Successful Treatment of Delusions of Parasitosis With Olanzapine

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Background: Delusional parasitosis is a rare disorder in which patients have a fixed, false belief of being infested with parasites. It is often accompanied by a refusal to seek psychiatric care. Delusions of parasitosis is classically treated with typical antipsychotic agents, the traditional dermatologic choice being pimozide. However, pimozide’s adverse effect profile and the need for frequent electrocardiographic monitoring make such treatment less practical.

Observation: We describe 3 patients who were diagnosed as having delusional parasitosis that was successfully treated with a recently Food and Drug Administration–approved atypical antipsychotic agent, olanzapine (5 mg/d). Olanzapine has a more benign adverse effect profile than typical antipsychotic agents and eliminates the need for electrocardiographic monitoring. Olanzapine therapy has been associated with such adverse effects as sedation, hyperlipidemia, weight gain, and insulin resistance, all of which were infrequent in our patients.

Conclusion: Olanzapine is an atypical antipsychotic agent that can be used as a first-line agent in delusional parasitosis as a safer therapeutic option without a specialized monitoring regimen.

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In delusional parasitosis (DP), patients have a fixed, false belief that they are infested with parasites. This relatively rare psychiatric disorder most often presents as a monosymptomatic hypochondriacal psychosis, in which no other thought disorders exist and delusions are not secondary to an additional psychiatric illness. It is essential that dermatologists are well versed in diagnosing and treating this psychiatric disorder, as the delusional nature of the disease is often accompanied by a refusal to seek psychiatric care.

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In the dermatology literature, DP has classically been treated with the antipsychotic pimozide. However, pimozide, like other typical or classic antipsychotic medications, is associated with extrapyramidal adverse effects, including irreversible tardive dyskinesia, which can occur with long-term use. Also, pimozide therapy can cause a prolonged QT interval, requiring baseline and periodic electrocardiographic monitoring. A safer therapeutic option without specialized monitoring requirements is needed.

REPORT OF CASES

CASE 1

A 53-year-old male chemical engineer presented with a 5-month history of a pruritic rash that was characterized by pink patches with excoriations on his upper and lower extremities. His rash began after he cleaned out the house of his recently deceased mother. He stated that the house smelled “damp” and that there was a black mold in the basement. His rash began after he cleaned the house of his recently deceased mother. He stated that the house smelled “damp” and that there was a black mold in the basement. About 2 weeks after cleaning, he became increasingly itchy over his upper extremities and trunk. He persisted in cleaning his body to rid himself of any suspected exposure. He believed that he had inhaled “mold spores” that, after having infected his entire body, were causing the pruritus. He subsequently removed some of his skin with a
Olanzapine therapy (5 mg/d) was initiated, and the patient was increasingly frustrated and unable to sleep as a result of her recalcitrant condition. She brought in the pea (Figure 1), which she believed was the “bug” that was causing the itching sensation along the posterior aspect of her hairline.

His treatment regimen consisted of clobetasol propionate ointment for his hands, 0.1% triamcinolone acetonide ointment to be applied twice a day to his thighs and abdomen, and oral olanzapine (5 mg/d). He was given a listing of local psychiatrists, and one particular doctor was recommended to him. At the 1-month follow-up visit, his dermatitis improved, but he continued to believe that he had a fungal infection. His wife stated that he had purchased a “Hazmat suit” for the purpose of sterilizing their house, at a cost equivalent to their monthly income. The dosage of olanzapine therapy was increased to 10 mg/d, and a psychiatric referral was made, which the patient did not pursue. He did not like the idea of the stigmata that are often associated with psychiatric referral. At the 3-month follow-up visit, the rash had cleared, and the patient no longer voiced any concern about fungal infection. He only asked about treatment for a plantar wart.

**CASE 2**

A 56-year-old healthy Hispanic woman with a several-year history of depression complained of intense pruritus on the posterior aspect of her neck that had been present for approximately 2 months. She described the sensation as “bugs crawling.” Her itching was recalcitrant to treatment with several topical steroids and antipruritic agents. Her medications included paroxetine for depression.

Physical examination revealed a localized plaque of excoriated, crusted papules and nodules at the nape of her neck. The findings of microscopic examination of skin scrapings using a potassium hydroxide mount were negative for organisms, as were those of a scabies preparation. Impetiginized prurigo nodularis with delusions of parasitosis was diagnosed.

Intralesional triamcinolone acetonide was administered to the largest nodules, and the patient was told to apply clobetasol cream to the nape of her neck twice daily and to begin a 10-day course of oral cephalaxin therapy. At a follow-up visit, she was tearful and stated that she was increasingly frustrated and unable to sleep as a result of her recalcitrant condition. She brought in the “bugs” that she had found along the posterior hairline area, and they were found to be dried peas (Figure 1). Olanzapine therapy (5 mg/d) was initiated, and the paroxetine therapy was discontinued because her primary problem was not depression. At her 1-month follow-up visit, she showed dramatic improvement, with resolution of all symptoms. She no longer had any delusions of parasitosis. She left for Puerto Rico to visit her mother soon afterward, a trip she had delayed for several months owing to her condition.

**CASE 3**

A 54-year-old man with a history of work-related back injury presented with a more than 1-year conviction that “larva are being injected into me by insurance auditors.” He explained that these auditors would sneak into his house at night and hold him down as they injected larva into his skin and sinuses because he was receiving workers’ compensation. He would subsequently attempt to kill the larva by burning them with cigarettes or pulling them out with tweezers, which he physically demonstrated during the interview. He claimed that the larva matured into flies, which then flew away. He was taking no medications.

Physical examination revealed several erosions with a honey-colored crust and scars on his face. His hands had several deep, irregularly shaped, crusted ulcerations (Figure 2). His ulcers and impetiginization were treated with cephalaxin and mupirocin ointment. His delusions of parasitosis were treated with olanzapine (5 mg/d) and psychiatric referral. He did not return to our clinic because of the significant distance he had to travel. Follow-up telephone conversations with his local psychiatrist revealed that he had markedly improved, with only a few excoriations remaining. When contacted by telephone he spoke in a rational manner, never mentioning any infestation. He stated that his sores were healing and that he wanted a prescription for more olanzapine, which he agreed to obtain from his psychiatrist.
Typical antipsychotic agents block both mesolimbic and striatal dopamine receptors, causing extrapyramidal adverse effects at therapeutic doses.4 The newer atypical antipsychotics have a more benign adverse effect profile, perhaps owing to decreased occupancy of striatal dopamine receptors.5 The atypical antipsychotic olanzapine was therefore considered as an alternative therapeutic approach to pimozide. In the 3 patients described herein, treatment consisted of olanzapine therapy started at a dosage of 5 to 10 mg/d.

In the literature, DP can also be referred to as non-symptomatic hypochondriacal psychosis, psychogenic parasitosis, or Ekbom syndrome, which was named after the psychiatrist who summarized the first 22 case reports in 1938.6 Patients often present with specimens of “parasites” they have collected, the so-called matchbox sign, which is considered by some authors to be pathognomonic.7 In some cases, the delusion is shared by a significant other, and this is termed a folie a deux.7 Patients may resort to self-mutilation in an effort to rid themselves of parasites, and insomnia is a common complaint.8 Several steps are useful in approaching patients with DP.8 A true infestation must first be ruled out. Wet preparations of skin scrapings and skin biopsy can be useful and can help the physician establish trust with the patient. Empathetic listening, expressing concern about how the problem is affecting the patient’s life, and establishing rapport are also helpful in preventing “doctor hopping.”8

It is also important to rule out any systemic disorder or unrecognized cutaneous disease. Careful history taking can help establish whether the delusions might be associated with alcoholism or drug addiction. Both cocaine and amphetamine abuse have been associated with the sensation of formication. Kidney, liver, thyroid, and endocrine abnormalities and lymphomas may cause secondary generalized pruritus. It is essential to distinguish a shakable belief of infestation from an unshakable belief; during discussion, some patients are receptive to testing results that reveal no active infestation.8 A psychiatric referral is useful, but not always accepted by patients,8 who may perceive an associated stigma. Likewise, managed care has limited the availability of psychiatric care for many patients.

Patients with shakable beliefs of infestation are not truly delusional. Nondelusional patients with depression can be effectively treated with selective serotonin reuptake inhibitors, tricyclic antidepressants, and electroconvulsive therapy.10-13 Alternatively, patients with a fixed false belief of infestation are best treated with antipsychotic agents. Pimozide was the first antipsychotic drug that was broadly used to treat DP, and a meta-analysis of 1223 case reports demonstrated a full remission rate of 50% with pimozide treatment, compared with a 30% remission rate in patients treated before pimozide was used.14 These data caused pimozide therapy to become the standard of care for patients with DP. Like many older neuroleptic drugs, pimozide has adverse effects, including parkinsonism, tardive dyskinesia, and neuroleptic malignant syndrome (fever, muscle rigidity, confusion, and dysrhythmias). Electrocardiographic changes have also been observed; therefore, baseline and serial electrocardiograms are required for patients using pimozide.

Atypical antipsychotic agents have been suggested as potential alternatives for DP treatment owing to their more favorable benefit-risk ratio.15 A limited number of case reports have documented the effectiveness of risperidone,16,17 sulpiride,18 and sertindole19 in the treatment of DP. To our knowledge, this series of 3 case reports represents the first documentation of olanzapine’s effectiveness in treating DP in the English-language dermatology literature. Previous reports either have been single case reports in German20,21 or have been published in the psychiatry literature and have used the term non-symptomatic hypochondriacal psychosis, which is not as well recognized by dermatologists.22,23 Olanzapine, which was introduced by Eli Lilly in 1997, acts as a potent antagonist at dopaminergic and serotonergic receptors, with weaker antagonism at α-adrenergic and muscarinic receptors.24 Olanzapine shows selectivity for mesolimbic and mesocortical over striatal dopamine tracts, thereby minimizing extrapyramidal adverse effects.25

Atypical antipsychotic agents can cause adverse effects, but the effects are considered less severe than those associated with typical antipsychotic agents. Olanzapine use has been associated with sedation, hyperlipidemia, weight gain, and insulin-resistant diabetes.26,27 Patients should be educated regarding these potential adverse effects. Fasting blood glucose and lipid determinations can be used to monitor for adverse effects, especially in the presence of additional risk factors such as obesity, advancing age, hypertension, a family history of diabetes, and a low level of physical activity. Although the incidence of tardive dyskinesia and neuroleptic malignant syndrome is considerably less than with the use of traditional antipsychotic agents, at high doses the possibility of their occurrence should be taken into consideration.28 Evidence is growing that monitoring the blood levels of atypical antipsychotics in patients may improve efficacy and safety; however, further investigation is necessary before therapeutic drug monitoring can be implemented.29

At present, olanzapine is prescribed predominantly by psychiatrists for patients with schizophrenia at a dosage of 20 mg/d. However, there are reports of success at a...
dosage as low as 1.25 mg/d in cases of mild DP. We suggest initiating therapy at a dosage of 2.5 to 5 mg/d and then seeing the patient in 6 weeks. If there is little improvement, the dosage is increased. In our experience of treating several other patients with DP, no more than 10 mg/d is usually necessary for monosymptomatic hypochondriacal psychosis.

Because patients are often hesitant to try a psychiatric medication, some encouragement may be needed. For example, we often state that there is “no evidence of infestation today,” which establishes no confirmation of the delusion but also demonstrates to the patients that you trust them. Then, we suggest that although olanzapine is a psychiatric medication, patients with similar symptoms have had remarkable improvement. In summary, our experience suggests that olanzapine should be considered as first-line therapy for DP.

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