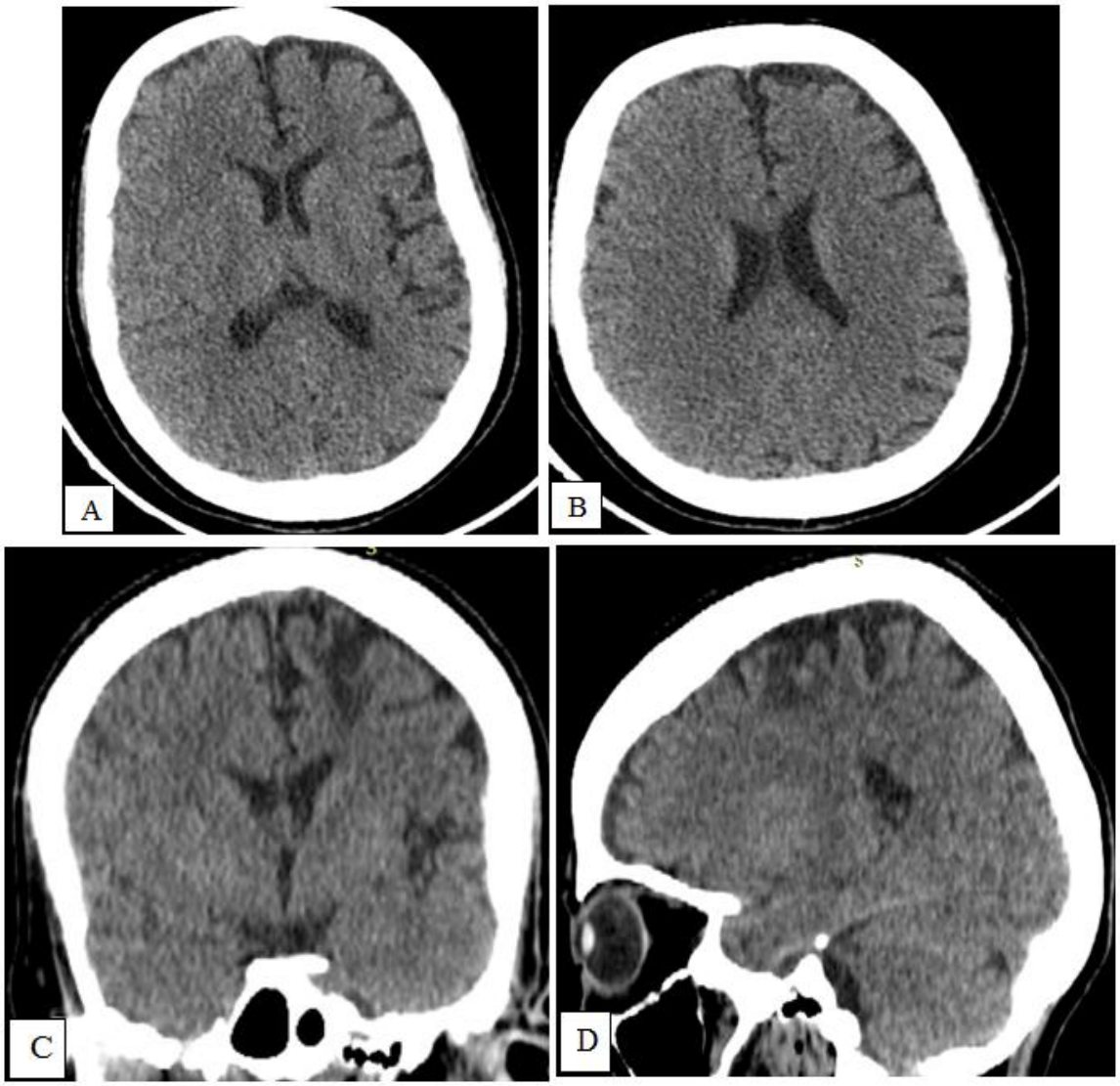


**Fig-1: Axial (A and B), coronal (C ) and sagittal (D) Brain CT showing a left cortical hemiatrophy with a subcortical non enhancing hypodensity, ill-defined, with a minimal cortical involvement. Adding to that is the discreet dilatation of the ipsilateral ventricle**

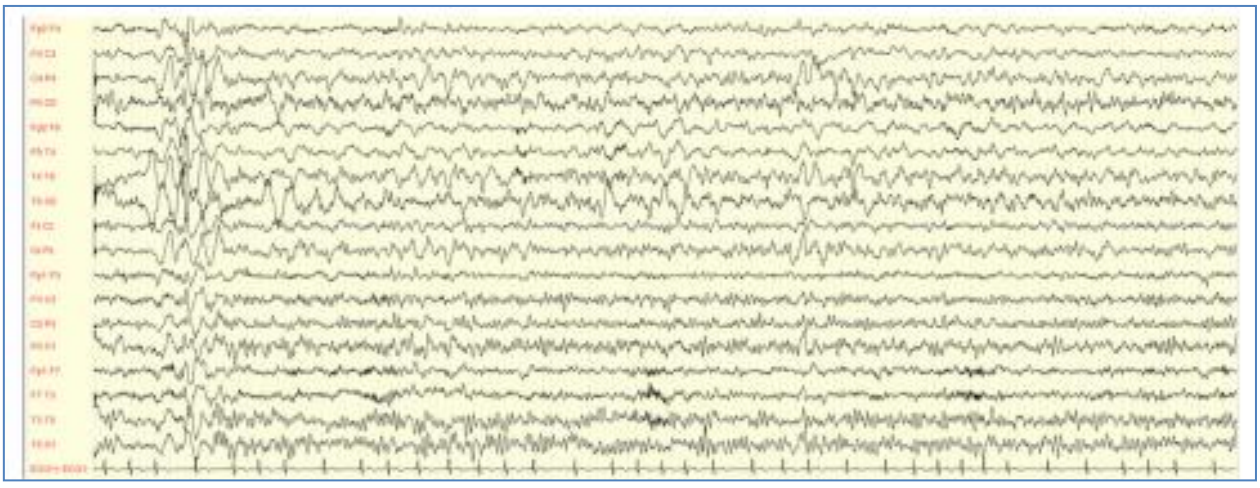

**Fig-2: EEG revealing slowing over the left hemisphere and interictal spike-and-wave discharges in the left fronto temporal region**

## DISCUSSION

Although MRI is more specific to assess the diagnosis, only the Brain CT was available during our shift. CT usually show a cortical hemiatrophy with unilateral hydrocephaly associated with a low attenuated area of the subcortical white matter and absence of contrast enhancement.

MRI may be normal during the first years of early stages the RE or demonstrate cortical swelling with a hyperintense T2/FLAIR signal, followed by a normal volume and a hyperintense signal. Although, after months or years of drug resisting epilepsy [2].

After months or years of drug resisting epilepsy, the typically described MRI features of RE are progressive unihemispheric focal cortical atrophy, commonly involving the frontal and temporal lobes, although the parietal lobe atrophy has also been reported, with a T2-weighted (T2W)/fluid attenuated inversion recovery (FLAIR) hyperintense signal in the grey or white matter, hyperintense T2W/FLAIR signal or atrophy of the ipsilateral caudate head, progression of atrophy on serial imaging and absence of contrast enhancement [1].

A European consensus statement was published in 2005 to aid in the diagnosis and early treatment of Rasmussen's encephalitis, often referred to as the "Bien criteria" [3]. Among them, two criteria from the following are necessary to assess the diagnosis: a- partial seizures or progressive unilateral cortical deficits, b- Imaging findings of progressive unihemispheric atrophy, white and grey matter involvement, and c- histopathological findings of T cell-dominated encephalitis with activated microglia and reactive astrogliosis[4]. 67% of late onset Rasmussen Encephalitis patients fulfil the consensus diagnostic criteria for RE [5].

The brain biopsy may demonstrated diffuse active chronic encephalitis characterized by perivascular and intraparenchymal infiltrate of CD45+ T-cell lymphocytes and microglial activation [6]. The degree to which such antibodies is causative or secondary to a neurologic insult remains in some circumstances unclear [7].

In an adult patient with RE, a favorable prognosis was reached after functional hemispherectomy which safety and efficacy is highlighted [3]. Immunomodulatory therapies, even performed in a late stage, improved late-onset RE patients in 61% of cases [4].

## CONCLUSION

ER produces a characteristic clinico-radiological and electrical epileptic syndrome. It is a severe form of epilepsy, the pathogenesis and treatment modalities of which are still debated.

**Competing interests**

The authors have no conflicts of interest to disclose.

**Authors' contributions**

All the above authors contributed on the writing of this manuscript, the lecture of the imaging studies, or the care of the patient during his hospitalization.

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