

LETTER TO THE EDITOR

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Acute herpetic encephalitis with atypical radiological presentation

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Abstract

A 39-year-old male without any preceding medical ailment presented with 12 day history of fever followed by behavioral changes with left sided weakness. He was found to have Herpes Simplex Virus-1 encephalitis (polymerase chain reaction positive) and with Magnetic Resonance Imaging finding of asymmetrical frontotemporoparietal (right side affected more than left side) involvement with patchy enhancement with atypical nodular enhancement and subtle diffusion restriction. Nodular enhancement is rare in acute inflammations and is reported mainly with chronic granulomatous infections.

Keywords Atypical nodular enhancement, HSV-1 encephalitis, Chronic granulomatous herpetic encephalitis

To the editor

Introduction

Herpes Simplex Virus-1 (HSV-1) is the most common identified cause of sporadic fatal encephalitis. HSV-1 being commoner than Herpes Simplex Virus-2 (HSV-2), accounting for 90% of herpetic encephalitis in adults. Rest 10% of the cases is caused by HSV-2. It usually presents with fever, headache, behavioral disturbance, altered sensorium, seizures and runs a monophasic course usually. Atypical presentations occur in children and immunocompromised adults, such as granulomatous chronic herpes and extralimbic involvement.

Case summary

A 39-year-old male without any preceding ailment came to emergency with history of low grade fever for 12 days followed by change in behavior in the form of decreased talking and decreased interaction with family members. After 3–4 days, family members noticed weakness of left side of body. Patient was unable to recognise family

members and showed disinhibited behaviour. There is no history of seizure or any other movement disorder. Patient exhibited apathy and was non co-operative. He had no neck stiffness. He had left sided hypotonia and hyporeflexia. His plantar reflexes were non elicitable. He was ambulant with unilateral support. His Magnetic Resonance Imaging (MRI) brain showed asymmetrical widespread frontotemporoparietal (right side affected more than left side) involvement with patchy enhancement and diffusion restriction with atypical nodular enhancement (Fig. 1). Cerebro spinal fluid (CSF) showed raised protein levels, increased cells (lymphocytosis), normal sugar and was positive for HSV-1 on polymerase chain reaction (PCR). He was started on acyclovir, and after 7 days of treatment, he started talking and social disinhibition started recovering. He was discharged after 14 days in an independent ambulatory state.

Discussion

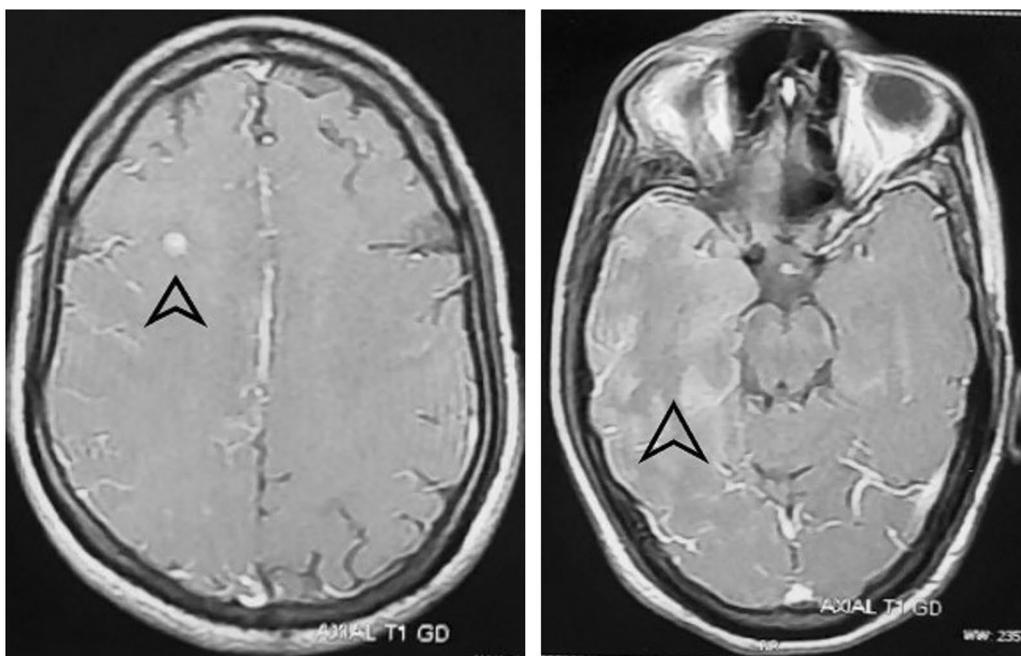
HSV-1 is the most common identified cause of sporadic fatal encephalitis, HSV-1 being commoner than HSV-2, accounting for 90% of herpetic encephalitis in adults remaining by HSV-2. Conversely, HSV-2 is a more common cause of meningitis and neonatal meningoencephalitis than HSV-1. Early recognition is crucial as the efficacy of the antiviral drug acyclovir

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(a)



(b)

Fig. 1 a, b MRI revealing leptomenigeal and gyral enhancement in frontotemporoparietal (right side affected more than left side) with right frontal nodular enhancement

reduces as the disease progresses in reducing mortality and morbidity. Fever is found in 90% and headache in 80% in PCR proven cases. Other common features include disorientation, personality change, focal or generalized seizures, memory disturbance, motor

deficit and aphasia. It usually runs an acute monophasic course and rarely these patients relapse with recurrent symptoms or signs weeks, months or years after the initial episode and such chronic persistent diseases. Our patient with the classical clinical presentation

without any preceding ailment and MRI brain showing asymmetrical frontotemporoparietal (right side affected more than left side) involvement with atypical finding of nodular enhancement. In immunocompromised patients, involvement can be more diffuse, and more likely to involve the brainstem [1]. Herpes Simplex Virus Encephalitis (HSVE) typically affects the limbic system: most frequently the medial temporal lobes, but insular, cingulate and frontobasal cortex are also affected; in 64–68% of cases lesions are unilateral. Brain MRI typically shows area of T2 and FLAIR hyperintensities involving both the cortex and the white matter; areas of contrast enhancement can also be present (Fig. 2). Basal ganglia are usually spared, although a few cases of basal ganglia involvement have been reported in HSVE. Acute cases show high signal on Diffusion Weighted Image (DWI) with low signal in Apparent Diffusion Coefficient (ADC) but during subacute phase of treatment (> 10 days), the ADC and DWI seems less sensitive than T2 and FLAIR imaging for the lesion due to change of signal [2–4]. Enhancement is usually absent early in the disease and occurs later

and is variable in pattern with gyriform enhancement, leptomeningeal enhancement, ring enhancement, diffuse enhancement. The worsening and severe inflammatory changes can destroy the bloodbrain barrier, gyriform, ring-like, or even patchy enhancement may appear later on. More severe presentations may include cortical and subcortical necrosis with hyperintense T1 and hypointense T2 signal with cortical laminar necrosis. Lobar hematoma is rare [5–7]. Therefore, nodular enhancement in our patient is an not a regular finding. Nodular enhancement like in our case usually occurs in young children with an entity called chronic granulomatous herpes encephalitis [8] which is almost exclusively described in children, with rare reports describing the disease in adults. Analysis of cerebrospinal fluid in cases of chronic granulomatous herpes encephalitis is commonly negative for detection of herpes simplex viral deoxyribonucleic acid (DNA) when utilizing polymerase chain reaction detection techniques. Host differences may contribute towards the propensity to develop chronic granulomatous inflammation [9]. This case depicts atypical imaging with widespread

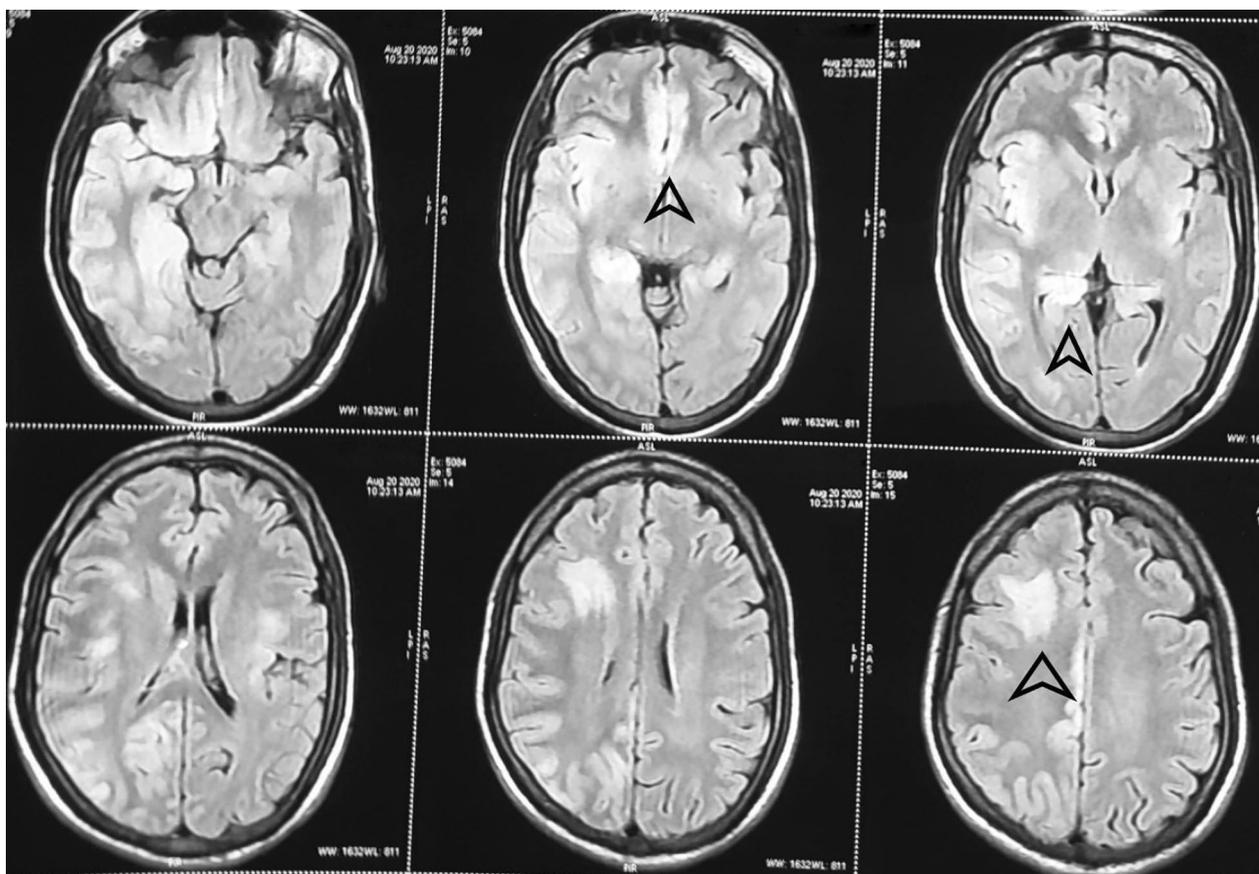


Fig. 2 T2 FLAIR imaging reveals bilateral frontal, temporal and parietal T2 FLAIR hyperintensity (right side affected more than left side)

involvement (fronto-temporo-parietal) with nodular enhancement in acute herpes simplex encephalitis without any evidence of immunosuppression.

Abbreviations

| | |
|-------|-----------------------------------|
| HSV-1 | Herpes Simplex Virus-1 |
| HSV-2 | Herpes Simplex Virus-2 |
| DTR's | Deep tendon reflexes |
| MRI | Magnetic resonance imaging |
| CSF | Cerebro spinal fluid |
| PCR | Polymerase chain reaction |
| HSVE | Herpes simplex virus encephalitis |
| DWI | Diffusion weighted image |
| ADC | Apparent diffusion coefficient |
| DNA | Deoxyribonucleic acid |

Acknowledgements

Not applicable.

Author contributions

MS collected the data and drafted the manuscript. MS was involved in the case directly, and has been involved in revising the manuscript for important intellectual content. DK supervised the making of this case report. VM and CS were involved in the case. All authors read and approved the final manuscript.

Funding

This study was self-funded.

Availability of data and materials

The authors confirm that the data supporting the findings of this study are available within the article [and/or] its supplementary materials.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

All patients included in this research gave written informed consent to publish the data contained within this study.

Competing interests

All authors disclose that they have no conflict of interest that could inappropriately influence this work.

Received: 2 March 2022 Accepted: 16 January 2023

Published online: 06 March 2023

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