Case Report

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When to Choose Paroxetine Treatment in Skin-Picking Disorder: A Case Report

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Abstract

Skin picking disorder (SPD) characterized by repetitive compulsive scratching in the absence of a primary skin disease is strongly associated with psychiatric comorbidities, including obsessive-compulsive disorder (OCD) and depression (MDD). Selective serotonin reuptake inhibitors (SSRIs) have been used in the treatment of SPD with variable success. Nevertheless, the optimum treatment choice for SPD is an issue for clinicians. This case report presents a 32-year-old female SPD patient treated with four-week paroxetine monotherapy. Based upon the clinical interview and standardized questionnaires, the patient was diagnosed with OCD with depressive features and Skin Picking Disorder. In addition to symptom severity scales, quantitative electroencephalography (qEEG) was also applied. Paroxetine treatment was started (titrated from 5 to 40 mg/day) and doubled each week. After four-week paroxetine monotherapy, OCD symptoms were diminished, and skin lesions were completely regressed leaving solely post inflammatory hyperpigmentation. Post-treatment qEEG assessment also showed a normalization of frontal alpha power and amplitude asymmetry. It can be concluded that if OCD includes SPD with abnormal EEG patterns; then the treatment success using paroxetine will be very high.

Keywords

alpha oscillations, neurotic excoriation, obsessive-compulsive disorder, paroxetine, QEEG, skin-picking disorder

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Introduction

Skin picking disorder (SPD), also known as psychogenic excoriations or excoriation disorder is characterized by repetitive, compulsive scratching or picking of the skin in the absence of any primary skin disease.[1] It was classified under the obsessive-compulsive and related disorders (OCRD) in the Diagnostic and Statistical Manual of Mental Disorders fifth edition (DSM-V).[2] Although the word "neurosis" has been removed from the Diagnostic and Statistical Manual of Mental Disorders, the term "neurotic excoriation" is also still vastly used among dermatologists.[3] SPD is strongly associated with psychiatric comorbidities, including obsessive-compulsive disorder, depression, and bipolar disorder.[4] Selective serotonin reuptake inhibitors (SSRIs) have been used in the treatment of OCD and related disorders,[5] including SPD.[6] Response rate of patients to SSRIs is approximately 50%.[7] Therefore, investigating treatment response via objective measures such as quantitative electroencephalography (qEEG) plays a crucial role in clinical psychiatry.

In this case report, we describe the complete resolution of cutaneous lesions along with normalization of quantitative electroencephalography (qEEG) findings in a patient with SPD following paroxetine monotherapy.

Case Report

The patient signed an informed consent form for the publication of patient information, including images. A 32-year-old woman was referred to our psychiatry outpatient clinic from dermatology clinic, with a one-year history of severe pruritus. Dermatologic examination of the patient had revealed multiple angulated, linear, sharply demarcated erosions located on flexor aspects of arms and chest, which was consistent with neurotic excoriations (Figure 1A and B). The patient did not have a history of atopy and the results of laboratory studies (complete blood count, fasting glucose level, liver, and renal functions, thyroid hormones) were within normal limits. Topical steroids and local anesthetics were ineffective to reduce the severe itch, which was worse at night.

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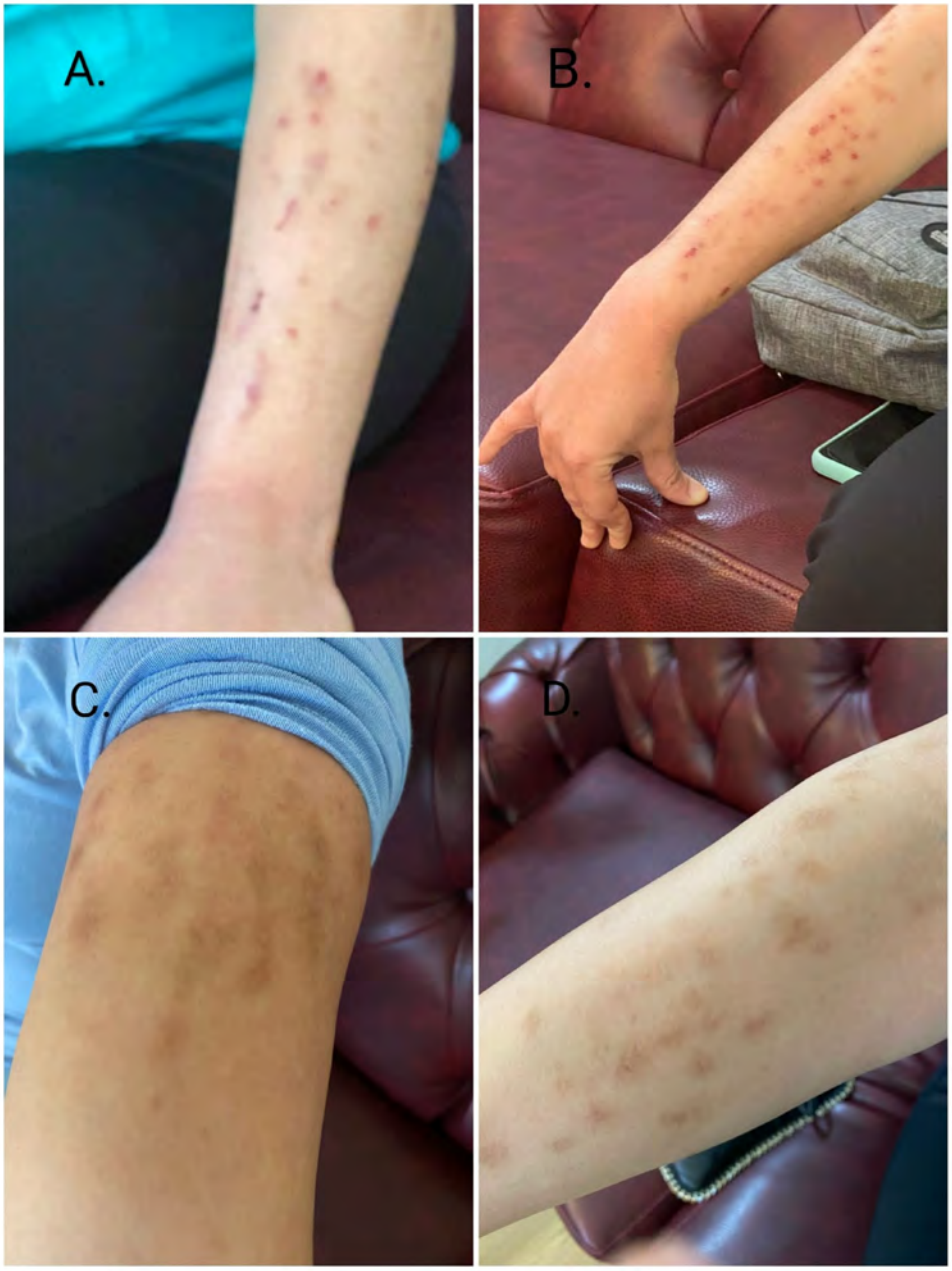

Figure 1. (A) and (B): The pre-treatment skin lesions of the patient. (C) and (D): post-treatment state of the patient, resolution of cutaneous lesions is prominent.

Psychiatric Examination

The patient reported a bad social life, insomnia, and the absence of any hobbies. She complained of a lack of sexual desire towards her partner and referred the onset of her symptomatology to her worsening relationship with her husband. Thereafter, her mood had changed into sadness, sorrow, and anger. The psychiatric clinical interview also revealed that the patient was convinced of being infested with scabies. This statement led the clinician to consider a possible psychosis or cocaine addiction; yet further investigation disproved these hypotheses. Standardized psychiatric examinations; Yale-Brown Obsessive-Compulsive Scale (Y-BOCS),[8] Hamilton Depression Rating Scale-17 (HDRS-17),[9] Hamilton Anxiety Rating Scale (HARS),[10] indicated the presence of depressive, anxious, and compulsive symptomatology along with considerable somatic symptoms. The total Y-BOCS score was 20 and all the symptoms were compulsions. The total HDRS score was 36, including somatic:18, and psychic:15 symptoms. The total HARS

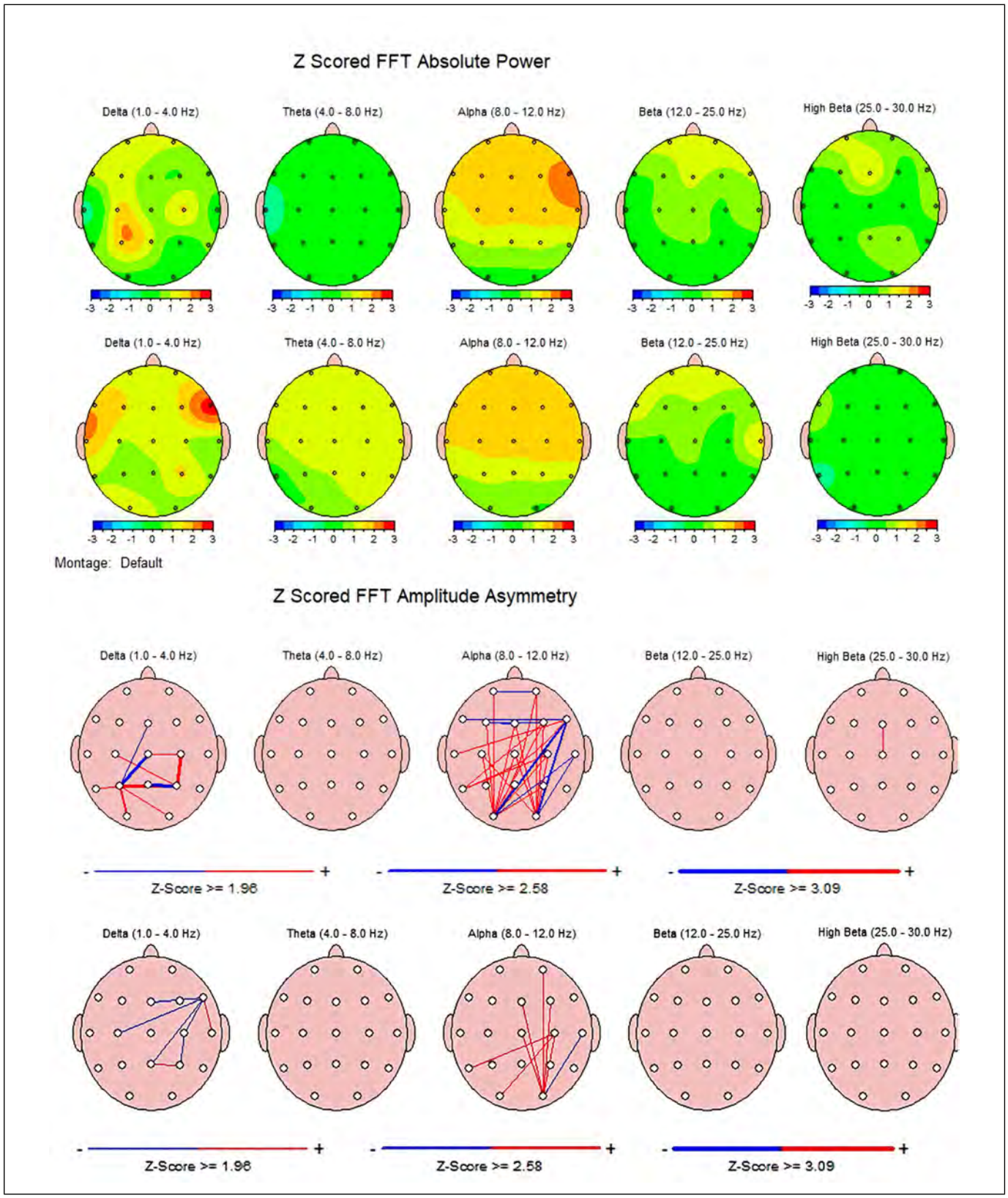

Figure 2. Shows the average Z-score maps of absolute power and amplitude asymmetry for the delta, theta, alpha, beta and high beta frequency bands obtained from pre-treatment (first rows of absolute power and amplitude asymmetry sections) and post-treatment (second rows of each section) qEEG recordings. Color bars for absolute power and amplitude asymmetry represent Z-scores relative to the normal population ranging from −3 to +3 standard deviation.

score was 33, including agitated:9, anxiety:12, physical:6, somatic:12, retarded:10. The diagnosis of SPD and OCD was made according to the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5).[10] Electrophysiological evaluation was also performed by quantitative electroencephalography. The details of qEEG recordings were elsewhere.[11] Based on her pre-treatment qEEG data, salient frontal alpha power, and amplitude asymmetry in alpha frequency were observed (Figure 2).

Treatment

The patient was started one-month paroxetine monotherapy. She was drug-free before treatment. Paroxetine dose (titrated from 5 to 40 mg/day), was doubled each week, reaching a double oral dose in the morning.

Second Examination

After one month of paroxetine therapy, psychiatric and electrophysiological examinations were repeated. Both physiological and psychological improvement was evident. The lesions were completely regressed leaving solely post-inflammatory hyperpigmentation (Figure 1C and D). All the clinical rating scores, ie, Y-BOCS, HDRS-17, HARS, reduced to zero. As for qEEG, excess frontal alpha activity decreased, and amplitude asymmetry disappeared (Figure 2).

Discussion

To date, there is no FDA-approved treatment of SPD.[1] Behavioral treatments such as habit reversal therapy and cognitive behavioral therapy are considered as the treatment of choice in SPD. Regarding pharmacological therapies, SSRIs, SNRIs, antipsychotics, and glutamatergic agents have most commonly been studied. SSRIs have shown variable success for the treatment of SPD. In a randomized controlled trial of fluoxetine with a mean dosage of 55 mg/day authors have reported improvement in only one of three outcome measures.[12] Another small open-label study with fluoxetine revealed that 8 of 15 subjects responded to therapy.[13] A larger randomized controlled trial involving 45 patients revealed no significant difference in skin picking severity with citalopram 20 mg/day compared to placebo.[14] In an open-label trial of escitalopram, a complete response rate of 44.8% was reported.[15] Another uncontrolled study reported significant improvement of skin findings in all of 14 patients treated with fluvoxamine.[16] An open-label study using sertraline revealed a clinical improvement in 19 of 28 patients.[17] There is no clinical trial of paroxetine in SPD; however, case reports of successful treatment do exist.[18,19]

As for biomarkers of paroxetine treatment response, studies indicate that excess relative alpha power[20,21] sourced in corpus striatum, orbito-frontal and temporo-frontal regions was observed in OCD patients who would respond to paroxetine treatment. In addition, the normalization of excess alpha power after successful treatment for these OCD patients was reported.[22] The treatment response of our SPD case diagnosed with one of obsessive-compulsive related disorder was in line with these qEEG studies. The rapid resolution of long-lasting cutaneous findings following paroxetine treatment and the disappearance of asymmetrical amplitudes in post-treatment EEG objectively supports the efficacy of pharmacological treatment in our patient. We emphasize the importance of the identification and treatment of underlying psychopathology in SPD patients.

As is seen, there are several therapy choices in the pharmacological treatment of SPD. Despite the effectiveness of SSRIs, the clinician might be confused over which SSRI to choose. Besides, the selection of optimum treatment agent is timesaving and ethically important. In this regard, we propose an algorithm that might be useful to clinicians. We suggest that if a subject with this dermatological illness has the following characteristics; 1. If obsessive-compulsive, depressive and anxious symptoms exist altogether, 2. If the biological markers of electrophysiological signs, particularly, increased frontal alpha activity and amplitude asymmetry are observed; then the treatment success rate using paroxetine will be very high. Thus, a trial of high-dose paroxetine therapy is warranted.

We suggest that the same parameters, ie, Y-BOCS, HDRS, HARS, and qEEG recordings should also be used for follow-up studies. As is the case in our observation, the efficacy of paroxetine in SPD could be objectively evaluated when all parameters are normalized within a month after paroxetine treatment in a large SPD population.

Declaration of Conflicting Interests

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Ethical Approval

Not applicable, because this article does not contain any studies with human or animal subjects.

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