

# Letter to the Editor

# Depression and Psoriasis Comorbidity. Treatment with Paroxetine: Two Case Reports

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#### TO THE EDITOR:

Two cases of major depression (DSM-IV) associated with psoriasis (clinical and laboratorial) were treated with paroxetine. Depression remitted and psoriasis achieved a remarkable improvement or remitted during follow-up. Despite the extensive clinical use of SSRIs, to our knowledge there is only one small open label study investigating the use of bupropion SR for the treatment of psoriasis and atopic dermatitis in patients where treatment failed with conventional medications (1). Therefore, this report describes the paroxetine induced improvement of two female patients suffering from psoriasis and depression.

# CASE 1

Ms. A., a 66-year-old retired widow, has a 36-year history of psoriasis. Her psoriatic plaques started on her elbows and rapidly spread to her knees and bilateral calves. During these years Ms. A. was treated with topical hydrocortisone and experienced mild to moderate improvement. However, during her vacations, usually in sunny places, significant dermatological improvement was achieved. The reduction of affected areas lasted no longer than 10 to 20 days after vacations ended.

During the last 3 years she devoted herself to taking care of her husband due to devastating cerebral cancer. Ms. A's depression symptoms began 7 months after the death of her

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husband. A major depressive disorder was characterized by depressive mood, frequent crying, psychomotor retardation, lack of interest in daily activities, decreased ability to think and concentrate, severe insomnia, constant worrying, and irritability. She started pharmacotherapy with 10 mg of paroxetine a day for the first two weeks and 20 mg thereafter. Depressive symptoms remitted from the 6th to the 8th week, allowing Ms. A. to readapt rapidly to her normal activities. Surprisingly, six months later, along with her mood improvement, Ms. A. experienced a complete psoriasis recovery with no subsequent attack during a follow-up of a year and a half while continuing paroxetine use. This was the longest psoriasis-free period she had ever had during her 36-year clinical history.

#### CASE 2

Ms. B, a 54-year-old married woman, with a 4-year history of chronic remitting dermatological disease, received a diagnosis of either unspecified or atopic dermatitis. Psoriasis was finally confirmed one and a half years ago through histopatological examination. Skin plaques developed around the abdomen, breasts, arms, and armpits. Treatment included topical hydrocortisone and antibiotics when infections occurred. In addition, the patient went through several unsuccessful treatment regimens such as homeopathy, vitamins, orthomolecular therapy, and acupuncture. Her skin problems hardly improved and severely worsened in winter. One and a half years ago, in winter, her preexisting psoriasis spread throughout her body. She was treated with topical and systemic corticosteroids and refused to undergo ultraviolet A light therapy. Treatment reduced the affected body surface to her preexisting condition after 4 weeks.

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As Ms. B's skin disease improved, a major depression began, characterized by depressive mood, severe insomnia, lack of interest, irritability, impaired concentration, loss of appetite, weight loss (10% of total body weight in six months), fatigue, anxiety (both worsening in the morning), and serious impairment of social functioning. Her physician prescribed fluoxetine 20 mg/day with no clinical improvement after three months. Referred to the psychiatric service, paroxetine was introduced and kept at 30 mg/day. After 6 weeks, the major depression had remitted and her psoriasis plaques completely disappeared. During follow-up (one year) neither depression nor psoriasis relapsed.

The literature includes a few reports of psoriasis induced or exacerbated by SSRIs therapy, mainly fluoxetine and paroxetine (2,3). These two cases provide important evidence of paroxetine's effectiveness in treating both conditions. In addition, they show no evidence of harm. Modell and coworkers mentioned "that normalization by bupropion, of potential causative

neuroendocrine, immunologic or catecholaminergic abnormalities in both of these dermatological disorders is a possible mechanism of action for the observed salutary effects of this drug ..." (1). These cases illustrate the importance of further investigating the causes underlying dermatological problems and depression as well as treatment approaches in comorbidity.

### REFERENCES

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