Clinical Case Report

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Abnormal parietal encephalomalacia associated with schizophrenia

A case report

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Abstract

Rationale: It is widely believed that structural abnormalities of the brain contribute to the pathophysiology of schizophrenia. The parietal lobe is a central hub of multisensory integration, and abnormities in this region might account for the clinical features of schizophrenia. However, few cases of parietal encephalomalacia associated with schizophrenia have been described.

Patient concerns and Diagnoses: In this paper, we present a case of a 25-year-old schizophrenia patient with abnormal parietal encephalomalacia. The patient had poor nutrition and frequently had upper respiratory infections during childhood and adolescence. She showed severe schizophrenic symptoms such as visual hallucinations for 2 years. After examining all her possible medical conditions, we found that the patient had a lesion consistent with the diagnosis of encephalomalacia in her right parietal lobe and slight brain atrophy.

Interventions: The patient was prescribed olanzapine (10 mg per day).

Outcomes: Her symptoms significantly improved after antipsychotic treatment and were still well controlled 1 year later.

Lessons: This case suggested that parietal encephalomalacia, which might be caused by inflammatory and infectious conditions in early life and be aggravated by undernutrition, might be implicated in the etiology of schizophrenia.

Abbreviations: IQ = intelligence quotient, MRI = magnetic resonance imaging, PANSS = Positive and Negative Syndrome Scale.

Keywords: case report, malnutrition, parietal encephalomalacia, schizophrenia, young woman

1. Introduction

Schizophrenia has been regarded as a neurodevelopmental disorder, and psychosis is a late, potentially preventable stage of the illness.[1] Many factors can significantly influence the development of the brain, such as viral infections.[2] One type of infection-related brain lesion, encephalomalacia, has been reported in schizophrenia patients.[3,4] However, parietal lobe encephalomalacia has rarely been reported in patients with schizophrenia. In this report, we present the case of a 25-year-old undernourished schizophrenia patient with an aberrant parietal encephalomalacia. We speculate that the emergence of her psychosis in adulthood might be associated with this parietal lesion and her malnutrition. The present study complies with the Declaration of Helsinki (Edinburgh version, 2000). It was approved by the ethics committee of The First Affiliated Hospital of Zhejiang University, and written informed consent was obtained from the patient and her parents.

2. Case presentation

A 25-year-old Chinese woman was admitted to our hospital due to a 2-year history of soliloquy, bizarre smile, and visual hallucination. Her condition had been worsening in the previous 6 months before admission. She had also experienced decreased social activity, poverty of thinking, and apathy. Consequently, she could not continue to work and had to stay at home.

The patient reported no history of trauma, epilepsy, alcohol consumption, or substance use. According to her mother, she was not significantly different from her peers in the growth and development process, except for her timid and introverted character. The patient passed the senior high school entrance examination, although her score was a little below the class average. Additionally, she successfully graduated from a vocational and technical school when she was 19 years old. However, she had poor nutrition and frequently had upper respiratory infections during childhood and adolescence. In addition, she had a poor appetite for 5 years. In the last 3 years, her weight decreased gradually, and the average decline was 2 to 3 kg per year. Her mother took her to the gastroenterology department but no abnormality was found. Gastroscopy only indicated superficial nonatrophic gastritis, and she had no thought of intentionally controlling her diet to achieve a slim

Editor: Zelena Dora.

Funding/support: This work was supported by the Department of Health Foundation of Zhejiang Province (2014RCA005) and the Key Research Project of Zhejiang Province (No.2015C03040 and 201503054).

Ethics approval and consent to participate: This study was approved by the ethics committee of The First Affiliated Hospital of Zhejiang University and written consent was obtained.

The authors have no conflicts of interest to disclose.

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Medicine (2017) 96:10(e6310)

Received: 16 December 2016 / Received in final form: 20 January 2017 / Accepted: 10 February 2017

http://dx.doi.org/10.1097/MD.0000000000006310

Table 1

Wechsler Adult Intelligence Scale-Revised.

Wechsler Adult Intelligence Scale-Revised			
Verbal tests		**Performance tests**	
Subtests	Score	Subtests	Score
Information	11	Digit symbol	7
Comprehension	11	Picture completion	12
Arithmetic	7	Block design	10
Similarities	9	Object assembly	6
Digit span	5	Picture arrangement	9
Vocabulary	9		

Table 2

V-P difference.

V-P difference			
Verbal IQ	**Performance IQ**	**V-P difference**	**Significant level**
88	91	3	–

IQ = intelligence quotient, V-P = verbal–performance.

figure. Her menstruation had been irregular for 2 years and once stopped for 3 months. Furthermore, she had a short marriage but divorced because of her husband's addiction to gambling. No family history of psychiatric illness was reported. This young woman was 1.57 m in height and 35 kg in weight, with a body mass index of 14.1. Physical examination showed that her waistline was 63 cm, her hipline was 81 cm, and her abdominal skinfold thickness was 12 mm. Her neurological signs were negative. A full psychiatric assessment revealed symptoms of apathy, social withdrawal, stupor, and waxy flexibility. She complained of visual hallucinations with various scenes and occasional olfactory hallucinations involving smoke. Cognitive function was measured by the Wechsler Adult Intelligence Scale-Revised (Tables 1 and 2), and the severity of schizophrenia symptoms was assessed by the Positive and Negative Syndrome Scale (PANSS) upon admission. Her full-scale intelligence quotient (IQ) was 89 and PANSS total score was 105. In addition, her routine blood test and thyroid function, liver panel, and cerebrospinal fluid examinations were all normal. However, cranial magnetic resonance imaging (MRI) scan revealed an abnormal encephalomalacia lesion in her right parietal lobe and slight brain atrophy (Fig. 1). Her magnetic resonance angiography examination showed no abnormality.

According to the diagnostic criteria for schizophrenia in the International Statistical Classification of Diseases and Related Health Problems, 10th revision, she was diagnosed with schizophrenia and prescribed olanzapine (10 mg per day). Her symptoms, including hallucinations, soliloquy, and bizarre smile, significantly improved, whereas her apathy remained. She was discharged from the hospital after 4 weeks of hospitalization.

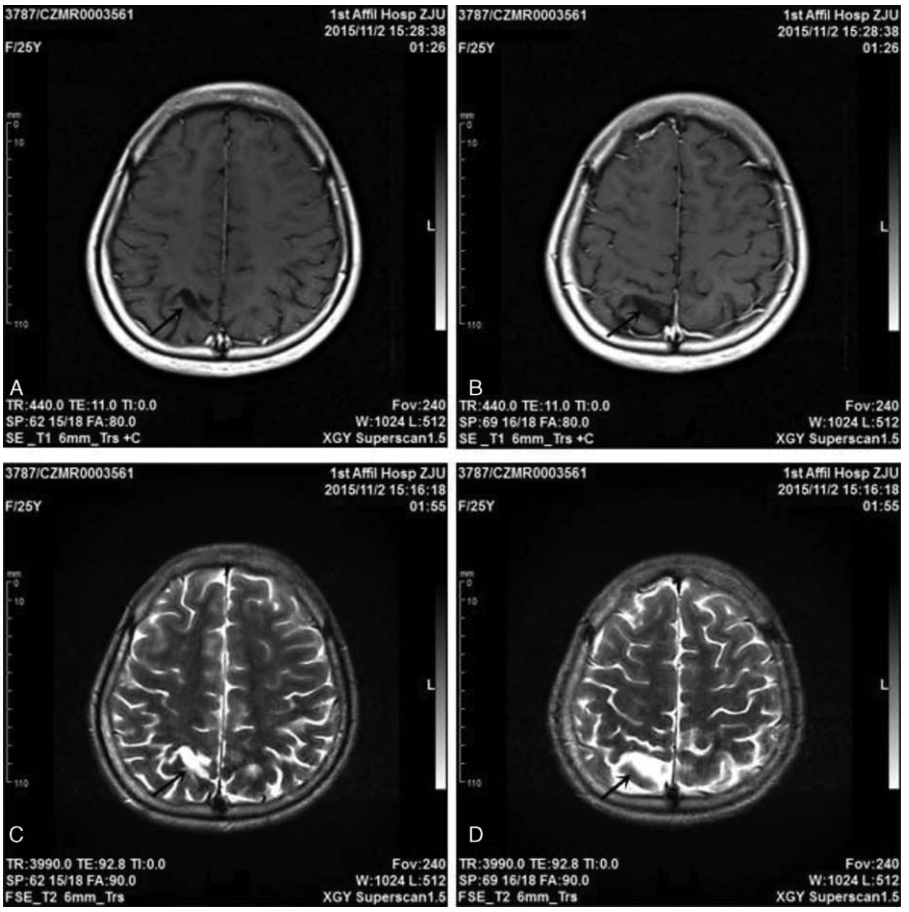

Figure 1. (A, B) T1 cranial magnetic resonance imaging (MRI) scan shows an abnormal signal in the gray and white matter junction of the parietal lobe. (C, D) T2-weighted cranial MRI scan indicates a focal lesion in the right parietal lobe with abnormally increased intensity, which was believed to be encephalomalacia. (A–D) Given her age, the sulci were widened and the gyri were shrunk, which suggests slight brain atrophy.

This patient returned to our department for 2 separate follow-up visits after 4 months and 1 year. During this period, her psychotic symptoms disappeared, and her motor activities were more flexible. She could complete some handicraft work at home. Her mother paid more attention to her nutrition, and her appetite improved after initial treatment. After 4 months, her PANSS total score was 57, and her full-scale IQ was 90 during the visit. Moreover, she was 1.57 m in height and 45 kg in weight, with a body mass index of 18.3. Physical examination showed that her waistline was 70 cm, hipline was 85 cm, and abdominal skinfold thickness was 22 mm. Her condition did not change significantly, except for a total PANSS score of 47 after 1 year. Her cranial MRI scan result also had no obvious change.

3. Discussion

Encephalomalacia in schizophrenia patients has been previously reported in the periventricular regions of brain.[5] Inflammatory and infectious conditions are believed to be involved in the development of encephalomalacia.[4] It is worth noting that exposure to prenatal and childhood infections is also associated with an increased risk of schizophrenia.[2,6] However, the pathophysiology of the correlation between infections and schizophrenia still remains unclear. The patient in our case had a history of abnormally frequent upper respiratory infections during childhood and adolescence. Upper respiratory tract infections are caused by many different bacteria (such as *Streptococcus*) and viruses (such as influenza virus). Infection-related encephalomalacia has been previously reported in children (caused by group B streptococcus)[7] and rats (caused by influenza virus).[8] In addition, a review of the literature showed that 5% of acute childhood encephalitis is associated with influenza.[9] One of the potential mechanisms that underlies influenza-related encephalopathy is the direct invasion of the central nervous system, which is supported by the evidence showing that the influenza virus has been detected in the cerebrospinal fluid by PCR in some cases of encephalitis.[10] Another hypothesis suggests that systemic immune response plays an important role in pathogenesis. High levels of proinflammatory cytokines, including IL-6 and TNF-alpha, have been found in the serum and cerebrospinal fluid of children with influenza-associated encephalopathy.[11] Given the condition of the patient in our case, we speculate that infection might have contributed to the development of her abnormal parietal encephalomalacia, which was related to her subsequent schizophrenic symptoms and diminished cognitive function.

The parietal cortex is involved in various neuropsychological functions that are impaired in schizophrenia patients, such as the senses, cognitive function, and self-awareness.[12,13] Previous studies also found that lesions in the white matter of the parietal lobe disrupt fronto-parietal connectivity and might be responsible for the motor deficits as well as the social dysfunction and negative symptoms in schizophrenia patients.[14] In our case, the patient's encephalomalacia in the junction of the gray and white matter of the parietal lobe might be clinically significant and correlated with her hallucinations, negative symptoms, and social dysfunction. In addition, Yildiz et al[15] proposed that structural and functional alterations might start in the parietal lobe and progress to the frontal regions in a small proportion of individuals with emerging schizophrenia.

The patient in the present report is right-handed. Additionally, according to the Wechsler Adult Intelligence Scale-Revised test, her verbal IQ and performance IQ were not different. However, after precise analysis, we found that she obtained lower subtest scores on arithmetic, digit span, digit symbol, and object assemble, which might indicate her poor functioning in quantitative reasoning, attention, and processing speed.[16] In general, the right brain is responsible for attention, spatial perception processing, and approximate arithmetic (language independence).[17] Therefore, the patient's detailed Wechsler Adult Intelligence Scale scores might also conform to her right hemisphere lesion.

Notably, brain atrophy was also found in our patient. This finding should not be neglected, as it might be attributable to severe malnutrition.[18] Studies found that brain is at 95% of the maximum total size by age 6; however, cortical and subcortical components of the brain change dramatically during childhood and adolescence.[19] Certain types of nutritional deficiency such as protein undernutrition, iron deficiency, and iodine deficiency clearly impair oligodendrocytes and microglia, which are responsible for myelination and damage repair, respectively.[20,21] Researchers also found that the abnormality of oligodendrocytes differentiation, myelination, antioxidant enzyme production, and synaptogenesis could make brains vulnerable to cerebral white and gray matter injury during developmental processes.[22] Moreover, the neurodevelopment perspective hypothesizes that psychosis does not arise from a completely healthy brain and that a lesion that emerges early does not manifest until a much later developmental stage when compensatory changes can no longer suffice.[1] Therefore, with regard to our case, we suspected that her parietal encephalomalacia appeared in the early stage and that the emergence of typical schizophrenic symptoms in recent years might have been the result of the more damaged brain structure and functions caused by malnutrition. Evidence suggested that some but not all of the negative effects of early malnutrition on brain development can be reversed through subsequent improvement in nutrition, health care, and enriched environments.[20] In our case, after 4 months of care and treatment, the patient's nutrition improved a great deal, and she gradually gained 10 kg in weight. Although her cranial MRI scan showed no obvious change, we still suggested that it be reevaluated after a period of time.

An organic lesion of the brain should always be considered when attempting to detect the etiology of schizophrenia. This case might also provide new evidence to support the association between early-life infection, malnutrition, and subsequent schizophrenia. Our case might be helpful in the prevention of schizophrenia.

Acknowledgments

The authors thank all the participants in this study and the staff of the Department of Mental Health, The First Affiliated Hospital, School of Medicine, Zhejiang University. The authors also thank the Department of Health Foundation of Zhejiang Province (2014RCA005) and the Key Research Project of Zhejiang Province (No. 2015C03040 and 201503054) for the support.

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