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Selection of psychotropics in dermatologic practice

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Review Article

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ABSTRACT

There is an increased prevalence of psychiatric symptoms in dermatologic disorders. However, these are often underrecognized and undertreated contributing to suboptimal adherence and therapeutic outcomes. A working knowledge of psychotropic medications and their use in dermatology is essential for comprehensive management of psychodermatological conditions. The present review provides a framework for use of psychotropic agents in dermatological settings and is intended to serve as a ready reckoner for the dermatologist. We initially review the general considerations involved in prescribing psychotropic agents in skin conditions. Next, we discuss individual classes of psychotropic agents such as anti-depressants, mood stabilizers, antipsychotics, and anxiolytics focusing on preferred agents while prescribing. Finally, we discuss the common adverse cutaneous reactions reported with psychotropic agents.

Keywords: Antipsychotics, Antidepressants, Dermatology, Psychodermatology, Adverse drug reaction

INTRODUCTION

A bidirectional relationship exists between psychiatric disorders and general medical explanations. Increased prevalence of psychiatric disorders has been noted in chronic medical conditions while patients with severe mental illness have increased rates of medical comorbidity compared to the general population. On a similar note, 25–30% of patients with chronic dermatological disorders concurrently have psychiatric comorbidities often influencing treatment outcomes.^[1] More specifically, psychological factors have been postulated to play a major role in the genesis of a range of dermatological conditions such as psoriasis, acne, alopecia areata, atopic dermatitis, vitiligo, and rosacea.^[2,3]

Several mechanisms may explain this link. Inflammation is a common substrate for the development of dermatological conditions and is increasingly recognized as being relevant to psychiatric conditions. There are complex physiological links between the skin and the brain. Stress is known to activate the hypothalamopituitary axis resulting in increased production of corticotropin-releasing hormones and glucocorticoids. These compounds play key roles in the "brain-skin axis," mediate interactions between stress response pathways and skin resulting in skin changes such as perturbations in epidermal permeability which are related to severity of manifestations in psoriasis.^[4] Finally, the process of psychosocial adjustment and emotional distress of contending with a cosmetically disfiguring or intensely symptomatic dermatologic disorder can predispose to a secondary psychiatric condition such as major depression,^[5] panic disorder, or social anxiety.^[6] There is also a category called "psychophysiological disorders"

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wherein the dermatologic symptoms can fluctuate in conformity with the mood and stress levels; well-known examples of such conditions are psychogenic urticaria, psoriasis, and acne.^[7,8]

In this review, we examine the use of psychotropic medications in patients with dermatologic disorders. Initially, we start with general considerations and then move on to discuss issues relevant to specific classes of psychotropic agents such as antidepressants and antipsychotics. Finally, we end with a discussion on adverse skin effects of psychotropic medications. The objective of the review is to provide evidence-based recommendations in selection and dosing psychotropics in the management of skin conditions for those working with such populations.

MATERIAL AND METHODS

Search strategy

Electronic search of MEDLINE through PubMed and Google Scholar databases was carried out in September 2021 to look for relevant articles published over the past 30 years. The following keywords were used to search "Dermatology/Psycho-dermatology" PubMed: AND ("Antidepressants" OR "Antipsychotics" OR "Mood stabilizers" OR "Anxiolytics"). We also searched the database using "cutaneous adverse drug reactions (CADRs)" AND "psychotropics." Searches were done independently by two qualified psychiatrists. In addition, hand searches of reference lists of included articles were done to identify relevant crossreferences.

Study selection

We screened the pool of generated articles initially based on their titles, abstracts, and, subsequently, their full texts, if necessary, to identify relevant studies. Since our aim was to provide evidence-based recommendations, we also included relevant systematic reviews and meta-analyses apart from original papers. Only English language articles published in peer reviewed journals were included in the study. Based on these criteria, a total of 92 articles were included in the present review. As this was not intended to be a systematic review, we neither computed effect estimates nor performed a quality assessment of included studies.

Data synthesis

Included studies were categorized under the following major headings: General considerations when prescribing psychotropic agents in dermatology, evidence for use of various classes of psychotropic agents such as antidepressants, mood stabilizers, antipsychotics, and anxiolytics in skin disorders, and literature on adverse skin reactions to psychotropic agents. Accordingly, the review is structured under these headings.

GENERAL CONSIDERATIONS IN PRESCRIBING PSYCHOTROPIC AGENTS IN DERMATOLOGY

Situations in which a working knowledge of psychotropics is required by a dermatologist include management of skin conditions associated with psychiatric disorders (such as pruritus in depression), management of psychiatric conditions in skin disorders (such as depression in psoriasis), and management of common adverse effects of psychotropic agents such as the anti-cholinergic effects of antidepressants and antipsychotics.^[1]

When to start and when to stop psychotropics in dermatologic settings are an important consideration as pointed out earlier. In certain primary psychiatric conditions such as delusional parasitosis, a dermatologist may be the only physician that the patient agrees to visit as such patients typically lack insight into their psychopathology and refuse psychiatry referral.^[9] Therefore, it is important that a dermatologist is sufficiently familiar with antipsychotics to prescribe them confidently in such cases. Physicians can use explanations such as a disorder with abnormal sensations which the antipsychotic will control to convince patients to take the antipsychotic medication. When stopping psychotropics, the general rule to be followed is to taper them over several weeks to months to pre-empt symptom relapse.

Diagnosis of major depression syndrome is an indication to start antidepressant treatment. However, if the patient is acutely suicidal or psychotic, then a prompt liaison must be made with a psychiatrist. Selective serotonin reuptake inhibitors (SSRIs) have supplanted tricyclic antidepressants (TCAs) as the preferred agents when treating depression because of their superior tolerability profile. Treatment can be initiated with dosages in the therapeutic range. A trial period of 4-6 weeks at adequate dosages must elapse before a decision is made on non-response; this also applies to antipsychotic treatment. The medication is usually continued for 6-12 months among those who respond to the medication.^[10] About a third of patients respond to the first antidepressant trial; next steps would include nonresponders being tried on sequential monotherapy with other antidepressant agents.^[11] Antidepressants and antipsychotics are comparable in terms of efficacy and selection of agents is usually based on adverse effect profiles and economic considerations.^[12,13]

Certain drug interactions, mainly relating to changes in functioning of hepatic microsomal enzymes, must be borne in mind when coprescribing psychotropic and dermatologic medications. Of these, the most important interactions relate to those involving CYP2D6 and CYP3A4 isoenzymes. Many of the antidepressants belonging to the SSRI group such as paroxetine and fluoxetine are potent inhibitors of CYP2D6 isoenzyme,^[14] thus, increasing the blood levels of drugs that are metabolized by this enzyme. Examples of CYP3A4 isoenzyme inhibitors among antidepressants include fluoxetine, fluvoxamine, and sertraline^[15] while agents that induce CYP3A4 include anticonvulsants such as carbamazepine used primarily as a mood stabilizer in psychiatry.^[16] The latter may induce metabolism of corticosteroids and immunomodulators such as cyclosporine used in the management of treatment-resistant psoriasis and atopic eczema. Likewise, antifungal agents used in dermatology such as ketoconazole and itraconazole are potent inhibitors of CYP3A4 and may raise blood levels of psychotropics such as carbamazepine and quetiapine which are primarily metabolized by CYP3A4 pathway.^[17] Table 1 summarizes the general considerations involved in use of different classes of psychotropic agents in dermatologic settings.

USE OF ANTIDEPRESSANTS IN DERMATOLOGY

Antidepressants are a group of medications that are beneficial in the treatment of different types of psychodermatological conditions, namely, primary psychiatric disorders (such as delusional parasitosis), secondary psychiatric disorders, and psychophysiological disorders; each of these is explained in subsections below.^[8]

Primary psychiatric disorders

These are conditions where there is an underlying primary psychiatric disorder resulting in secondary cutaneous lesions which are often self-induced. The first point of contact can frequently be a dermatologist in these disorders due to the prominent cutaneous manifestations.

Obsessive compulsive disorder (OCD)

OCD is a psychiatric disorder characterized by the presence of obsessions and compulsions. They can develop obsessions related to contamination and thereby frequent hand washing as a form of compulsion which can result in skin lesions over the palm when the symptoms become severe. SSRIs such as fluoxetine and sertraline can be beneficial in such cases. In certain cases, TCAs such as clomipramine are also used.

Trichotillomania (TTM)

This is a condition characterized by recurrent and difficult to control urges to pull out body hair, especially from the scalp and eyebrows. Chronic engagement in this behavior can result in cutaneous manifestations. Severe forms of the disorder can respond to SSRI, though the evidence is mixed. $\ensuremath{^{[18]}}$

Skin picking disorder (SPD)

This is a disorder where there is repetitive compulsive picking of the skin resulting in damage to it. This is also conceptualized as a form of obsessive-compulsive spectrum disorder and SSRIs have been found to be beneficial in certain cases.^[19]

Body dysmorphic disorder (BDD)

Patients with BDD tend to be preoccupied with a perceived flaw in their physical appearance which is a source of significant anxiety for them. They may present first to dermatologists due to lack of insight about the disorder. Such people may require treatment with SSRIs in case of inadequate response to first-line psychotherapy.^[20]

Secondary psychiatric disorders

These patients present with an underlying chronic dermatological condition and can develop secondary psychiatric conditions due to accompanying significant psychosocial adversities such as disfigurement and decreased self-esteem.^[8]

Depressive disorders and anxiety disorders

Chronic dermatological conditions can lead to depressive symptoms and anxiety symptoms in patients as a reactive phenomenon to the chronic condition. In mild severity of the condition, psychotherapies such as cognitive behavior therapy may be beneficial.^[21] In severe cases, the patients might benefit from antidepressant medication. Most commonly, SSRIs are preferred due to their favorable adverse effect profiles.

Psychophysiological disorders

The skin conditions which are either triggered or worsened by stress are included under this category. Conditions such as acne and hyperhidrosis have an established link with stress. Other skin disorders including eczema, urticaria, and psoriasis are also known to be worsened during stress.^[22] Identification of stress as a triggering agent is of utmost importance in the management of these conditions. Management of such cases should include cognitive behavior therapy and antidepressant medications such as SSRI.^[8]

Anti-inflammatory property of antidepressant medications

It has been postulated that systemic inflammation can be central to the pathogenesis of depression in certain

Table 1: General guidelines for using psychotropics in dermatology.			
Class of psychotropic agent	When to start and stop	Selection of agents	Important drug interactions
Antidepressants	Start when diagnosed with major depression and waiting for psychiatry referral Taper and stop after sufficient period of maintenance treatment following response and remission of depression	Based on adverse effect profile and cost considerations	SSRI such as paroxetine and fluvoxamine can inhibit CYP2D6 and fluoxetine inhibits CYP3A4. Blood levels of drugs metabolized by these enzymes may rise leading to increased risk of adverse effects
Antipsychotics	Start when psychotic symptoms (delusion and hallucinations) are elicited Taper and stop after use for minimum possible period to prevent risk of tardive dyskinesia	Based on adverse effect profile (risk of extrapyramidal symptoms)	Aripiprazole, quetiapine, ziprasidone, risperidone, and haloperidol are metabolized by CYP3A4. Their blood levels may be raised by antifungals such as ketoconazole which inhibit this isoenzyme
Mood stabilizers	Start for patients diagnosed with bipolar disorder. Decision to stop treatment may need to be discussed on a case-by-case basis with a psychiatrist	Avoid lithium in pre-existing psoriasis as it may exacerbate the condition. In such cases, prefer divalproex sodium or carbamazepine	Coadministration of lamotrigine with divalproex sodium, both mood stabilizers, can elevate the risk of skin rash, a known side effect of lamotrigine
Antianxiety	Start for indications such as short term (<4 weeks) relief of anxiety in dermatological conditions. Use minimum effective dose for shortest period of time possible	Selection of agents with long half-life (e.g.: Clonazepam) may avoid rebound symptoms. In those with liver disease, prefer lorazepam or oxazepam which require minimal liver metabolism	Avoid concurrent use of triazolam (CYP3A4 substrate) along with CYP3A4 inhibitors to avoid risk of adverse effects
SSRI: Selective serotonin reuptake inhibitor			

cases. Inflammatory conditions of the skin may have an impact on the inflammatory process in the brain. The antiinflammatory properties of antidepressants may be useful in the treatment of inflammatory conditions such as atopic dermatitis, psoriasis, and chronic urticaria. However, further research is required in this area.^[23]

Management of pain

Antidepressant medications have been effective for the treatment of pain caused by the dermatological conditions such as hidradenitis suppurativa.^[24] In addition to pain management, these medications can also benefit the patients due to their antidepressant and anxiolytic effects. The commonly used medications include the serotonin norepinephrine reuptake inhibitor (SNRI) group of antidepressants, namely, duloxetine and venlafaxine along with the TCA amitriptyline. These drugs are mainly effective in reducing neuropathic pain. Studies have shown that amitriptyline is effective in reduction of pain caused by post-herpetic neuralgia, peripheral neuropathy, and mixed neuropathy.[25] Box-1 below summarizes the important considerations from this section while prescribing antidepressants in dermatology while Table 2 summarizes dosing suggestions and monitoring guidelines for major antidepressant agents.[26]

- Anti-depressant medications are beneficial in the treatment of primary psychiatric disorders, secondary psychiatric disorders and psychophysiological disorders in dermatology
- SSRIs are often preferred as first line agents due to their relatively better adverse effect profile
- The anti-inflammatory property of antidepressants is being investigated such that it can be used as treatment option in inflammatory skin conditions such as atopic dermatitis and psoriasis
- SNRIs and TCAs have also been useful in the management of pain in various dermatological conditions.

Box 1: Key considerations when using antidepressants in dermatologic settings.

USE OF MOOD STABILIZERS IN DERMATOLOGY

Mood stabilizers are a class of psychotropic medications used in the treatment of bipolar disorder. In dermatologic settings, the use of mood stabilizers has been found to be beneficial in several cases of pruritus and some cutaneous sensory syndromes. Most of these psychotropics can have

Table 2: Dosing and monitoring suggestions for commonly used antidepressants.			
Agent	Dosing	Adverse effects	Monitoring guidelines
SSRI			
Fluoxetine	Initiate at 20 mg daily. Titrate gradually. Target dose is 20–60 mg/day	Nausea, vomitting, diarrhea, headache, dry mouth, insomnia, agitation,	Baseline ECG, LFT, BMI and waist circumference, Vitamin D, B12,
Fluvoxamine	Initiate at 25 mg daily. Titrate gradually. Target dose is 50–200 mg/day	orthostatic hypotension, appetite changes, sexual dysfunction. QTc prolongation is a concern with most	folate, zinc, magnesium Repeat LFT when clinically indicated. Monitor BMI at
Paroxetine	Initiate at 20 mg daily. Titrate gradually. Target dose is 20–40 mg/day	agents in this class. Weight gain is also seen with all except fluoxetine	1 month and 6 months Monitor at baseline and after
Sertraline	Initiate at 50 mg daily. Titrate gradually. Target dose is 50–200 mg/day	-	1 month for hyponatremia, particularly in elderly patients. Monitor for emergent suicidality,
Escitalopram	Initiate at 10 mg daily. Titrate gradually. Target dose is 10–20 mg/day		particularly in adolescents
TCA			
Amitriptyline	Initiate at 25 mg nightly daily. Titrate gradually. Target dose is 100–300 mg/day in moderate and severe depression	Anticholinergic side effects such as dry mouth and constipation. Drowsiness, orthostatic hypotension, weight	In addition to the above, particularly monitor ECG at baseline and after each dose
Imipramine	Initiate at 25 mg nightly daily. Titrate gradually. Target dose is 150–300 mg/day	gain, and sexual dysfunction are also common. QTc prolongation is a concern. In addition, with imipramine, there is a risk of agitation/insomnia in some	change. Monitor blood pressure at baseline and with each significant dose increase
SNRI			
Venlafaxine	Initiate at 37.5–75 mg/day. Titrate gradually. Target dose is 75–375 mg/day	Drowsiness, agitation, QTc prolongation, gastrointestinal toxicity, sexual dysfunction	In addition to the above, monitor blood pressure at baseline, after each dose titration, and at
Duloxetine	Initiate at 30–60 mg/day. No titration needed. Usual dose is 60 mg/day	Insomnia, agitation, gastrointestinal symptoms, sexual dysfunction	3–6 months after dose stabilization
ECG: Electrocardiogr	am, LFT: Liver function test, BMI: Body mass i	index, TCA: Tricyclic antidepressants, SSRI: Sel	ective serotonin reuptake inhibitor.

SNRI: Serotonin norepinephrine reuptake inhibitor

adverse reactions ranging from benign cutaneous lesions to life-threatening lesions. Below, we discuss key considerations when using different mood stabilizing agents.

Lithium

Patients with skin picking and TTM, both of which are associated with impulse dyscontrol, have been found to benefit with the agent. The drug is dosed commonly at a range of 600-1200 mg/day, titrated to achieve a target serum level of 0.5-1 mEq/l. Renal and thyroid function must be regularly monitored while on lithium.[27] The commonly reported dermatologic side effects of this agent are discussed in a subsequent section.

Carbamazepine

Mainly used for the treatment of bipolar disorder, epilepsy, and neuropathic pain syndromes, its therapeutic use in postherpetic neuralgia, a painful condition involving the nerves and skin, deserves special note. It is generally recommended to start with low doses and slowly up titrate to a target dose of 600-1200 mg/day. An important pharmacokinetic consideration is the phenomenon of auto-induction, wherein the drug induces its own metabolism. This may necessitate a dose adjustment for some individuals.

Lamotrigine

Primarily used in the treatment of seizures, lamotrigine has demonstrated efficacy in the treatment of skin picking.^[27] Evidence suggests the use of doses between 12.5 mg and 300 mg/day. The exact mechanism of action is unclear, but the agent acts by reducing neuronal excitability, stabilizing membranes, and inhibiting glutamate release.

Sodium valproate

It is one of the first-line agents for the treatment of bipolar disorder and epilepsy worldwide and acts by potentiating gamma-amino butyric acid (GABA)-based neurotransmission in the central nervous system. It is also used for the treatment of neuropathic pain syndromes. There are few reports of valproate being successfully used to manage cases of inflammatory verrucous epidermal nevi.^[28] The preferred target dosage of sodium valproate is 20–30 mg/kg/day in divided doses with a starting dose of 10–15 mg/kg/day.

Treatment of comorbid mood disorders

Mood stabilizers have been effective in the management of bipolar disorders as well as occasional augmentation in unipolar depressive disorders. For patients presenting with both chronic dermatological conditions and mood disorders, the mood stabilizer must be chosen keeping in mind the adverse reaction profile of the drug. Table 3 summarizes indications for treatment with different mood stabilizers in dermatology while Table 4 summarizes dosing and monitoring considerations for commonly used mood stabilizers.^[29]

USE OF ANTIPSYCHOTICS IN DERMATOLOGY

In addition to the dopamine blockade action of antipsychotics, they have cholinergic muscarinic receptor, α 1-adrenergic receptor, and histamine H1-receptor blocking effects, to varying extent. This property can be used to manage histamine or sympathetically mediated dermatologic symptoms (e.g., urticaria, pruritus, and hyperhidrosis).^[30,31] At present, usage of antipsychotic drugs for the treatment of a primary dermatologic disorder represents an off-label indication.^[32]

Delusional disorder, somatic type (delusional infestation [DI]; monosymptomatic hypochondriacal psychosis; delusions of parasitosis; and Morgellons disease)

Patients complain of being infested with living pathogens (e.g., insects) or inanimate materials such as fibers or strands (Morgellons disease). Pimozide has been mentioned extensively as a treatment of DI; most of the supporting data come from non-randomized trials and case series/reports on the use of first-generation antipsychotics (FGAs) or second-generation antipsychotics (SGAs) in DI.^[33,34] Pimozide has been tried for a period of 4–6 weeks with good response across trials; dosages range from 1 mg to 5 mg.^[35,36] However, its use is significantly limited by side effects such as QTc prolongation and extrapyramidal symptoms.^[34,37]

Table 3: Indications for use of mood stabilizers in dermatology.

Mood stabilizing agent	Indication in dermatology
Lithium	TTM Skin picking
Carbamazepine Lamotrigine Sodium valproate	Post-herpetic neuralgia Skin picking Inflammatory verrucous epidermal nevi
TTM: Trichotillomania	

For SGAs, there is limited evidence for their efficacy in DI; evidence is mainly from case series using relatively low doses of risperidone (1–2 mg daily) and olanzapine (2.5–5 mg daily). Risperidone and olanzapine have been associated with partial or full remission of DI in 69% and 72% of cases, respectively, with maximum effects being reported after at least 6 weeks of treatment.^[37,38] A systematic review of the use of FGAs and SGAs in delusional parasitosis concluded that despite weaker evidence for the effectiveness of SGAs in treating DI in comparison with pimozide, their use may improve patient adherence and treatment outcome, due to their more favorable side effect profile.^[33] Pimozide is no longer considered to be the drug of choice due to safety concerns.^[33,34,39]

BDD

BDD is a psychiatric disorder that is encountered more commonly in dermatologic and cosmetic surgical settings than psychiatric clinics, where patients present with one or more perceived "defects" in the appearance of their skin or hair that is slight or not observable to others.^[40] While at least one-third of BDD patients have delusional BDD, the current evidence supporting the use of antipsychotics in BDD is weak.

A Cochrane review of pharmacotherapy, psychotherapy, or combination of the two for BDD (non-delusional or delusional) identified two randomized controlled trials (RCTs) involving antidepressants fluoxetine and clomipramine, and three RCTs involving cognitive behavior therapy. There were no RCTs using antipsychotics and treatment responses in both medication trials were not affected by whether the BDD was delusional or non-delusional.^[41] Studies have used antipsychotics to augment the effect of fluoxetine or venlafaxine (selective norepinephrine reuptake inhibitor) in BDD.^[42,43] These antipsychotics include olanzapine,^[42,44,45] pimozide,^[43] haloperidol,^[46] and risperidone.^[47]

Body-focused repetitive behaviors (BFRBs)

TTM (hair pulling disorder) and SPD are both considered BFRBs and are classified under the obsessive-compulsive and related disorders in the Diagnostic and Statistical Manual of Mental Disorders-5.^[40] Olanzapine dosed up to 20 mg/ day, showed positive effects in a 12-week placebo-controlled RCT of adults diagnosed with TTM.^[48] Positive evidence for efficacy of olanzapine, in uncontrolled observations, exists in SPD^[49,50] and conditions associated with self-excoriation such as prurigo nodularis.^[51]

Dermatitis artefacta (DA)

DA includes a varied group of self-inflicted injury-related disorders affecting the skin, which usually present across a

Table 4: Dosing and monitoring suggestions for commonly used mood stabilizers.			
Agent	Dosing	Adverse effects	Monitoring guidelines
Lithium	Initiate at 600–900 mg/day in two or three divided doses. Smaller starting doses (150– 300 mg/day) may be suitable in the elderly. Titrate gradually based on serum level (target serum level for acute treatment is 0.8–1.2 meq/L)	Nausea, loose stools, polyuria, thirst, tremors, weight gain, cognitive dulling	Baseline ECG, RFT, TFT, and calcium. Renal function test, thyroid function test, and calcium. Pregnancy test for women of reproductive potential. Lithium levels to be checked 5–7 days after starting treatment, after every dose increase, and subsequently till levels are stable. Monitor RFT every 2–3 months for the first 6 months and every 6–12 months subsequently. Monitor TFT every 3 months in the first 6 months, and every 6–12 months thereafter
Carbamazepine	Initiate at 100–200 mg once or twice daily. Titrate gradually. Usual target dose is 800–1600 mg/day	Nausea, vomiting, diarrhea, pruritus, leukopenia, rash, and fluid retention. Life-threatening rash (SJS and TEN) may be seen, particularly in the first 8 weeks after initiating therapy	Perform baseline complete blood count, liver function test, and serum sodium. Monitor each of these along with serum carbamazepine at 6–12 months intervals
Lamotrigine	Initiate at 25 mg/day for weeks 1 and 2. Titrate as follows: Weeks 3 and 4: 50 mg, week 5: 100 mg. Week 6: 200 mg. Usual maintenance dose is≤200 mg/day. Titration schedule should be slower when concurrently taking drugs that inhibit lamotrigine metabolism and, conversely, can be faster when taking inducers of lamotrigine metabolism	Nausea, chest pain, edema, increased libido, weight gain, insomnia, agitation, anxiety, abnormal dreams, drowsiness, flushing, hypertension, orthostatic hypotension, hepatitis, polyuria. It can cause both benign and serious rash, particularly in pediatric patients, and within 2–8 weeks of treatment initiation. History of hypersensitivity (rash, angioedema, urticaria, pruritus) to lamotrigine is a contraindication	Baseline LFT, RFT, and inquiry of hypersensitivity should be performed. Routine monitoring of serum lamotrigine is not indicated but everyone prescribed the agent should be educated about the potential for fever and skin rash and to report emergence of any of these signs immediately. LFT and RFT may be monitored regularly
Sodium valproate	Initiate at 500–750 mg/day in two or three divided doses. Increase by 250–500 mg every 2–3 days as tolerated to reach a target dose of 20–30 mg/kg body weight	Common side effects are nausea, vomiting, tremor, weight gain, and hair loss. Uncommon or rare side effects include fulminant hepatic failure, thrombocytopenia, and pancreatitis	Avoid in women of childbearing potential. Baseline blood counts and liver function tests are to be done before initiation. Monitor these at regular intervals, especially during the first 6 months after initiation. Symptoms of abdominal pain and vomiting while on valproate mandate a serum amylase and lipase testing together with a surgical evaluation to rule out emergent pancreatitis

ECG: Electrocardiogram, RFT: Renal function test, TFT: Thyroid, LFT: Liver function test, SJS: Stevens-Johnson syndrome, TEN: Toxic epidermal necrolysis

range of psychiatric diseases (e.g., psychotic disorders, posttraumatic stress disorder, dissociative disorders, and personality disorders). There are reports of the successful use of pimozide 0.5–1 mg daily, aripiprazole 2–5 mg daily, risperidone 0.5 mg daily, and olanzapine up to 5 mg daily, in DA.^[52-54]

Pruritus

Pruritus is one of the most prevalent and disturbing symptoms of dermatologic disease. Recent literature supports the use of antipsychotics mostly as adjunctive therapy for sleep difficulties in chronic pruritus which may be linked to its antihistaminic effects.^[55] A 2016 Cochrane review of pharmacologic treatments for pruritus in adult palliative care patients did not identify any RCTs involving antipsychotic agents.^[56]

Older studies have described the beneficial effect of chlorpromazine, dosed up to 200 mg, for chronic pruritus.^[57] Subsequent larger studies have described the possible beneficial effect of the phenothiazine FGAs, such as trimeprazine, in pruritus as an adjunct to other primary therapies, possibly related to their tranquilizing properties.^[58,59]

Hyperhidrosis

The effect of antipsychotics on hyperhidrosis is thought to be related to both the peripheral anticholinergic and central anxiolytic effects associated with α -adrenergic receptor blockade. There are case reports demonstrating efficacy of thioridazine (10–25 mg) and olanzapine (10 mg) for treating hyperhidrosis in late-stage malignancies and low-dose quetiapine (12.5–25 mg) for treating axillary hyperhidrosis.^[60-63]

Others

Pimozide is a dopamine receptor antagonist and has been shown to have an inhibitory effect in melanoma.^[64,65] Chlorpromazine, a calmodulin inhibitor, has been shown to have a beneficial effect in psoriasis, a disorder characterized by aberrant cell proliferation and cell division linked to abnormal regulation of calmodulin activity.^[66,67] Preferred antipsychotics in various dermatologic disorders are depicted in Table 5 while Table 6 summarizes dosing and monitoring considerations for commonly prescribed antipsychotic agents.^[68]

USE OF ANXIOLYTICS IN DERMATOLOGY

These agents, used to reduce anxiety, are divided into benzodiazepines and non-benzodiazepines.^[1] They are particularly useful in the management of social anxiety which, more often than not, may be secondary to skin lesions.^[69] Anxiety symptoms often cooccur with dermatologic disorders and there is a bidirectional association between them. Major anxiety disorders include generalized anxiety disorder, panic disorder, social phobia, and obsessive-compulsive disorder.^[9]

Table 5: Use of antipsychotics in dermatology.		
Dermatological conditions	Antipsychotics preferred	
Delusional infestation	Pimozide	
	Low-dose risperidone	
	Low-dose olanzapine	
Body dysmorphic disorder	Olanzapine	
	Risperidone	
	As augmenting agent with	
	Haloperidol	
	SSRIs	
	Pimozide	
BFRBs	Olanzapine	
Dermatitis artefacta	Pimozide	
	Aripiprazole	
	Risperidone	
	Olanzapine	
Pruritus	Chlorpromazine	
	Trimeprazine	
Hyperhidrosis	Thioridazine	
	Olanzapine	
BFRBs: Body-focused repetitive behavior reuptake inhibitors	s, SSRIs: Selective serotonin	

Benzodiazepines

They act through modulation of GABA, an inhibitory neurotransmitter. They have high sedative potential and a tendency to develop a dependence on long-term use and potential for abuse. Hence, their use should be limited to short courses (<4 weeks) with slow tapering to prevent withdrawal symptoms.

Non-benzodiazepines

Buspirone

Being a serotonin partial agonist (5HT1A), its advantage lies in its minimal potential for dependence as well as withdrawal symptoms following cessation. Onset of action is slower and typically requires several weeks; this may limit utility in acute cases. Hence, it is preferred in cases where longer therapies need to be instituted. Preferred initial dose is 15 mg/day in divided doses due to its short half-life, with subsequent increases by 15 mg every week, till a maximum dose of 60 mg/day, as indicated. Side effects include dizziness, headache, sedation, nausea, and restlessness.^[1,27,70]

Zolpidem

Used as a sleep-inducing agent, zolpidem should be used at a dose of 5-10 mg and taken $\frac{1}{2}$ h before bedtime. The drug acts in a similar way to benzodiazepines and tolerance to its effects can occur overtime; therefore, it should be used for short periods as well.

Hydroxyzine

First-generation histamine-receptor antagonists (H1), such as diphenhydramine and hydroxyzine, readily cross the blood–brain barrier and have anti-anxiety and sedative effects. Hydroxyzine has minimal dependence potential as well as withdrawal symptoms on stoppage. It is one of the preferred anxiolytics for patients with pruritus due to its antihistaminic effects and has Food and Drugs Authority approval for this indication. Anticholinergic antihistamines like diphenhydramine are more likely to cause confusion in the elderly and hence they should be used cautiously in this population.^[9]

ADVERSE SKIN REACTIONS TO PSYCHIATRIC DRUGS

The incidence of adverse skin reaction in a hospitalized patient ranges between 1% and 3% whereas the incidence of the same reaction in patients taking psychotropics is estimated between 2% and 5%.^[71-73] Risk factors for developing CADRs include winter season, female sex, Afro-American ethnicity, old age, initiating with high dose, and

Table 6: Dosing and monitoring suggestions for commonly used antipsychotics.			
Agent	Dosing	Adverse effects	Monitoring guidelines
SGA	Initiate at 1 2 ma daily Tituate	Naussa dumansis unisht ssin	Deceling LET DET DML visiot
Risperidone	gradually. Target dose is 4–8 mg/day given as a single dose nightly or in divided doses	sedation, akathisia, prolactin elevation, sexual dysfunction	circumference, blood pressure, ECG, and fasting plasma glucose, and lipid profile. Pregnancy
Olanzapine	Initiate at 5–10 mg once daily or in two divided doses. Titrate gradually. Target dose is 10–30 mg/day taken at bedtime or in divided doses	Sedation, dry mouth, constipation, increased appetite, weight gain, and orthostatic hypotension, sexual dysfunction	test in women of childbearing potential. Weight must be monitored at 4, 8, 12 weeks and quarterly thereafter while fasting
Quetiapine	Initiate at 50–100 mg/day in divided doses. Titrate gradually. Target dose is 400–800 mg/day taken at bedtime or in divided doses	Headache, sedation, dry mouth, weight gain, constipation, and orthostatic hypotension, sexual dysfunction	glucose, lipids, blood pressure, and waist circumference must be repeated at 12 weeks and annually thereafter
Aripiprazole	Initiate at 10–30 mg once daily. Titrate gradually. Target dose is 15–30 mg/day	Nausea, constipation, vomiting, headache, insomnia, akathisia	
FGA			
Haloperidol	Initiate at 2.5–5 mg nightly daily. Titrate gradually. Target dose is 5–20 mg/day given as a single dose or in two divided doses	Extrapyramidal symptoms (tremor, rigidity, bradykinesia), akathisia, prolactin elevation, and tardive dyskinesia, sexual dysfunction. QTc prolongation seen with intravenous formulations	Same as above. In addition, if intravenous haloperidol is being given, daily ECG monitoring for QTc prolongation must be carried out
Chlorpromazine	Initiate at 25 mg nightly daily. Titrate gradually. Target dose is 150–300 mg/day	Sedation, dry mouth, cholestatic jaundice, pigmentary retinopathy, prolactin elevation, sexual dysfunction, orthostatic hypotension. Rarely, agranulocytosis may occur. QTc prolongation is seen	
I FT. Liver function te	st RFT Renal function test RMI Body mass in	dev ECG: Electrocardiogram SGA: Second-g	eneration antipsychotic EGA:

First-generation antipsychotic

human leukocyte antigen subtypes clustering.^[72,74-80] Below, we outline common adverse skin reactions to common psychotropic agents.

Mood stabilizers

The incidence of CADRs with this class of drugs is 0.23%.^[81] Despite this low figure, there are serious adverse drug reactions associated with this class of drugs. Carbamazepine and lamotrigine are common offending agents among this group.^[81]

Lithium

The incidence of CADRs with lithium is 0.01%; these are more common among women.^[81] The most common CADR reported with lithium use are exacerbation of psoriasis, acneiform eruptions, folliculitis, alopecia, and exanthems.^[82] The mechanism of acneiform eruption and psoriasis is believed to be due to inhibition of adenylate cyclase activity in keratinocytes.^[81-83]

Carbamazepine

Incidence of CADR is around 0.32–17% of patients depending on the dose initiated.^[81,84] The most common cutaneous adverse reaction is erythematous macular and papular exanthem while serious drug reactions include Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug reaction with eosinophilia and systemic symptoms (DRESSs). Oxcarbazepine also has similar profile of adverse skin reactions as carbamazepine.^[85]

Lamotrigine

The incidence of CADRs is around 0.6–5%.^[86,87] The most common adverse effect is erythematous macular and papular exanthem. SJS/TEN has an incidence of 0.08% with lamotrigine.^[88] Rarely, urticaria and erythema multiforme are also reported with this drug. The risk of eruptions is associated with higher loading doses.^[86,89] Rechallenging the drug after the rash should be considered only when benefits clearly outweigh the risks.

Sodium valproate

When used along with lamotrigine, there is an increased risk of CADR. Valproate use is also associated with alopecia.^[90] Hair loss is noted to be diffuse, non-scarring, and dose related with valproate and may result in poor compliance to the drug.^[91]

Antidepressants

SSRIs

The incidence of CADR with this class of drugs is 0.051%.^[81] Petechiae, purpura, and ecchymoses have been reported with fluoxetine due to its property of inhibiting platelet aggregation.^[92] Alopecia has been reported with all SSRIs with the highest incidence for fluoxamine and lowest for citalopram.^[93] Hirsutism has been reported with paroxetine and fluoxetine.^[74] Hyperhidrosis is also reported with SSRIs with an incidence ranging from 0.1% to 1% for paroxetine, sertraline, and citalopram to 8.6% for fluoxetine.^[94]

TCAs and other antidepressants

TCAs have the highest reported incidence (0.073%) of cutaneous adverse reactions among antidepressants.^[81] SNRIs are most commonly associated with hyperhidrosis which is caused by stimulation of peripheral adrenergic receptors.^[95] Mirtazapine has been associated with rash, acne, exfoliative dermatitis, and alopecia.^[96]

Antipsychotics

The incidence of skin rash with neuroleptics is 0.029%.^[81] The most common CADRs include photosensitivity and pigmentation changes. Among antipsychotics, chlorpromazine is maximally implicated in causing these reactions. These changes are more common among women taking treatment for more than 3 years.^[97] Increased formation of free radicals which activates tyrosinase resulting in increased production of melanin is believed to underlie the drug-induced pigmentation.^[97] Discontinuation of the offending drug, avoiding exposure to sunlight, and using alternative agents such as loxapine are strategies for treating the reaction.^[98]

Anxiolytics

The rate of occurrence of CADRs with benzodiazepine ranges from 0.5/1000 for flurazepam to 4.2/1000 for chlordiazepoxide.^[71] Exanthematous eruptions are the most common type of reaction. This type of skin reaction is also found among barbiturates with a slightly higher propensity to progress to serious reactions such as TEN and exfoliative dermatitis. Blisters and bullae type eruptions have also

Table 7: Adverse skin reactions to psychiatric drugs.		
Psychiatric drugs	Adverse skin reactions	
Lithium	Acneiform eruptions Exacerbation of psoriasis Folliculitis Exanthems	
Sodium valproate	Increases the risk of lamotrigine induced skin reaction Alopecia	
Carbamazepine	Erythematous maculopapular exanthem TEN/SJS DRESS	
Lamotrigine	Erythematous maculopapular exanthem TEN/SJS DRESS	
SSRIs	Petechiae Purpura Ecchymoses	
SNRIs and mirtazapine	Alopecia Hyperhidrosis Hyperhidrosis Acne Exfoliative dermatitis	
Antipsychotics	Alopecia Photosensitivity Skin pigmentation	
SSRI: Selective serotonin reuptake norepinephrine reuptake inhibito	e inhibitor, SNRI: Serotonin r; TEN: Toxic epidermal necrolysis,	

norepinephrine reuptake inhibitor; TEN: Toxic epidermal necrolysis, SJS: Stevens-Johnson syndrome, DRESS: Drug rash with eosinophilia and systemic symptom

been described with benzodiazepine and barbiturate toxicity.^[99] Table 7 summarizes the common CADR to important psychotropic agents.

CONCLUSION

Due to the considerable cooccurrence of psychiatric symptoms in dermatologic settings, there is a need for the dermatologist to have reasonable awareness and knowledge of psychotropic medications, their indications, and adverse effect profiles. Further, treatment of psychiatric symptoms is part of the comprehensive management of patients and may lead to improved therapeutic results, better adherence to dermatologic agents, and improved quality of life. It is also important to note that patients are more likely to accept psychotropics when prescribed by a dermatologist due to less stigma attached compared to consulting a mental health professional. Choice of psychotropic agent should be based on considerations such as the underlying psychiatric diagnosis, potential for pharmacokinetic interactions, and adverse effect profiles. Whereas it is always best to refer to a psychiatrist, in ordinary cases of common mental illnesses, all doctors can prescribe common anti-anxiety and antidepressant drugs, particularly those elaborated in this chapter.

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Declaration of patient consent

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

- 1. Weber MB, Recuero JK, Almeida CS. Use of psychiatric drugs in dermatology. An Bras Dermatol 2020;95:133-43.
- Connor CJ. Management of the psychological comorbidities of dermatological conditions: Practitioners' guidelines. Clin Cosmet Investig Dermatol 2017;10:117-32.
- Menon V, Kuppili PP, Chandrasekhar L. Research in psychodermatology. In: Ashwini PK, Kishor M, editors. Essentials of Psychiatry for Dermatology and Aesthetic Practice, 978-81-948549-6-8. Tamil Nadu: Apsara Prakashana; 2021. p. 207-19.
- Connor CJ, Liu V, Fiedorowicz JG. Exploring the physiological link between psoriasis and mood disorders. Dermatol Res Pract 2015;2015:e409637.
- 5. Dowlatshahi EA, Wakkee M, Arends LR, Nijsten T. The prevalence and odds of depressive symptoms and clinical depression in psoriasis patients: A systematic review and metaanalysis. J Invest Dermatol 2014;134:1542-51.
- Fleming P, Bai JW, Pratt M, Sibbald C, Lynde C, Gulliver WP. The prevalence of anxiety in patients with psoriasis: A systematic review of observational studies and clinical trials. J Eur Acad Dermatol Venereol 2017;31:798-807.
- 7. Gupta MA, Gupta AK. Psychodermatology: An update. J Am Acad Dermatol 1996;34:1030-46.
- 8. Koo J, Lebwohl A. Psycho dermatology: The mind and skin connection. Am Fam Physician 2001;64:1873-8.
- 9. Shah B, Levenson JL. Use of psychotropic drugs in the dermatology patient: When to start and stop? Clin Dermatol 2018;36:748-55.
- Blier P. Optimal use of antidepressants: When to act? J Psychiatry Neurosci 2009;34:80.
- Ionescu DF, Rosenbaum JF, Alpert JE. Pharmacological approaches to the challenge of treatment-resistant depression. Dialogues Clin Neurosci 2015;17:111-26.
- 12. Cipriani A, Furukawa TA, Salanti G, Chaimani A, Atkinson LZ,

Ogawa Y, *et al.* Comparative efficacy and acceptability of 21 antidepressant drugs for the acute treatment of adults with major depressive disorder: A systematic review and network meta-analysis. Lancet 2018;391:1357-66.

- 13. Huhn M, Nikolakopoulou A, Schneider-Thoma J, Krause M, Samara M, Peter N, *et al.* Comparative efficacy and tolerability of 32 oral antipsychotics for the acute treatment of adults with multi-episode schizophrenia: A systematic review and network meta-analysis. Lancet 2019;394:939-51.
- 14. Brøsen K. Differences in interactions of SSRIs. Int Clin Psychopharmacol 1998;13 Suppl 5:S45-7.
- 15. Hemeryck A, Belpaire FM. Selective serotonin reuptake inhibitors and cytochrome P-450 mediated drug-drug interactions: An update. Curr Drug Metab 2002;3:13-37.
- 16. Spina E, Pisani F, Perucca E. Clinically significant pharmacokinetic drug interactions with carbamazepine. An update. Clin Pharmacokinet 1996;31:198-214.
- 17. Sharma P, Savitha S, Ghosh S, Behere R. Relevant issues in pharmacotherapy of psycho-cutaneous disorders. Indian J Dermatol 2013;58:61.
- 18. Bewley A, Taylor R, Reichenberg J, Magid M. Practical Psychodermatology. Chichester: John Wiley & Sons; 2014.
- Grant JE, Odlaug BL, Chamberlain SR, Keuthen NJ, Lochner C, Stein DJ. Skin picking disorder. Am J Psychiatry 2012;169:1143-9.
- 20. Overview Obsessive-compulsive Disorder and Body Dysmorphic Disorder: Treatment, Guidance. NICE. Available from: https://www.nice.org.uk/guidance/cg31 [Last accessed on 2021 Oct 11].
- 21. Darnall BD. Psychological Treatment for Patients with Chronic Pain. 1st ed. Washington, DC: American Psychological Association; 2018.
- 22. Wong JW, Koo JY. Psychopharmacological therapies in dermatology. Dermatol Online J 2013;19:18169.
- 23. Eskeland S, Halvorsen JA, Tanum L. Antidepressants have anti-inflammatory effects that may be relevant to dermatology: A systematic review. Acta Derm Venereol 2017;97:897-905.
- 24. Savage KT, Singh V, Patel ZS, Yannuzzi CA, McKenzie-Brown AM, Lowes MA, *et al.* Pain management in hidradenitis suppurativa and a proposed treatment algorithm. J Am Acad Dermatol 2021;85:187-99.
- 25. Moore RA, Derry S, Aldington D, Cole P, Wiffen PJ. Amitriptyline for neuropathic pain in adults. Cochrane Database Syst Rev 2015;2015:CD008242.
- Dodd S, Malhi GS, Tiller J, Schweitzer I, Hickie I, Khoo JP, et al. A consensus statement for safety monitoring guidelines of treatments for major depressive disorder. Aust N Z J Psychiatry 2011;45:712-25.
- Kuhn H, Mennella C, Magid M, Stamu-O'Brien C, Kroumpouzos G. Psychocutaneous disease: Clinical perspectives. J Am Acad Dermatol 2017;76:779-91.
- 28. Gupta MA, Pur DR, Vujcic B, Gupta AK. Use of antiepileptic mood stabilizers in dermatology. Clin Dermatol 2018;36:756-64.
- 29. Ng F, Mammen OK, Wilting I, Sachs GS, Ferrier IN, Cassidy F, *et al.* The international society for bipolar disorders (ISBD) consensus guidelines for the safety monitoring of bipolar disorder treatments. Bipolar Disord 2009;11:559-95.
- 30. Procyshyn RM, Bezchlibnyk-Butler KZ, Jeffries JJ. Clinical

Handbook of Psychotropic Drugs. Germany: Hogrefe Publishing; 2021.

- 31. Gupta MA, Vujcic B, Pur DR, Gupta AK. Use of antipsychotic drugs in dermatology. Clin Dermatol 2018;36:765-73.
- 32. Gee SN, Zakhary L, Keuthen N, Kroshinsky D, Kimball AB. A survey assessment of the recognition and treatment of psychocutaneous disorders in the outpatient dermatology setting: How prepared are we? J Am Acad Dermatol 2013;68:47-52.
- Lepping P, Russell I, Freudenmann RW. Antipsychotic treatment of primary delusional parasitosis: Systematic review. Br J Psychiatry 2007;191:198-205.
- Generali JA, Cada DJ. Pimozide: Parasitosis (delusional). Hosp Pharm 2014;49:134-5.
- Hamann K, Avnstorp C. Delusions of infestation treated by pimozide: A double-blind crossover clinical study. Acta Derm Venereol 1982;62:55-8.
- 36. Zomer SF, De Wit RF, Van Bronswijk JE, Nabarro G, Van Vloten WA. Delusions of parasitosis. A psychiatric disorder to be treated by dermatologists? An analysis of 33 patients. Br J Dermatol 1998;138:1030-2.
- 37. Lepping P, Huber M, Freudenmann RW. How to approach delusional infestation. BMJ 2015;350:h1328.
- Freudenmann RW, Lepping P. Second-generation antipsychotics in primary and secondary delusional parasitosis: Outcome and efficacy. J Clin Psychopharmacol 2008;28:500-8.
- Mothi M, Sampson S. Pimozide for schizophrenia or related psychoses. Cochrane Database Syst Rev 2013;11:CD001949.
- 40. Edition F. Diagnostic and Statistical Manual of Mental Disorders. United States: American Psychiatric Association; 2013. p. 21.
- 41. Ipser JC, Sander C, Stein DJ. Pharmacotherapy and psychotherapy for body dysmorphic disorder. Cochrane Database Syst Rev 2009;1:CD005332.
- 42. Phillips KA. Olanzapine augmentation of fluoxetine in body dysmorphic disorder. Am J Psychiatry 2005;162:1022-3.
- 43. Phillips KA. Placebo-controlled study of pimozide augmentation of fluoxetine in body dysmorphic disorder. Am J Psychiatry 2005;162:377-9.
- 44. McWillliams S, Whitty M, Lydon D, Clarke M. Body dysmorphic disorder treated with venlafaxine, olanzapine and cognitive behavioural therapy. Irish J Psychol Med 2005;22:143-6.
- 45. Nakaaki S, Murata Y, Furukawa TA. Efficacy of olanzapine augmentation of paroxetine therapy in patients with severe body dysmorphic disorder. Psychiatry Clin Neurosci 2008;62:370.
- 46. Koblenzer CS. The dysmorphic syndrome. Arch Dermatol 1985;121:780-4.
- 47. Goulia P, Mantas C, Bassukas ID, Hyphantis T. Treatment with risperidone and venlafaxine of a patient with doublecoded diagnosis of body dysmorphic disorder and delusional disorder somatic type. Hippokratia 2011;15:286-7.
- Van Ameringen M, Mancini C, Patterson B, Bennett M, Oakman J. A randomized, double-blind, placebo-controlled trial of olanzapine in the treatment of trichotillomania. J Clin Psychiatry 2010;71:1336-43.
- 49. Gupta MA, Gupta AK. Olanzapine is effective in the

management of some self-induced dermatoses: Three case reports. Cutis 2000;66:143-6.

- 50. Blanch J, Grimalt F, Massana G, Navarro V. Efficacy of olanzapine in the treatment of psychogenic excoriation. Br J Dermatol 2004;151:714-6.
- 51. Hyun J, Gambichler T, Bader A, Altmeyer P, Kreuter A. Olanzapine therapy for subacute prurigo. Clin Exp Dermatol 2006;31:464-5.
- 52. Garnis-Jones S, Collins S, Rosenthal D. Treatment of selfmutilation with olanzapine. J Cutan Med Surg 2000;4:161-3.
- 53. Pascual A, Miguelez A, Vanaclocha F, Rubio G, Iglesias L. Periocular and perioral artefactual dermatitis: Dermatological and psychiatric management in a hospital setting. Dermatol Psychosom Dermatol Psychosom 2001;2:200-2.
- 54. Koblenzer CS. Dermatitis artefacta. Clinical features and approaches to treatment. Am J Clin Dermatol 2000;1:47-55.
- 55. Weisshaar E, Szepietowski JC, Darsow U, Misery L, Wallengren J, Mettang T, *et al.* European guideline on chronic pruritus. Acta Derm Venereol 2012;92:563-81.
- 56. Siemens W, Xander C, Meerpohl JJ, Buroh S, Antes G, Schwarzer G, *et al.* Pharmacological interventions for pruritus in adult palliative care patients. Cochrane Database Syst Rev 2016;11:CD008320.
- 57. Tilley RF, Barry H. Chlorpromazine treatment for relief of itching in severe refractory neurodermatitis. N Engl J Med 1955;252:229-30.
- 58. Fanburg SJ. Ancillary therapy of dermatoses with perphenazine. J Med Soc N J 1959;56:332-4.
- 59. Shanon J. A dermatologic and psychiatric study of perphenazine (trilafon) in dermatology. AMA Arch Dermatol 1958;77:119-20.
- 60. Dickmann LM, Dickmann JR. Quetiapine in the treatment of hyperhidrosis axillaris. Br J Dermatol 2010;163:1126-7.
- Mold JW, Holtzclaw BJ, McCarthy L. Night sweats: A systematic review of the literature. J Am Board Fam Med 2012;25:878-93.
- 62. Abbas SQ. Use of thioridazine in palliative care patients with troublesome sweating. J Pain Symptom Manage 2004;27:194-5.
- 63. Zylicz Z, Krajnik M. Flushing and sweating in an advanced breast cancer patient relieved by olanzapine. J Pain Symptom Manage 2003;25:494-5.
- 64. Taub RN, Baker MA. Treatment of metastatic malignant melanoma with pimozide. Lancet 1979;1:605.
- 65. Neifeld JP, Tormey DC, Baker MA, Meyskens FL, Taub RN. Phase II trial of the dopaminergic inhibitor pimozide in previously treated melanoma patients. Cancer Treat Rep 1983;67:155-7.
- 66. Humbert P, Renaud A, Agache P. Calmodulin inhibitor therapy in psoriasis. Arch Dermatol 1986;122:856-7.
- 67. Reiss F. Psoriasis and stress. Dermatologica 1956;113:71-8.
- 68. Peh AL. Safety monitoring of patients on atypical antipsychotics. Qual Saf Health Care 2008;17:469-72.
- 69. Shenoi SD, Soman S, Munoli R, Prabhu S. Update on pharmacotherapy in psychodermatological disorders. Indian Dermatol Online J 2020;11:307-18.
- 70. Lee CS, Koo J. Psychocutaneous drug therapy. Semin Cutan Med Surg 2003;22:222-33.
- 71. Arndt KA, Jick H. Rates of cutaneous reactions to drugs. A report from the Boston collaborative drug surveillance

program. JAMA 1976;235:918-23.

- Bigby M, Jick S, Jick H, Arndt K. Drug-induced cutaneous reactions. A report from the Boston collaborative drug surveillance program on 15,438 consecutive inpatients, 1975 to 1982. JAMA 1986;256:3358-63.
- 73. Kimyai-Asadi A, Harris JC, Nousari HC. Critical overview: Adverse cutaneous reactions to psychotropic medications. J Clin Psychiatry 1999;60:714-25.
- 74. Warnock JK, Morris DW. Adverse cutaneous reactions to antidepressants. Am J Clin Dermatol 2002;3:329-39.
- 75. Naldi L, Conforti A, Venegoni M, Troncon MG, Caputi A, Ghiotto E, *et al.* Cutaneous reactions to drugs. An analysis of spontaneous reports in four Italian regions. Br J Clin Pharmacol 1999;48:839-46.
- Alvestad S, Lydersen S, Brodtkorb E. Rash from antiepileptic drugs: Influence by gender, age, and learning disability. Epilepsia 2007;48:1360-5.
- 77. Fattinger K, Roos M, Vergères P, Holenstein C, Kind B, Masche U, et al. Epidemiology of drug exposure and adverse drug reactions in two swiss departments of internal medicine. Br J Clin Pharmacol 2000;49:158-67.
- Wang X, Lang S, Shi X, Tian H, Wang R, Yang F. Antiepileptic drug-induced skin reactions: A retrospective study and analysis in 3793 Chinese patients with epilepsy. Clin Neurol Neurosurg 2012;114:862-5.
- McCormack M, Alfirevic A, Bourgeois S, Farrell JJ, Kasperavičiūtė D, Carrington M, *et al.* HLA-A*3101 and carbamazepine-induced hypersensitivity reactions in Europeans. N Engl J Med 2011;364:1134-43.
- Mitkov MV, Trowbridge RM, Lockshin BN, Caplan JP. Dermatologic side effects of psychotropic medications. Psychosomatics 2014;55:1-20.
- Lange-Asschenfeldt C, Grohmann R, Lange-Asschenfeldt B, Engel RR, Rüther E, Cordes J. Cutaneous adverse reactions to psychotropic drugs: Data from a multicenter surveillance program. J Clin Psychiatry 2009;70:1258-65.
- 82. Gupta AK, Knowles SR, Gupta MA, Jaunkalns R, Shear NH. Lithium therapy associated with hidradenitis suppurativa: Case report and a review of the dermatologic side effects of lithium. J Am Acad Dermatol 1995;32:382-6.
- Sarantidis D, Waters B. A review and controlled study of cutaneous conditions associated with lithium carbonate. Br J Psychiatry 1983;143:42-50.
- 84. Chadwick D, Shaw MD, Foy P, Rawlins MD, Turnbull DM. Serum anticonvulsant concentrations and the risk of drug induced skin eruptions. J Neurol Neurosurg Psychiatry

1984;47:642-4.

- 85. Beran RG. Cross-reactive skin eruption with both carbamazepine and oxcarbazepine. Epilepsia 1993;34:163-5.
- Huang CW, Tsai JJ, Lai ML. Lamotrigine-related skin rashes in adults. Kaohsiung J Med Sci 2002;18:566-72.
- Drake LA, Dinehart SM, Farmer ER, Goltz RW, Graham GF, Hordinsky MK, *et al.* Guidelines of care for cutaneous adverse drug reactions. Am Acad Dermatol J 1996;35:458-61.
- Sadock BJ. Kaplan and Sadock's Synopsis of Psychiatry: Behavioral Sciences/Clinical Psychiatry. Philadelphia, PA: Lippincott Williams and Wilkins; 2007.
- Wong IC, Mawer GE, Sander JW. Factors influencing the incidence of lamotrigine-related skin rash. Ann Pharmacother 1999;33:1037-42.
- Arslan G, Ayranci U, Unsal A, Arslantas D. Prevalence of depression, its correlates among students, and its effect on health-related quality of life in a Turkish university. Ups J Med Sci 2009;114:170-7.
- Ramakrishnappa SK, Belhekar MN. Serum drug level-related sodium valproate-induced hair loss. Indian J Pharmacol 2013;45:187-8.
- 92. Pai VB, Kelly MW. Bruising associated with the use of fluoxetine. Ann Pharmacother 1996;30:786-8.
- Hedenmalm K, Sundström A, Spigset O. Alopecia associated with treatment with selective serotonin reuptake inhibitors (SSRIs). Pharmacoepidemiol Drug Saf 2006;15:719-25.
- Beasley CM, Koke SC, Nilsson ME, Gonzales JS. Adverse events and treatment discontinuations in clinical trials of fluoxetine in major depressive disorder: An updated metaanalysis. Clin Ther 2000;22:1319-30.
- 95. Schwartz TL. Diaphoresis and pruritus with extended-release venlafaxine. Ann Pharmacother 1999;33:1009.
- MacMorran WS, Krahn LE. Adverse cutaneous reactions to psychotropic drugs. Psychosomatics 1997;38:413-22.
- 97. Zelickson AS. Skin changes and chlorpromazine: Some hazards of long-term drug therapy. JAMA 1966;198:341-4.
- 98. Ewing DG, Einarson TR. Loxapine as an alternative to phenothiazines in a case of oculocutaneous skin pigmentation. Am J Psychiatry 1981;138:1631-2.
- 99. Edwards JG. Adverse effects of antianxiety drugs. Drugs 1981;22:495-514.

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