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Case Report

The hot cross bun sign in a patient with encephalitis

Yingyan Gan [a], Huici Liang [a], Xiaojing Li [a], Yiru Zeng [a], Wenlin Wu [b], Jing Han [c], Jianning Mai [a,*]

[a] Guangzhou Women and Children's Medical Center, Department of Pediatric Neurology, China
[b] Guangzhou Women and Children's Medical Center, Department of Pediatric, China
[c] Tongji Hospital, Tongji Medical College, Department of Oncology, China

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Abstract

The 'hot cross' bun (HCB) sign refers to pontine cruciform hyperintensity on T2 weighted magnetic resonance image (MRI) which is frequently seen in multiple system atrophy and spinocerebellar ataxia types 2 and 3. We describe a 3 years old boy of encephalitis and his MRI image showed HCB sign in the pontine. After immunosuppressive treatment and followed up 14 months, he got a good outcome and the HCB sign narrowed nearly disappeared.

Keywords: Encephalitis; Hot cross bun; MRI

1. Introduction

The HCB sign refers to pontine cruciform hyperintensity on T2 weighted MRI [1,2]. It is frequently seen in multiple system atrophy and spinocerebellar ataxia types 2 and 3. And it has also been reported in other diseases like cancer [3,4]. We now report the HCB sign in a patient with encephalitis.

2. Case report

Our patient was a 3 years old boy who presented to our hospital with a 45-day fever (40 °C) and headache history and a 36-day rash, not willing to talk, irritability, impaired vision, deafness, dysphagia, double lower limb movement disorder, urinary incontinence history. Cerebrospinal fluid (CSF) a week after this onset at a local clinic showed 38/mm^3, the protein concentration was 0.558 g/L, the glucose concentration was normal. 4 days after onset, influenzas virus, enterovirus and mycoplasma from sample of throat swabs and 7 days after onset, enterovirus from sample of Cerebrospinal fluid were tested in local clinic and the results for these tests were normal. And brain MRI (field strength of 1.50 Tesla) performed 11 days after this onset had not seen abnormities (Fig. 2A&G). One month before visiting our department he had been diagnosed with viral encephalitis and had been treated with acyclovir, dexamethasone and intravenous immunoglobulin (IVIG, 0.4 g/kg for 5 days) for 3 weeks. After treatment, he was afebrile and his rash subsided, but there was no change in other symptoms on admission to our unit. On examination, he was not willing to communicate and has a dysarthria. His vision was impaired so he had to put the

* Corresponding author at: Guangzhou Women and Children's Medical Center, No. 9, Jinsui Road, Tianhe District, Guangzhou City 510623, Guangdong Province, China.
E-mail addresses: lhc588@yeah.net (H. Liang), wenlinwu@csu.edu.cn (W. Wu), 761740039@qq.com (J. Han), maijianning_new@163.com (J. Mai).

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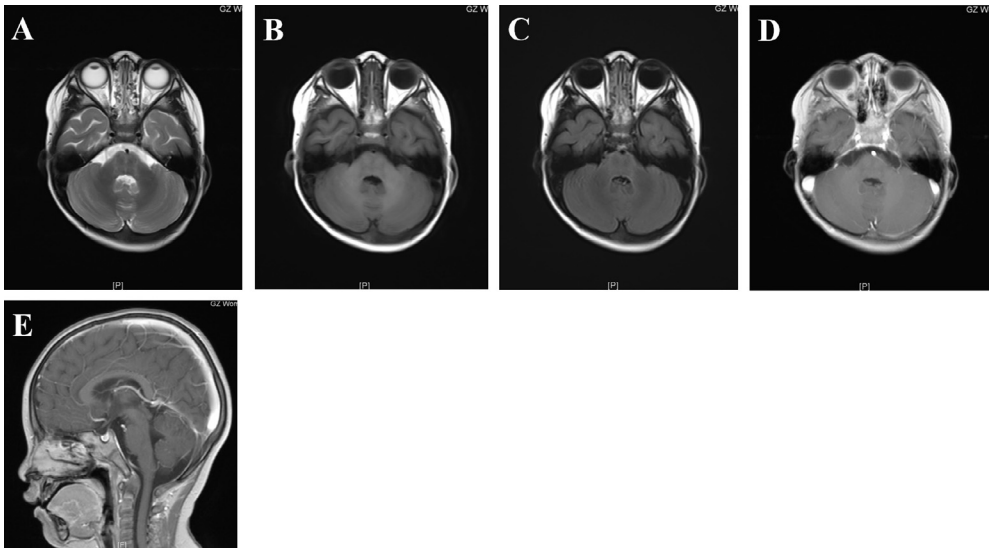

Fig. 1. Cerebral MRI (field strength of 3.0 Tesla) on 48 days after onset in our department. T2-axial showed cruciform-sharped hyperintensity in the pons (A). while it was mild hypointensity signal in T1-axial (B) and in T2 fluid-attenuated inversion recovery (FLAIR) (C). It was not enhanced in T1-axial with contrast medium (D). T1-sagittal with contrast medium (E). No significant atrophy was found in cerebellar (A-E).

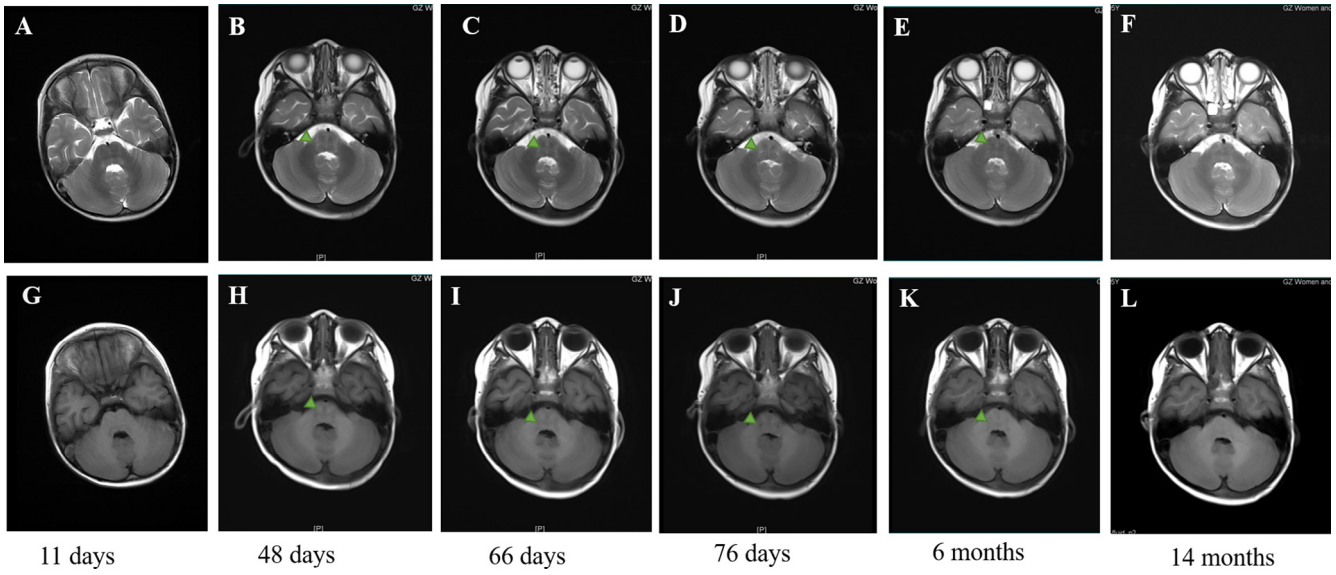

Fig. 2. Cerebral MRI (field strength of 1.5 Tesla) on 11 days in local clinic department and Cerebral MRI (field strength of 3.0 Tesla) from 48 days to 14 months after onset in our department. T2-axial showed cruciform-sharped hyperintensity signal in the pons and improved gradually (A-E). T1-axial showed cruciform-sharped hypointensity signal in the pons and improved gradually (F-J).

phone 5–10 cm away from his eyes in order to play the phone. His eyes had a esotropias and eyes' abduction were limited, but did not present nystagmus. The muscle bulk of lower limbs decreased slightly. The muscle strength of the lower limbs were graded on III and the muscle tone of them increased like clasp-knife phenomenon, while these of the upper limbs were normal. He had tremor in the upper limbs. Sensory evaluation and deep tendon reflexes were normal. Babinski' sign was spontaneous positive. Laboratory investigations in our department including routine blood chemistry, vasculitic and routine test, biochemistry indicators and etiology of CSF showed no remarkable findings. 50 days after onset, enterovirus, mycoplasma, adenovirus, herpes simplex and chlamydia pneumoniae from sample of throat swabs, enterovirus from sample of stool and enterovirus, mycoplasma, herpes simplex, and chlamydia pneumoniae from sample of throat swabs Cerebrospinal fluid were tested. Serum Ig G and Ig M antibody for enterovirus, mycoplasma, influenzas virus and herpes simplex were tested. Antibody to N-Methyl-D-Aspartate-Receptor in both CSF and serum

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