REVIEW

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DRUG-INDUCED PRURITUS

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Abstract. *Drug-induced pruritus is a common problem in daily medical practice. Many drugs can cause pruritus after systemic or topical administration, but often the exact mechanism underlying the pathogenesis of itching caused by drugs remains unclear. Therefore, the diagnosis and treatment of drug-induced pruritus are challenging. In the present review, the current knowledge of the most common medications inducing itch with or without skin rash and their underlying mechanisms is summarized. Opioids can cause pruritus, especially if they are administered intrathecally. Some antineoplastic agents can frequently cause intense pruritus due to immune-related cutaneous reactions (immune checkpoint inhibitors) or via triggering hypersensitivity reactions (taxanes) in cancer patients. Recently, it was found that MAS-related G protein-coupled receptor X2 (MRGPRX2), expressed on mast cells in humans, is responsible for drug-induced non-histaminergic pruritus and pseudoallergic reactions.*

Key words: *itch, pruritus, medications, drugs, ADR*

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INTRODUCTION

Itching is an unpleasant somatic sensation on the skin that leads to scratching. Chronic itching (lasting more than 6 weeks) is the most common symptom in dermatology. It can occur with or without visible skin lesions. Many conditions that affect the skin or various systemic diseases may be accompanied by itching. Pruritus also accounts for approximately 5% of all skin-related adverse reactions to medications [1].

Drug-induced pruritus can be acute (lasting a few days) or chronic (persisting for weeks or months), starting with the first administration of the drug or days after it. It can be localized or generalized and may resolve soon after discontinuing the therapy or can persist for months in some cases. The pathogenesis of drug-induced itch depends on the causative medication. Also, itching may be secondary to drug-induced dry skin [2] or due to cutaneous lesions, e.g., induced by immune checkpoint inhibitors [3, 4]. There are many other potential mechanisms for its occurrence, including cholestasis [5], neurological involvement [6], xeroderma [7], or photoallergy [8]. Often, the mechanisms remain unclear, especially for drugs that induce chronic itching [9].

The most important groups of drugs that could be associated with itching are presented in Table 1. Only

Table 1. Summary of common medications with a higher frequency of pruritus

Medication group	Mechanisms of pruritus	Incidence
Opioids	Centrally mediated process via μ-opioid receptors	2-100%
Antimalarials	Unknown; genetic predisposition; through the local GABAergic system; involvement of ATP- sensitive potassium channels	60-70% of the African population; rare in Caucasian and Asian populations
Hydroxyethyl starch (HES)	Deposition of HES in small peripheral nerves or Schwann cells of skin nerves	12.6-54%
NSAIDs	↑ synthesis of leukotrienes	1-7%
Penicillins	Secondary due to allergic skin lesions or cholestasis	2-20%
Cephalosporins	Unknown or secondary due to allergic skin rash	< 2%
Macrolides	Secondary due to allergic skin rash, or cholestasis	< 0.3%
Tetracyclines	Unknown; or cholestasis	1-2%
Quinolones	Unknown or secondary due to allergic conditions	1-4%
Metronidazole	Unknown or secondary due to allergic rash	< 5%
Trimethoprim/Sulfamethoxazole	Secondary due to allergic rash	1.5-10%
Statins	Unknown or secondary due to allergic skin lesions	16%
ACE inhibitors	↑ bradykinin levels; cholestasis; or secondary skin rash	1-15%
Calcium channel blockers	Secondary due to allergic skin lesions, or unknown	< 2%
Sulfonylurea antidiabetics	Unknown	< 5%
Cytokines, growth factors, and monoclonal antibodies		
● Interleukin 2 (IL-2)	Direct pruritic effect of IL-2	Very common
● Matuzumab	Unknown	< 1 0%
● Lapatinib	Unknown or urticarial reaction	3%
● PD-1 inhibitors (Pembrolizumab)	Immune-related adverse cutaneous reactions	18-34%
● CTLA-4 inhibitors (Ipilimumab)	Immune-related adverse cutaneous reactions	43-45%
● Target cancer therapeutics (generally)		17%
Cytostatics:		
● Tamoxifen	Sebostasis/xerosis	1-7%
● Paclitaxel	Unknown or secondary to hypersensitivity reactions	5-10%

Acronyms: ACE – angiotensin-converting enzyme; CTLA-4 – anti-cytotoxic T-lymphocyte-associated antigen-4; NSAIDs – non-steroidal anti-inflammatory drugs; PD-1 – programmed cell death protein 1

a few groups of them have been extensively studied concerning their relationship to itching, such as antimalarials, opioids, and hydroxyethyl starch (HES). Pruritus can also occur after the topical application of several medications like ciprofloxacin or topical calcineurin inhibitors [10].

Morphinomimetics-induced pruritus

Morphinomimetics are prescribed for the treatment of acute and chronic pain. One of the common adverse effects associated with their use is itching. Many opioids can cause this unwanted symptom that appears in 2-10% of patients treated with oral opioids [11]. The occurrence of itching increases with higher doses of morphinomimetics, as well as when they are administered epidurally or intrathecally. The highest frequency (up to 100%) of itching is associated with intrathecal morphine administration. Itching is most often manifested in the area of the trigeminal nerve, likely due to the high number of opioid receptors in the spinal nuclei of this nerve. Typically, patients scratch their nose or the area around the nose, and the upper part of their face. In rare cases, the symptom may become generalized [12].

The main mechanism associated with opioid-induced itching is centrally mediated via μ-opioid receptors [13]. Additionally, the stimulation of opioid receptors in the skin by various morphinomimetics cannot be ruled out. The modulation of the serotonergic pathway and the involvement of prostaglandins and histamine are also significant. The critical site of opioid action for causing itching is likely the dorsal horn of the spinal cord. In experiments with monkeys, unilateral injection of morphine into this region caused facial itching and scratching [14].

Although neuraxial opioid-induced pruritus can be easily treated due to the clear mechanisms of its occurrence, some therapeutic challenges arise in clinical practice [15]. Naloxone, a classic μ-receptor antago-

nist, can prevent or eliminate itching that occurs with the intrathecal or epidural morphine administration, but it reduces the analgesic effect of morphinomimetics, especially when administered in high doses. Therefore, the use of naloxone is impractical [16]. However, the activation of κ-opioid receptors effectively attenuates intrathecal opioid-induced pruritus without interfering with the nociception [17, 18]. The mixed agonist-antagonist, nalbuphine, decreases the incidence and severity of itch, and its antipruritic effect seems to be linked to decreased IL-31 and increased IL-10 levels [19, 20]. Moreover, during cesarean section, nalbuphine is a preferred agent rather than ondansetron for women with morphine-induced pruritus since the former is not excreted via breast milk [21].

Other agents, such as H1 blockers and droperidol, have been tried with varying success in alleviating itching due to opioids. It was shown that preoperative use of gabapentin prevents itching induced by intrathecal application of morphine in patients undergoing lower limb surgery with spinal anesthesia [22]. Other possibilities for the prevention of pruritus include the reduction of the dose of morphinomimetics administered in combination with anesthetics like sufentanil with bupivacaine. Such combinations provide sufficient analgesia with minimal itching.

Pruritus induced by antimalarials

Chloroquine, widely used as an antimalarial drug, is a prototypical nonhistaminergic pruritogen. It can cause itching in 60-70% of the African population. Interestingly, the drug rarely leads to pruritus in Caucasian or Asian populations [23]. Most patients experience chloroquine-induced itch within the first 24 hours of administration of the drug, which persists for more than 48 hours after the last dose in almost half of the cases. It mainly occurs in young people (under the age of 40), primarily localized on the palms and soles, though in rare cases, the itch can be generalized. There are also reports of itching after the use of other antimalarials, such as hydroxychloroquine and amodiaquine, but it is much less frequent and weaker in intensity [24, 25].

The pathogenesis of chloroquine-induced pruritus is still unclear. A genetic predisposition is suspected, since it primarily occurs in the black-skinned population. The role of pharmacogenomics is supported by the fact that patients with glucose-6-phosphate dehydrogenase deficiency are more common among those who experience itching as a result of the drug administration [26]. Another possible mechanism for the symptom's occurrence in malaria patients is the activation of μ-opioid receptors by endogenous opioid peptides [27, 28].

Some new findings reveal that the medial septum (a part of the basal forebrain) has a modulatory role in the chloroquine-induced pruritus through local increased GABA and an inhibition of non-GABAergic neurons, and via activation of the GABAergic pathway of the anterior cingulate cortex [29]. Other recent experimental data suggested that adenosine triphosphate-sensitive potassium channels are possibly involved in chloroquine-induced itch [30]. Thus, chloroquine-induced itching is a result of a multifactorial physiological mechanism.

Pruritus induced by hydroxyethyl starch

Hydroxyethyl starch, known commercially as hespan is a colloidal solution, one of the most commonly used blood plasma substitutes. The use of HES can be complicated by characteristic adverse effects, including coagulation disorders, bleeding, anaphylactic reactions, and itching. Due to the delayed onset of itching (3-6 weeks) after HES infusion, the symptom was not initially recognized as HES-related adverse effect. The first report of itching was published in the 1980s, but it was well documented as an adverse drug reaction only in the early 1990s. The incidence of HES-induced pruritus is very high, ranging from 12.6% to 54%, depending on the studied population [31, 32].

Itching can occur even with the infusion of small amounts of HES, but it has been found that larger cumulative doses are associated with a higher frequency and intensity of the symptom. Clinically, itching most often manifests as crises lasting from 2 minutes to 1 hour, which can be triggered by rubbing, hot baths, or physical exertion. The pruritus may be generalized or localized, affecting any part of the body without preference. Due to the severe persistent HES-induced pruritus and the limited options for its prevention and treatment, patients often suffer from sleep disturbances, which worsen their quality of life. In some patients, psychiatric help may even be necessary [32, 33].

The pathogenesis of HES-induced itch is still not completely understood, but it is believed that neuronal deposition of HES leads to direct activation of pruritic nerves. The deposition of HES has been found in the form of intracellular vacuoles in skin nerves (Schwann cells, perineural cells, endoneural macrophages), dermal macrophages, endothelial cells of blood and lymphatic vessels, and in some keratinocytes and Langerhans cells, suggesting a cutaneous origin of HES-induced itch [34]. The deposition of HES in the nerves persists for more than 17 months. It is believed that these colloid deposits may mechanically irritate nerve endings, causing a burning itch sensation [35-37].

The treatment of HES-induced pruritus is very difficult. For example, the most commonly used therapeutics against itching, such as H1 blockers, do not lead to improvement in HES-induced itching. Glucocorticoids and neuroleptics are also insufficiently effective. One study found a good response to 0.05% topical capsaicin, but this local therapy is poorly tolerated due to a burning sensation at the application site [38]. Some patients respond favorably to orally administered naltrexone [39]. Other studies have shown a gradual reduction in the sensation of itching in some patients after UV-exposure for several weeks. Despite these attempts, no controlled studies have standardized methods for treating HES-induced pruritus to date. Most modern anti-pruritic agents are ineffective.

Pruritus and immune-related cutaneous reactions induced by checkpoint inhibitors

The use of humanized monoclonal programmed cell death 1 (PD-1) (pembrolizumab and nivolumab) and programmed cell death ligand-1 (PD-L1) (atezolizumab, ipilimumab, avelumab, durvalumab) immune checkpoint inhibitors as antineoplastic agents is rapidly increasing in the treatment of advanced solid malignancies. Cutaneous immune-related adverse effects are the most frequent adverse drug reactions induced by anti-cytotoxic T-lymphocyte-associated antigen-4 (CTLA-4) (i.e., ipilimumab) and anti-PD-1/PD-L1 immune-checkpoint monoclonal antibodies [40]. The adverse inflammatory reactions on the skin are predominantly manifested with pruritus, maculopapular rash, psoriasiform and lichenoid eruptions, and vitiligo-like hypopigmentation/depigmentation. Complications of existing skin lesions can also appear, including vitiligo, worsened psoriasis, mucosal involvement in lichen planus, etc. Skin toxicity and itching can occur at any time during treatment but usually develop early, within the first 6 weeks after the beginning of administration of immune-checkpoint inhibitors [41, 42].

Pruritus due to drug-induced allergic reactions

Taxanes (e.g., paclitaxel, docetaxel) and platinum can frequently induce hypersensitivity reactions (HSRs) and intense pruritus in cancer patients [43, 44]. Often, taxane HSRs occur after the first or second exposure to these drugs. Paclitaxel-treated patients develop mainly cutaneous allergic reactions compared to docetaxel, which, in its turn, causes more often respiratory HSRs [45]. In contrast to taxanes, platinum HSRs occur after multiple chemotherapeutic cycles and a time necessary for the development of IgE sensitization [44].

It is well known that the IgE-mediated or T lymphocyte-mediated drug allergy, clinically manifested with skin rash and pruritus, can be commonly seen after the administration of antibacterial β-lactams and sulfonamides [46, 47], nonsteroidal anti-inflammatory drugs [48], iodinated contrast media [49], and common chemotherapeutics.

Clinically, distinguishing the suspected underlying drug-induced mechanisms is of great importance for the treatment strategies. Immune-mediated and drug-induced skin allergic reactions could be confirmed using cutaneous pre-testing.

Pruritus due to drug-induced pseudoallergic reactions

Various FDA-approved peptidergic drugs can cause anaphylactoid reactions. Fluoroquinolones (e.g., ciprofloxacin, levofloxacin) are known to activate mast cells in a dose-dependent manner, triggering directly a release of a low amount of histamine but a large amount of tryptase, and many cytokines [50, 51]. It was recently found that MAS-related G protein-coupled receptor X2 (MRGPRX2), expressed on mast cells in humans, is responsible for drug-induced non-histaminergic pruritus and pseudoallergic reactions [52, 53]. To predict the diagnosis of fluoroquinolone-induced MRGPRX2-mediated anaphylactoid reactions, conventional basophil and mast cell activation tests by flow cytometry can be used [54]. Similarly to fluoroquinolones, neuromuscular blocking agents such as rocuronium and atracurium could lead to perioperative IgE-independent anaphylaxis by activating MRGPRX2 [55, 56]. In addition, intravenous infusion of vancomycin has led to pruritus in most of the patients enrolled in a recent study via non-immune MRGPRX2 receptor [57].

Drug-induced pruritus is a common problem in daily medical practice and physicians must be aware of it [58]. Subcutaneous or intramuscular injection of some therapeutic agents could lead to immediate local pseudoallergy, also named as injection site reaction, which is characterized by local erythema, swelling, pruritus and pain. Therefore, the MRGPRX2 receptor exhibits a promising potential as a novel therapeutic target for pruritus and drug-induced injection site reactions, and a direction for future research.

CONCLUSION

A number of drugs can cause pruritus, but in many situations, the exact underlying mechanism of the pathogenesis of itching caused by drugs remains unclear. In this review, some examples of well-documented drug-induced pruritus were provided; however, frequently, the direct relationship between a drug and pruritus is not so well established. Patients may

take many different drugs that can potentially cause pruritus, and the drugs cannot be stopped because of the risk of deterioration of the disease being treated. These factors make the final diagnosis and treatment of drug-induced pruritus challenging.

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