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A CASE STUDY ON SERTRALINE INDUCED PSORIASIS

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ABSTRACT
This case report describes a female diabetic patient presenting with chronic dermatological manifestations. Mrs. X, with a history of poorly controlled diabetes mellitus (DM) and sleep disorders, reported persistent erythema, pruritus, and scaly patches on her thighs and hands. Physical examination revealed a thin body habitus and eczematous, scaly lesions on the extremities. Laboratory findings showed significant hyperglycemia (random blood sugar: 320 mg/dL). The patient's medication regimen included metformin (500 mg), sertraline (85 mg), mirtazapine (7.5 mg), finasteride (0.5 mg), and an unspecified drug (Belavet, 8 mg), which may represent a reporting error. The clinical presentation raised differential diagnoses of diabetic dermopathy, fungal infections, or eczematous dermatitis, necessitating further diagnostic evaluation, such as skin scrapings for fungal microscopy or biopsy. Management focused on optimizing glycemic control, given the profound hyperglycemia, alongside empirical treatment with topical antifungals and emollients for symptom relief. A comprehensive medication review was recommended to assess potential drug- induced dermatological effects, particularly from psychotropic agents like sertraline and mirtazapine, which can contribute to dry skin and eczematous reactions. This case underscores the complex interplay between chronic hyperglycemia and skin pathology in diabetic patients. Poor glycemic control can impair skin barrier function, increase susceptibility to infections, and exacerbate inflammatory dermatoses. Additionally, the use of multiple medications, including psychotropics, may further complicate the clinical picture. Interdisciplinary collaboration among endocrinology, dermatology, and psychiatry is essential for holistic management.

KEYWORDS: Diabetes mellitus, diabetic dermopathy, fungal infection, hyperglycemia, psychotropic medications, interdisciplinary management.

INTRODUCTION

Sertraline is a medication belonging to the selective serotonin reuptake inhibitor (SSRI) class, commonly employed in the treatment of various psychiatric conditions. Its approved uses encompass major depressive disorder, generalized anxiety disorder, obsessive-compulsive disorder, post-traumatic stress disorder, and premenstrual dysphoric disorder. The drug's mechanism of action involves blocking the reuptake of the neurotransmitter serotonin into the presynaptic neuron. This elevated level of serotonin enhances neurotransmission and facilitates improved communication between brain cells, which is believed to contribute to the stabilization of mood and reduction of anxiety symptoms. Sertraline work's by increasing levels of a mood enhancing chemicals called serotonin in our brain.[1] It works by selectively blocking serotonin reuptake in the brain, thereby increasing serotonin levels and improving neurotransmission, which alleviates symptoms of these conditions.[2]

- **Serotonin Reuptake Inhibition**
- **Primary Action:** Potent and selective inhibition of the serotonin transporter (SERT), preventing presynaptic serotonin (5-HT) reuptake, thereby increasing extracellular serotonin levels in synaptic clefts.[3]
- **Receptor Effects:** Elevated 5-HT enhances stimulation of postsynaptic receptors (5-HT_1A, 5-HT_2A), contributing to mood regulation and anxiolytic effects.[4]

2. **High Selectivity for SERT**

- **Low Affinity for NET/DAT:** Unlike older antidepressants (e.g., TCAs), sertraline has minimal interaction with norepinephrine (NET) or dopamine (DAT) transporters at therapeutic doses, reducing side effects (e.g., anticholinergic, cardiovascular).[5]

- **Dopaminergic Activity at High Doses:** Weak dopamine reuptake inhibition may occur at elevated doses, possibly contributing to mild psychostimulant effects in some patients.[6]

3. Neuroadaptive Changes with Chronic Use

- **Autoreceptors Desensitization:** Chronic use down regulates presynaptic 5-HT_1A autoreceptors, enhancing sustained serotonin release.[7]
- **Neuroplasticity:** May increase brain-derived neurotrophic factor (BDNF) and promote hippocampal neurogenesis, linked to long-term antidepressant efficacy.[8]

4. Pharmacokinetics & Metabolism

- **Absorption:** Well-absorbed orally, but bioavailability is ~44% due to significant first-pass metabolism.[9]
- **Metabolism:** Primarily hepatic, via CYP3A4, CYP2C19, and CYP2B6. Drug interactions are possible with CYP3A4 inhibitors (e.g., ketoconazole) or inducers (e.g., carbamazepine).[10]
- **Half-Life:** ~26 hours, permitting once-daily dosing.[11]
- **Active Metabolite:** Sertraline, a member of the selective serotonin reuptake inhibitor (SSRI) class of antidepressants, undergoes hepatic metabolism to form its primary metabolite, N-desmethylsertraline. This resulting compound remains pharmacologically active, though it exhibits less potency than the parent drug. A key pharmacokinetic distinction lies in their half-lives; while sertraline itself has a half-life of roughly 24 to 26 hours, the N-desmethylsertraline metabolite possesses a substantially prolonged half-life that can range from 62 to 104 hours. This pronounced difference is a critical factor in the drug's clinical profile, as the persistent presence of the active metabolite contributes to a sustained therapeutic effect and plays a significant role in mitigating the severity of acute withdrawal symptoms following the cessation of treatment.[12]

RELATIONSHIP BETWEEN SERTRALINE AND PSORIASIS PATIENT'S

Psoriasis is a chronic immune-mediated skin disorder affecting approximately 2-3% of adults worldwide, marked by accelerated keratinocyte growth and abnormal immune activation through Th1 and Th17 pathways. The condition demonstrates significant psychiatric comorbidity, with research indicating that depression and anxiety disorders occur in up to 62% of psoriasis patients. This reciprocal connection arises from both the psychological impact of visible skin lesions and overlapping inflammatory mechanisms, where elevated levels of cytokines including TNF-α, IL- 6, and IL-17 contribute to both skin inflammation and mood dysregulation. Psychological stress intensifies this vicious cycle by disrupting HPA axis function and promoting inflammatory responses that trigger disease exacerbations.[13]

The serotonin system serves as a crucial link between these processes, as serotonin functions as both a neurotransmitter and a skin immunomodulator. In psoriatic lesions, increased expression of the serotonin transporter (SERT) has been observed, where it interacts with apoptosis-related proteins like caspase-3, suggesting involvement in inflammatory regulation. While SSRIs represent first-line treatment for depression in these patients, their dermatological effects remain inconsistent. Some individuals experience clinical improvement potentially through stress reduction and immunomodulation, while others report disease worsening possibly due to serotonin's proliferative effects on keratinocytes.[14]

Clinical observations demonstrate this variability, with certain SSRIs like sertraline showing beneficial effects in some studies but exacerbating symptoms in others. Sertraline in particular has been associated with psoriasis flares in case reports. This dichotomy highlights the need for careful monitoring when prescribing antidepressants to psoriasis patients. A collaborative approach between dermatologists and mental health professionals is recommended to balance psychiatric and dermatological outcomes. Further investigation into personalized treatment strategies and alternative antidepressant options with more predictable cutaneous effects would benefit this vulnerable patient population. Understanding the complex interplay between neuroendocrine factors and skin inflammation remains essential for optimizing care.[14,15]

POTENTIAL EXACERBATION OF PSORIASIS BY SERTRALINE

1. Serotonin-Mediated Immune Activation

Serotonin (5-HT) plays a role in both neuropsychiatric and immune functions. While most serotonin is produced in the gut, SSRIs increase extracellular serotonin levels in the central nervous system and periphery. Elevated serotonin may stimulate pro-inflammatory cytokines, including tumour psoriasis factor-alpha (TNF-α), interleukin-6 (IL-6), and IL-17, which are key drivers of psoriatic inflammation. A study in Clinical and Experimental Dermatology found that serotonin can modulate T-cell activity, potentially exacerbating Th17-mediated autoimmune responses seen in psoriasis.[16]

2. Case Reports of Drug-Induced Psoriasis

There have been documented cases where psoriasis developed or worsened shortly after initiating sertraline. A report in Psychosomatics described a patient who developed guttate psoriasis within weeks of starting sertraline, with lesions resolving after discontinuation. Another case in Dermatology and Therapy highlighted a patient with stable plaque psoriasis who experienced a severe flare after sertraline use, suggesting a possible drug-triggered Koebner phenomenon.[17]

3. SSRIs and Cytokine Dysregulation

A study that analysed antidepressant use in psoriasis patients and found that while SSRIs were not a major trigger, individual susceptibility played a role. Some researchers hypothesize that SSRIs may alter cytokine balance, leading to paradoxical inflammation in predisposed individuals.[18,19]

POTENTIAL BENEFITS OF SERTRALINE IN PSORIASIS PATIENTS

Psoriasis, a chronic inflammatory skin disorder, is often exacerbated by psychological stress due to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and elevated stress hormones like cortisol. Emerging evidence suggests that sertraline, a selective serotonin reuptake inhibitor (SSRI), may benefit psoriasis patients through multiple pathways.

➢ Neuroimmunological Pathways

The stress-psoriasis connection operates through complex neuroendocrine mechanisms. Chronic stress activates the hypothalamic-pituitary-adrenal axis, leading to elevated cortisol and catecholamine levels that can exacerbate cutaneous inflammation.[20] Research suggests that effective treatment of mood disorders in patients with psoriasis may lead to better dermatologic outcomes, highlighting the potential benefit of integrated psychotropic therapy. Studies have observed bidirectional links between psychological stress, depression, and psoriasis exacerbation, possibly mediated by Neuroimmunological mechanisms.[21]

➢ Anti-Inflammatory Properties

Beyond its neurological effects, sertraline demonstrates immunomodulatory capabilities. Experimental models reveal that this SSRI can down regulate pro-inflammatory cytokines, particularly those involved in psoriatic plaque formation.[22] These findings, while preliminary, suggest a possible direct pharmacological effect on disease activity.

➢ Treatment Adherence Enhancement

The significant prevalence of depression among psoriasis patients presents a major barrier to effective treatment. Studies demonstrate that untreated psychiatric comorbidities substantially reduce compliance with dermatologic therapies.[23] By addressing these mental health concerns, sertraline may indirectly improve treatment outcomes through enhanced adherence.

➢ Potential Dermatotropic Effects

Emerging laboratory evidence indicates that certain SSRIs may influence keratinocyte biology, though the clinical relevance for psoriasis remains theoretical.[24] This area requires further investigation to determine if these observations translate to therapeutic benefits.

HOW SERTRALINE WORSENING THE PSORIASIS PATIENTS

Sertraline, a popular SSRI (selective serotonin reuptake inhibitor) prescribed for depression and anxiety disorders, may unexpectedly worsen psoriasis in some patients due to its effects on immune responses, nerve signaling, and drug interactions. While generally safe, studies suggest that this medication could trigger or intensify psoriasis flare-ups in susceptible individuals. Doctors treating patients with co-existing psychiatric and skin conditions should be aware of this potential side effect to optimize treatment plans and avoid complications. A deeper understanding of these interactions can help balance mental health benefits with dermatological risks.

❖ Immune System Modulation

SSRIs like sertraline can affect the immune system, potentially impacting the inflammatory pathways involved in psoriasis. Some studies suggest that SSRIs may augment the Th-2 pathway, which is implicated in atopic dermatitis, and could potentially worsen psoriasis.[25]

❖ Potential for Flare-Ups

While rare, some individuals have experienced psoriasis flares or the onset of psoriasis while taking sertraline or other SSRIs. This may be due to individual variations in how the medication affects the immune system or other unknown factors.[26]

❖ Psychological Stress and Psoriasis

Psoriasis can be influenced by psychological stress, and antidepressants like sertraline are used to manage stress and depression. However, some studies suggest that psychological stress can also elevate inflammatory markers, which could potentially worsen psoriasis in susceptible individuals.[27]

❖ Serotonin-Mediated Immune Dysregulation

Sertraline increases synaptic serotonin (5-HT) levels, which interacts with immune cells, potentially worsening psoriasis. Serotonin receptors (e.g., 5-HT2A) are expressed on keratinocytes, T-cells, and dendritic cells, and their activation can promote pro-inflammatory cytokine release, including IL-6, TNF-α, and IFN-γ. These cytokines are central to psoriasis pathogenesis, driving keratinocyte hyperproliferation and inflammatory plaque formation. A study in Experimental Dermatology found that serotonin enhances Th17 responses, a key pathway in psoriasis. Additionally, reported that SSRIs may modulate mast cell degranulation, leading to histamine release and worsening pruritus and skin inflammation in psoriasis patients.[28]

❖ Stress and Paradoxical Flares

Although sertraline is prescribed to alleviate anxiety and depression, initial SSRI use can transiently increase stress due to adjustment effects, potentially triggering psoriasis flares via the hypothalamic-pituitary-adrenal (HPA) axis. Psychological stress elevates corticotropin-releasing hormone (CRH) and substance P, both of which exacerbate psoriasis by promoting neurogenic

inflammation. A case report in the Journal of Clinical Psychopharmacology described a patient whose psoriasis worsened within weeks of starting sertraline, possibly due to early-treatment anxiety before therapeutic effects stabilized.[29,30]

❖ **Drug-Induced Psoriasiform Eruptions**

SSRIs, including sertraline, have been linked to drug-induced psoriasis or psoriasiform dermatitis. A review in Dermatology and Therapy identified several cases where SSRIs triggered new-onset psoriasis or flare-ups in existing disease. The mechanism may involve T-cell activation or type I interferon responses, similar to other drug-induced psoriatic reactions. Additionally, sertraline's xerotic effects can worsen skin barrier dysfunction, increasing scaling and irritation in psoriatic plaques.[31,32]

PATHOPHYSIOLOGY OF SERTRALINE INDUCED PSORIASIS PATIENTS

Sertraline, a widely prescribed selective serotonin reuptake inhibitor (SSRI), may influence immune responses and inflammatory skin conditions such as psoriasis through several biological mechanisms.

1. Immune System Interactions

SSRIs can modulate immune activity by affecting cytokine production. Since cytokines regulate inflammatory pathways, alterations in their balance—particularly increases in pro-inflammatory cytokines like TNF-α, IL-17, and IL-23—could contribute to psoriasis development or flare-ups. Serotonin, whose reuptake is inhibited by sertraline, also plays a role in immune regulation, suggesting a potential link between SSRI use and skin inflammation.

2. Skin as a Serotonin-Responsive Organ

The skin contains its own serotonergic system, which helps maintain barrier function and immune homeostasis. Disruptions in serotonin signaling due to SSRIs may interfere with these processes, possibly increasing susceptibility to inflammatory skin disorders like psoriasis.

3. Possible Pathways Connecting SSRIs and Psoriasis

- **Cytokine Shifts:** SSRIs may skew cytokine profiles toward inflammation, worsening psoriasis.
- **T-Cell Activation:** These drugs might enhance Th17 cell activity, which drives psoriatic plaque formation.
- **Microbiome Effects:** Indirect changes to skin bacteria due to serotonin alterations could influence immune responses.

4. Conflicting Clinical Observations

While some research suggests SSRIs could help psoriasis by reducing stress (a known trigger), isolated case reports describe psoriasis onset or worsening after starting sertraline. This discrepancy highlights the need for individualized patient monitoring.

5. Key Considerations for Treatment

Since psoriasis arises from genetic, environmental, and immunological factors, sertraline's role if any likely varies by patient. Those with psoriasis or a family history should be observed for skin changes when initiating SSRIs.

INTRODUCTION

This case study examines the medical history, presentation, and management of Mrs. X, who presented with dermatological and systemic complaints. Her condition highlights the challenges of managing psoriasis in the context of multiple comorbidities, including sleep disorders and metabolic abnormalities. The study aims to provide a comprehensive overview of her clinical journey, emphasizing the interplay between her symptoms, past medical history, and treatment plan.

PATIENT PROFILE & CHIEF COMPLAINTS

Mrs. X, whose detailed demographic information was not fully documented, reported to the clinic with persistent itching, redness, and scaly patches in the thigh region and hands. These symptoms had been present for an extended period, significantly affecting her quality of life. The description of her lesions was consistent with psoriasis, a chronic autoimmune skin disorder characterized by rapid skin cell proliferation leading to scaly, inflamed patches. Her chief complaints pointed toward a flare-up of this condition, necessitating further evaluation and intervention.

PAST MEDICAL HISTORY

Mrs. X's medical history revealed a complex background of health issues. She had been diagnosed with a sleep disorder, the specifics of which were not detailed but could imply conditions such as insomnia or sleep apnea. These comorbidities are significant as they may contribute to her overall health burden and complicate the management of her primary dermatological condition.

MEDICATION HISTORY

Mrs. X's pharmacological regimen included several medications, indicating multiple underlying conditions:

T. Metformin: Commonly prescribed for type 2 diabetes mellitus, suggesting she had issues with glucose metabolism.

T. Sertraline: An antidepressant, indicating a history of depression or anxiety, which could also be linked to her sleep disorder.

T. Nitrate + 5 mg: Likely prescribed for cardiovascular issues, such as angina, pointing toward possible coronary artery disease.

T. Fonage P 0.5 mg: Possibly a misspelling of a medication like Finasteride or another drug, though its purpose remains unclear without further context.

T. Belavest & mg: Another medication with unclear specifics, possibly a brand name or typographical

error.

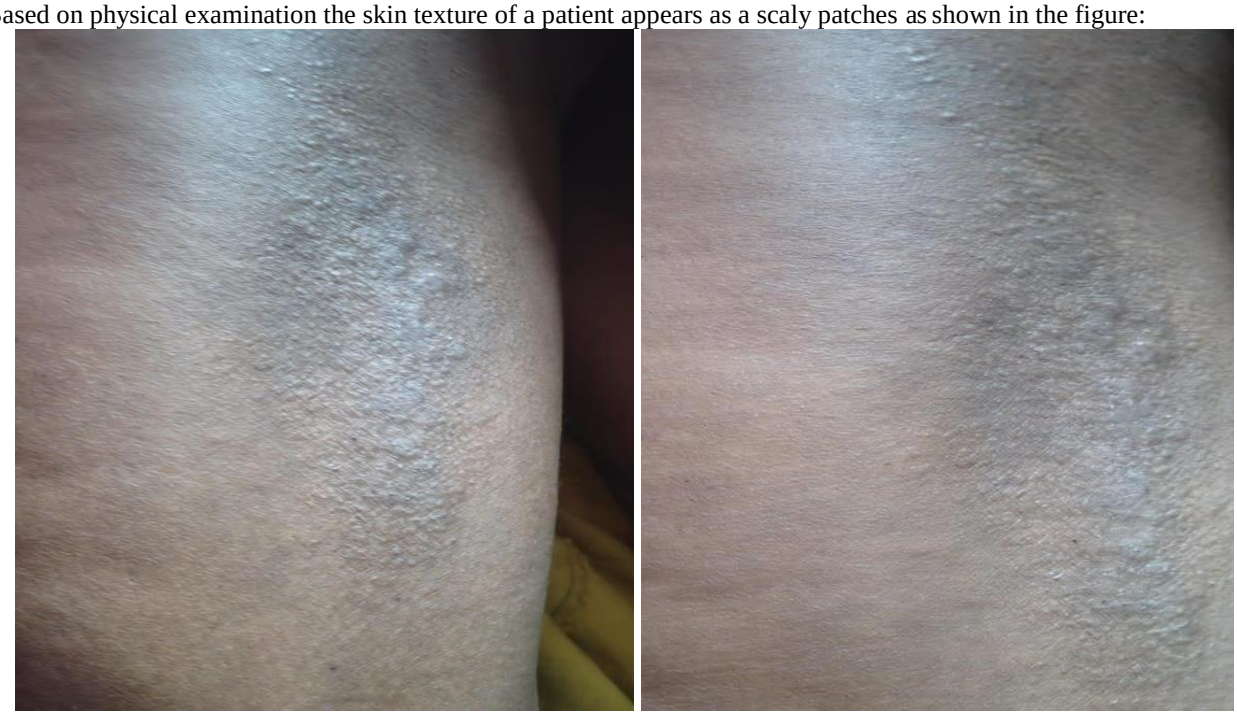
This polypharmacy underscores the complexity of her health status, with each medication addressing a different aspect of her comorbid conditions.

SYSTEMIC FINDINGS: The physical and systemic examination includes:

- **Hemoglobin (lb):**9.0 mg/dl, indicating anemia, which could explain symptoms like fatigue and exacerbate her psoriasis.
- **Random Blood Sugar (RBS):** 320 mg/dl, significantly elevated, confirming poorly controlled diabetes mellitus. This hyperglycemic state is noteworthy as it can worsen inflammatory conditions like psoriasis and complicate wound healing.

PHYSICAL EXAMINATION

Based on physical examination the skin texture of a patient appears as a scaly patches as shown in the figure:



DIAGNOSIS AND CLINICAL CORELATION

The primary diagnosis was psoriasis a condition often associated with systemic inflammation and linked to other autoimmune or metabolic disorders. The documentation also mentioned which might imply a consideration of sertraline-induced psoriasis or a religious/cultural aspect affecting her health beliefs. Psoriasis is known to have triggers, including stress and certain medications, and sertraline, while generally safe, can rarely exacerbate skin conditions in susceptible individuals.

The presence of anemia alongside poorly managed diabetes significantly worsened her condition. Anemia may have stemmed from chronic illness, lack of essential nutrients, or other hidden health issues, whereas uncontrolled diabetes likely indicated either insulin resistance or insufficient treatment both of which frequently occur in individuals with long-term inflammatory diseases.

TREATMENT PLAN: The treatment includes:

Topical Therapies: Corticosteroids or vitamin D analogues to reduce inflammation and scaling.

1. **Systemic Medications:** Depending on severity, drugs like methotrexate or biologics might be considered.
2. **Lifestyle Modifications:** Stress management, dietary adjustments to address diabetes and anemia, and weight control.
3. **Comorbidity Management:** Optimizing her diabetes control with metformin adjustment, addressing anemia with iron or B12 supplementation if deficient, and reviewing her psychiatric medication for potential alternatives if sertraline was suspected to aggravate her psoriasis.

DISCUSSION

Mrs. X's case illustrates the multifaceted nature of psoriasis, often intertwined with metabolic, psychological, and cardiovascular comorbidities. Her elevated RBS and anemia suggest poor metabolic control, which can perpetuate inflammation and skin manifestations. The mention of sertraline-induced psoriasis, if accurate, highlights the importance of medication review in managing chronic skin conditions.

CONCLUSION

Mrs. X's case underscores the need for a holistic approach in managing psoriasis, especially in patients

with multiple comorbidities. Her treatment should address not only the skin manifestations but also the underlying metabolic, hematological, and psychological conditions. Future follow-ups would benefit from detailed documentation, comprehensive investigations to clarify ambiguities (e.g., hip pathology, medication names), and a multidisciplinary team to optimize her care. This case serves as a reminder of the intricate connections between chronic inflammatory skin diseases and systemic health, necessitating tailored, patient-centered management strategies.

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