

qEEG in the Diagnosis and Prognosis of a Case with Delusional Infestation

Clinical EEG and Neuroscience
1–5
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DOI: 10.1177/15500594231163383
journals.sagepub.com/home/eeg



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Abstract

Stroke can have neuropsychological consequences, such as poststroke psychosis. One aspect of poststroke psychosis is delusional infestation (DI), also known as delusional parasitosis. Patients with DI have fixed sensations that they get infested by lice. Some explanations of DI indicate that striatal and fronto-cortical structural and functional abnormalities are related to DI symptomatology. In this case report, we present a patient with DI due to right intracarotid artery occlusion, which is detected radiologically. Before treatment with escitalopram and risperidone, the case had a frontal slowing in quantitative electroencephalography activity, which was normalized after 2 weeks of treatment. At the end of treatment, psychotic symptoms and OCD symptoms evaluated by the Yale-Brown Obsessive Compulsive Scale, the Scale for the Assessment of Positive Symptoms, were remarkably reduced. These results revealed the role of brain imaging studies in the diagnosis and prognosis of DI.

Keywords

delusional infestation, qEEG, MRI, stroke, cerebrovascular attack, secondary psychosis

Received December 7, 2022; revised January 28, 2023; accepted February 22, 2023.

Introduction

A stroke is an acute vascular event that can have neuropsychiatric consequences, disabling complications, and death.^{1,2} After a stroke attack, psychosis, mania, and depression are reported in several studies.³ One clinical manifestation of poststroke psychosis is delusional infestation (DI). DI is a psychodermatologic condition characterized by compulsive itching without any infection history.⁴

Aetiologically, DI can be primary such as in schizophrenia, or without any other psychiatric disorder. The latter case will appear secondary to a neurocognitive problem called organic psychosis.⁵ Organic psychosis is “the emergence of hallucinations or delusions in a patient with a recently acquired disturbance of brain function.”⁵

Large-population evidence as to the pathophysiology of the DI is lacking, but only several of the case series.^{6,7} There are some speculative explanations regarding the pathophysiology of DI, such as striatal dopaminergic dysfunction and abnormal extracellular dopamine levels in basal ganglia.⁸ However, this could explain antipsychotic treatment efficacy in primary DI patients. A systematic review states the similar efficacy of typical and atypical psychosis in primary DI.⁹ Nearly 75% of patients are expected to recover after first-line antipsychotic treatment.¹⁰ Antipsychotic treatment has also been demonstrated to be effective in the secondary DI.¹¹ When striatal dopaminergic activity is considered, this evidence may consolidate the dopaminergic approach to DI.¹¹

Neuroimaging techniques reveal the neural correlates of DI. For instance, in primary somatic hallucinations, a case diagnosed with schizophrenia reporting getting touched showed electrophysiological abnormalities in the quantitative electroencephalography (qEEG) gamma band in the somatosensory cortex.¹² The author associated this result with psychosomatic sensations.¹² Although fewer studies evaluated DI in a large sample, an investigation using EEG and computed tomography indicated that approximately 47.7% of the patients with pure DI had slowing activity in the EEG theta band.¹³ As for secondary psychosis of DI, an earlier case report indicated that a patient with postherpetic neuralgia developed DI and had an EEG abnormality in the left anterior parietal region.¹⁴ On the other hand, some patients with DI did not show EEG abnormalities but abnormal magnetic resonance imaging (MRI) findings.¹⁵

Some studies investigating the structural difference between people with DI and healthy control (HC) showed that patients with DI had greater cortical thickness in the right medial orbitofrontal cortex (mOFC) and less in temporoparietal areas compared with HCs.¹⁶ The authors linked these findings with

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previous studies which associate symptom misattribution of schizophrenic patients with structural abnormalities in mOFC and temporal area.¹⁶ Another study comparing MRI findings between DI and HC indicated that DI patients had less gray matter volume (GMV) in frontal and mediotemporal cortical regions and higher white matter volume (WMV) in cingulate and striatum regions.¹⁷ As for the secondary psychosis of DI, a case report revealed that a patient with mild vascular encephalopathy developed DI.¹⁸ Then, her MRI findings were compared with seven HCs. The results indicated that the patient had GMV abnormalities in the frontal cortex, parietal cortex, and putamen regions, along with perfusion.¹⁸

In this case report, we present a patient with DI secondary to a neurocognitive problem which is a cerebrovascular accident (CVA). The possible structural and functional neural correlates of DI were investigated through qEEG and MRI.

Case

A 71-year-old female patient presented with the following complaints referred to our private psychiatry clinic; pruritus, DI, persecutive thoughts, and compulsive hand-washing behavior nearly 35 times a day.

The case underwent a cerebrovascular attack four months before her first visit to our clinic. This attack impacted her speech and arm, leading to dysphasia and weakness in the hand. Then, she was hospitalized. The neurological intervention showed a blockage in the brain vessels, specifically in the right ICA. After a stent operation, the dysphasia problem was resolved.

After the cerebrovascular attack, she first consulted a dermatologist with infestation problems. The dermatologic examination revealed no somatic symptoms; thus, she was referred to a psychiatrist. The referred psychiatrist diagnosed her with OCD and started treatment with escitalopram 10 mg and olanzapine 5 mg. However, the patient reported that she did not benefit from that treatment within 4 weeks, and it caused weight gain.

Psychiatric Examinations

Upon admission to our clinic, she was examined following the Structured Clinical Interview for DSM-V (SCID-5).¹⁹ In addition, the psychiatrist applied a structured mental status examination²⁰ covering appearance, behavior, motor activity, speech, mood, affect, thought process, thought content, perceptions, cognition, insight, and judgment of the patient.

Neuroimaging Techniques

A routine qEEG recording was performed in the clinic. The report identified an intense delta increase in the left hemisphere and an increase in theta in the frontal region (Figure 1A). The results were interpreted as general slowing activity. Then, MRI was requested. The three-dimensional time of flight magnetic resonance angiography image demonstrated no flow-related signal in the right ICA petrous and cavernous segment (Figure 2A). On T2-weighted axial images, there is an increase in signal consistent with gliosis in the right frontal lobe (Figure 2B) in favor of chronic CVA and cystic encephalomalacia, including the head of the caudate nucleus and lentiform nucleus from the cortico-subcortical area to the deep white matter. Affected cortical areas were the medial, inferior frontal gyri, and striatum.

Diagnosis and Treatment

As a result of qEEG, MRI, patient history, and psychiatric examination, the patient was diagnosed with Psychotic Disorder due to Another Medical Condition according to DSM-5.²¹ Her psychotic feature was considered a DI, and the medical condition was cerebrovascular disease.

The patient was started with a single daily dose of an atypical antipsychotic for her psychotic features, risperidone 2 mg, and a daily dose of selective serotonin reuptake inhibitor, escitalopram 10 mg for her OCD-related symptoms, i.e. excessive

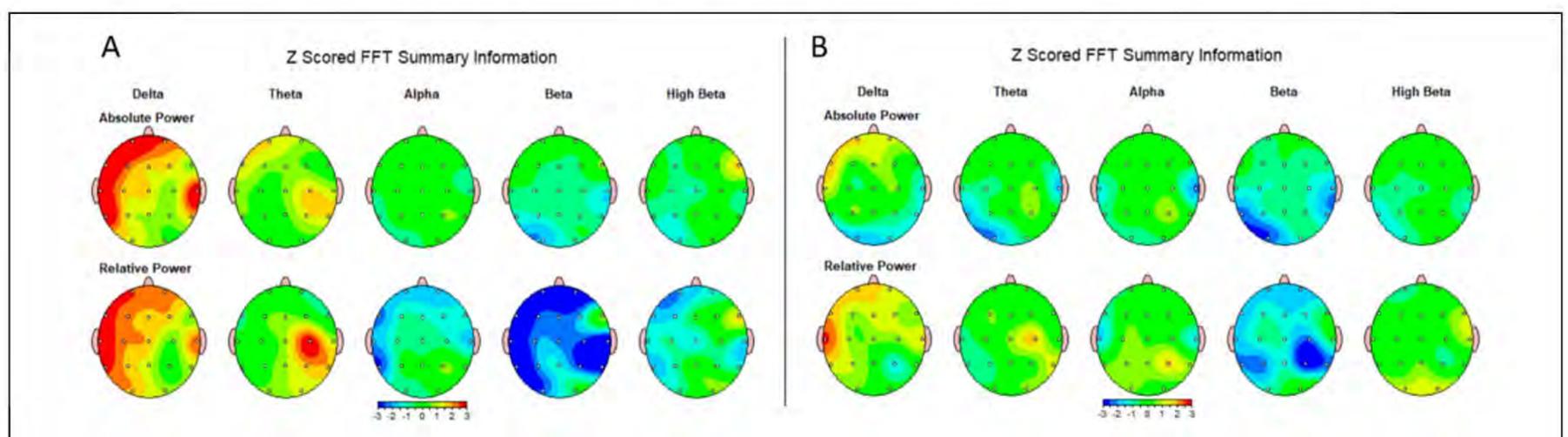


Figure 1. Shows the average Z-score maps of absolute and relative power for the delta, theta, alpha, beta, and high beta frequency bands obtained from pretreatment (A) and posttreatment (B) quantitative electroencephalography (qEEG) recordings. The absolute and relative power deviations in qEEG Delta and Theta frequency represent the increase in power, whereas deviations in Beta frequency indicate the decrease in absolute and relative power.

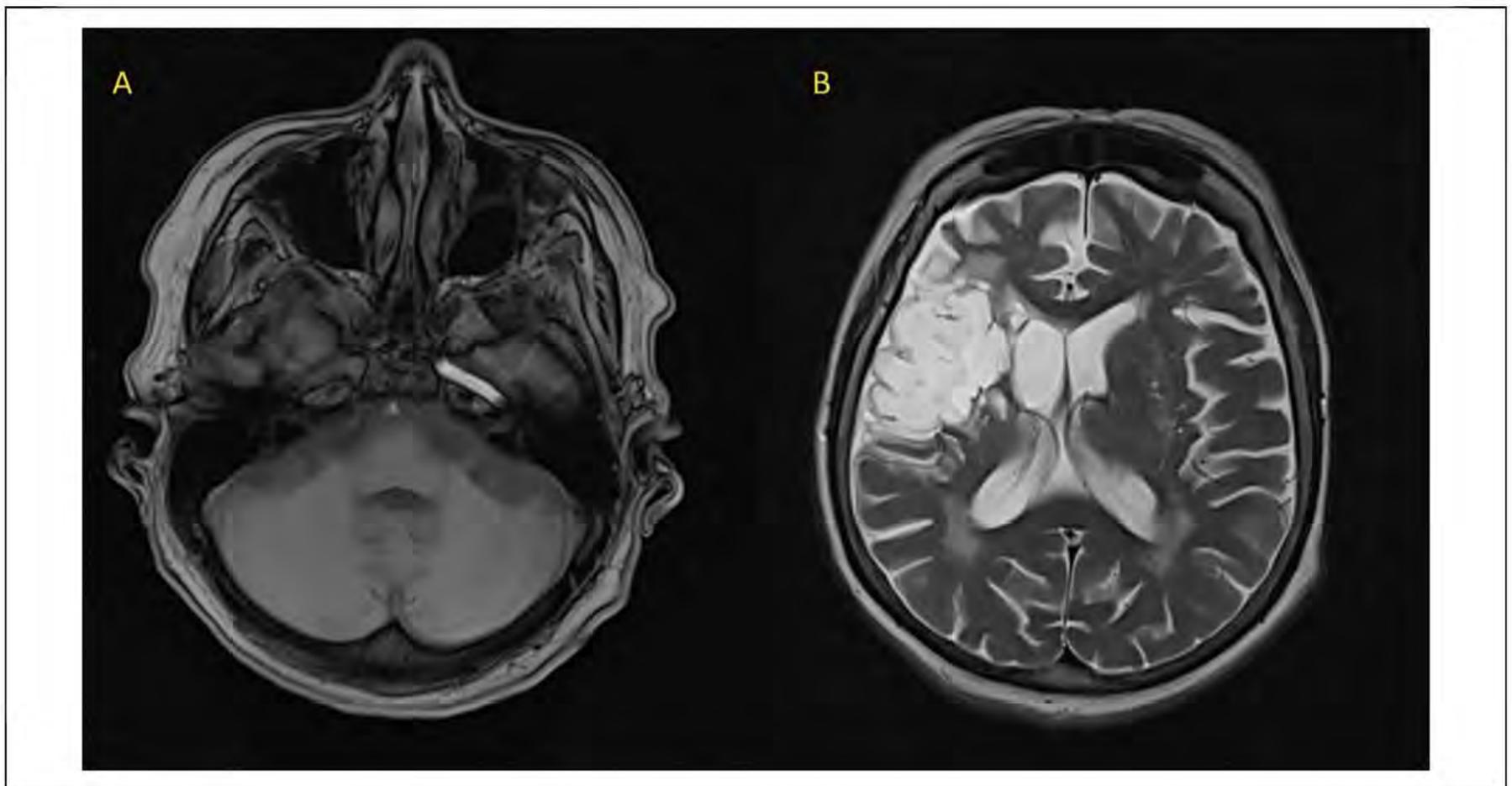


Figure 2. Depicts pretreatment images of MRI. (A) The 3D time of flight MRA image. (B) T2-weighted axial image. Abbreviations: 3D, three-dimensional; MRI, magnetic resonance imaging.

hand washing and counting rituals. After 15 days, she was re-examined, and qEEG was rerecorded.

qEEG Acquisition

All qEEG data were procured in a quiet, dimly illuminated room. Neuron-Spectrum 4-P Clinical Diagnostic EEG System (Neurosoft) was used to acquire EEG signals. A cap with 19 electrodes (FP1, FP2, F3, F4, F7, F8, C3, C4, P3, P4, T3, T4, T5, T6, O1, O2, Fz, Cz, and Pz) based on the international 10 to 20 system connected to the EEG amplifier. FPz was selected for the ground electrode, and reference electrodes were mastoid electrodes (A1-A2). The data-sampling rate was 500 Hz, and the acquired signals were band-pass filtered at 0.15 to 70 Hz and notch filtered at 50 Hz. Participants sat restfully with their eyes closed during the 7-min recording time. Acquired data was saved in European Data Format and transferred to the QEEE analysis program.

qEEG Analysis

Neuroguide Deluxe v.2.5.1 (Applied Neuroscience) software was used for qEEG analysis. First, data artifacts were manually eliminated offline by an experienced qEEG technician. After artifact elimination, 3 min artifact-free data were obtained. Then, Fast Fourier Analysis was applied. The data were averaged across the recording epochs for each electrode. The absolute power (percentage of total power) was computed for each of the following bands:

delta (1-4 Hz), theta (4-7 Hz), alpha (8-12 Hz), alpha1 (8-10 Hz), alpha2 (10-12 Hz), beta (12-25 Hz), beta1 (12-15 Hz), beta2 (15-18 Hz), beta3 (18-25 Hz), high beta (25-30 Hz), gamma (30-50 Hz), gamma1 (30-35 Hz), gamma2 (35-40 Hz), high gamma (40-50 Hz). Finally, a Z-scored qEEG topographic brain map was constructed through the software.

Second Examination

The patient's complaints were significantly reduced at the examination two weeks later. The patient said that she no longer felt lice crawling. At the same time, the patient's counting ritual and excessive hand washing disappeared. In the second qEEG recording, the delta and theta, which were excessively increased in the first recording, decreased noticeably (Figure 1B).

Scores on clinical scales also decreased significantly. Yale-Brown Obsessive-Compulsive Scale score decreased from 40 to 2. The Scale for the Assessment of Positive Symptoms (SAPS) score decreased from 13 to 0. Finally, on Global Impression Scale (CGI), the disease severity was decreased from 5 to 2. No side effects were observed.

Discussion

In this case, we selected qEEG and MRI techniques to make a proper diagnosis, which was the first step in selecting the optimal treatment regime. The qEEG results indicated that

the local slow brain activity might result from a structural abnormality in the corresponding regions. Then, MRI results confirmed this perspective. These neuroimaging results and patient history led to the diagnosis of organic brain syndrome with psychotic and OCD-related features.

The patient's qEEG and MRI results are partially concordant with previous studies. Like DI patients with slowing EEG theta activity,¹³ our patient also had increased theta power in the left frontal and right parietotemporal regions. Furthermore, she had increased delta activity located in the left frontal, temporal, and parietal regions. In line with these results, a case with DI also reports EEG abnormalities in the left anterior parietal area.¹⁴ As for MRI findings, previous studies also showed structural differences between HCs and DI patients in that DI patients had less cortical thickness in parieto-temporal regions.¹⁶ Finally, a case with DI secondary to cerebrovascular attack had GMV abnormalities similar to our case in the frontal cortex, parietal cortex, and putamen regions.¹⁸

From all these results, our case with right ICA occlusion and her associated symptomatology can have neural correlates in the frontal cortex and striatum lesions which were previously shown to be related to poststroke psychosis.^{3,22} There are substantial clues indicating that the symptoms of patients with DI secondary to neurocognitive problems emanate from the striatum and putamen lesions. Therefore, this tactile hallucination problem is related to the visual-tactile-perceptive function of the putamen and circuits, including the striatal-thalamic-cortical dorsal somatic gyrus.⁸

As for the treatment strategy, since the patient reported that olanzapine did not show any effect and caused weight gain, another potent atypical antipsychotic, risperidone, was preferred in the treatment. As later examinations show, risperidone worked well on the psychotic symptoms with no reported side effects in the chosen dosage. As for the anti-obsessive/anti-depressive compound, we extended the treatment duration with the same SSRI medication expecting the therapeutic response will appear within that period.

Based on this report, it could be suggested that clinicians can make the proper diagnosis using brain imaging techniques. In this way, the clinician can save time which has clinical and ethical importance. Furthermore, in the case of therapy-resistant patients, the patient can benefit from pharmacogenetic testing in which the pharmacokinetically most suitable medications are suggested.^{23,24} Finally, this case report highlights that qEEG can be used successfully in following up on the clinical outcome in patients with organic brain syndrome. However, more sample size is needed to generalize this electrophysiological proposal.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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