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INVITED REVIEW

Pruritus in Pregnancy

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Abstract
Pruritus is a commonly described symptom during pregnancy. Despite its high prevalence, it is often considered trivial but causes significant patient discomfort. It is important to assess and investigate the patient thoroughly as some conditions have a detrimental outcome for both mother and fetus. There is extensive literature on pruritus due to pregnancy-specific dermatoses, however, the evaluation of pruritus merits a broader approach. Various other conditions such as certain infections, systemic diseases, and pre-existing dermatological conditions should also be considered. Awareness of these conditions in obstetricians will also ensure adequate treatment and timely referral, if necessary. The purpose of this article is to describe the etiology, clinical features, diagnostic approach, and management of pruritus in pregnancy.

Keywords Pruritus · Pregnancy · Pregnancy-specific dermatoses · Antihistamines · Atopy

Introduction

Pruritus is an unpleasant sensation causing a desire to scratch [1]. The prevalence of pruritus in pregnancy ranges between 18 and 40% [1]. Pruritus is chronic if symptoms last longer than 6 weeks. Chronic pruritus is an overlooked symptom but is responsible for impairment in quality of life. Conventionally, the discussion of pruritus in pregnancy has been focused on pregnancy-specific dermatosis. However, coexistent dermatological disorders and underlying neurologic, psychological, and systemic conditions are also responsible for pruritus in sizeable expectant populations. Table 1 illustrates the various etiological factors responsible for pruritus in pregnancy. This article aims to provide a comprehensive review of the etiology, pathophysiology, clinical features, investigations, and management of pruritus in pregnancy.

Physiological Causes of Pruritus

Pregnancy is a complex physiological state resulting in several dermatological changes. Striae gravidarum (stretch marks) are seen in the second and third trimesters in > 90% of patients in pregnancy. They are linear pink-purple atrophic bands that develop perpendicular to skin tension lines over the abdomen, breasts, thigh, and buttocks and cause pruritus occasionally. The proposed pathophysiology appears to be related to stretching of the abdominal skin that causes activation of dermal nerve endings [2]. Xerosis (dry skin), hyperactivity of eccrine sweat and sebaceous glands, and hypoactivity of apocrine glands are other physiological causes of pruritus [1]. Awareness of the physiological causes helps avoid unnecessary diagnostic evaluation, dermatological referral, or over-treatment.

Pathological Causes of Pruritus

While there are many pathological causes of pruritus during pregnancy, dermatoses that are unique to pregnancy are most familiar to obstetricians. Table 1 provides the most up-to-date classification of pregnancy-specific dermatosis.

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Table 1 Etiology of Pruritus in pregnancy

Etiology of Pruritus in pregnancy	
Physiological	Striae gravidarum Xerosis
Pathological	Pregnancy specific dermatosis: Polymorphic eruption of pregnancy Pemphigoid gestationis Atopic eruption of pregnancy Intrahepatic cholestasis of pregnancy Dermatological: Pustular psoriasis, chronic plaque psoriasis, eczema, urticaria Infections and infestations Scabies, chicken pox, HIV, zika virus, pityriasis rosea, dermatophytosis Systemic diseases Diabetes mellitus, Hypothyroidism, chronic renal disease, iron deficiency anemia, polycythemia vera Neurological Metalgia paresthetica, post-herpetic neuralgia Psychological Prurigo nodularis, psychogenic pruritus

Pregnancy-Specific Dermatosis

Polymorphic Eruption of Pregnancy

Polymorphic eruption of pregnancy or 'Pruritic urticarial papules and plaques of pregnancy' (PUPPP), is a benign, self-resolving, inflammatory disorder of pregnancy with onset in the third trimester or post-partum. An association has been reported with multiple gestations and excessive maternal weight gain [3].

Etiology Hormonal, immunological, and abdominal distension are presumed etiological factors; however, damage to the connective tissue occurring during stretching is the most likely cause [3].

Clinical features Pruritic papules appear over the abdomen, initially within the striae. Lesions progressively spread to involve the buttocks and proximal thighs and coalesce to form plaques. Occasionally, a generalized involvement is apparent. Umbilical sparing is the hallmark feature. In the later course, the rash may become vesicular, targetoid, eczematous, or simply present as widespread erythema. The condition resolves within 4–6 weeks irrespective of obstetric management or delivery.

Diagnostic evaluation Differential diagnoses include pemphigoid gestationis, contact dermatitis, and atopic dermatitis. A careful history and physical examination are the key to diagnosis as investigations, including histology and immunofluorescence, are unlikely to be helpful. Umbilical sparing is an important clinical clue as it is not seen with other conditions.

Treatment Management is mainly with topical corticosteroids and antihistamines (Table 2). Emollients may provide significant alleviation of symptoms.

Maternal and fetal prognosis The prognosis remains unaffected by this condition with no recurrence in subsequent pregnancies.

Pemphigoid Gestationis

Pemphigoid gestationis (PG), also known as 'herpes gestationis' is a rare pregnancy-specific autoimmune blistering disease with an incidence of 1:2000–1:60,000 pregnancies [1]. Its incidence is dependent on the prevalence of human leukocyte antigen (HLA) haplotypes DR3 and HLA DR4. It commonly presents during the third trimester and postpartum. Associations have been reported with trophoblastic tumors, particularly choriocarcinoma and hydatidiform mole [1]. It predisposes affected individuals to the development of other autoimmune conditions such as Graves' disease.

Etiology Autoantibodies target bullous pemphigoid antigen 180 (BP180) which is found in the basement membrane of the skin, placental tissue, and fetal membranes. It is interesting to note that the placenta is the primary site of autoimmunity and not the skin. The mechanism of blister formation involves anti-placental IgG antibodies cross-reacting with BP180-2 proteins in the skin leading to complement activation with subsequent deposition of immune complexes, chemotaxis of eosinophils to the site, and degranulation. This process destroys the basement membrane and causes bulla formation.

Table 2 Treatment recommendations for pruritus in pregnancy

Treatment		The Food and Drug Administration (FDA) category	Adverse fetal outcomes
General measures	Emollients Glycerin, Liquid paraffin To be applied within 3–5 min of bath and as much as needed	–	–
Topical therapy	Topical steroids: High level of evidence for safety. The only topical corticosteroids contraindicated in pregnancy is Fluticasone propionate	FDA category B	–
	Calcineurin inhibitors Tacrolimus 0.1% ointment/cream and pimecrolimus 1% ointment/cream	FDA category C	Risk of prematurity. There have been contrasting reports and opinions; however, they can be used for flexural areas and the face where topical steroid use is not recommended
	Topical capsaicin 0.025% cream, 8% capsaicin patch	FDA category B	–
Antihistamines	Chlorpheniramine 4 mg every 4–6 h (first-line therapy in the first trimester) Loratadine 10 mg once daily Cetirizine 10 mg once daily Fexofenadine 60–180 mg once daily Levocetirizine 5 mg once daily	FDA category B FDA category B FDA category B FDA category C FDA category B	
Immunosuppressants	Prednisolone 0.5–2 mg/kg/day for control of flare/life threatening situations	FDA category C	Risk of oral clefts due to exposure in first trimester, preterm rupture of membrane, preeclampsia, and gestational diabetes
	Cyclosporine: Monitor blood pressure and renal function	FDA category C	Low body weight in infants and premature labor
	Azathioprine < 2 mg/kg/day (can be used when treatment response to ciclosporin is inadequate)	FDA category D	Increased risk of pre-term delivery), ventricular/atrial septal defects, and a higher risk of growth restriction and preterm
Biologics	Omalizumab (humanized recombinant monoclonal antibody against IgE) 150 mg every 2 weeks	FDA category B	
	Dupilumab (L-13/-4Rα chain inhibitor) 600 mg loading dose followed by 300 mg every 2 weeks	Not assigned	Should be used with caution and after careful consideration of the risk–benefit ratio given its potential to cross and accumulate in the placenta
	Rituximab 1 g IV 2 doses, 2 weeks apart	FDA category C (contraindicated in pregnant women, within 1 year of conception and during lactation as per drug leaflet)	Risk of preterm birth and low birth weight
	TNF alpha inhibitor Infliximab 5 mg/kg IV (limit use to first 30 weeks of gestation)	FDA category B	
Phototherapy	NBUVB and UVA1 supplemented by folic acid 0.5–0.8 mg/day is safe	-	-
Intravenous immunoglobulin (IVIG)	2 g/kg per cycle	FDA category C	-

Table 2 (continued)

Treatment		The Food and Drug Administration (FDA) category	Adverse fetal outcomes
Antivirals	Acyclovir Suppressive therapy 400 mg twice daily Varicella zoster 10 mg/kg every 8 h IV	FDA category B	
Topical Antifungals	Clotrimazole, oxiconazole	FDA category B	–
	Terbinafine	FDA category B	–
	Ciclopirox	FDA category B	–
Oral antifungals	Terbinafine (Not recommended)	FDA category B	–
Topical anti-parasitic	Permethrin 5% cream, apply for 8 h overnight from the neck down. Repeat application after a week is helpful	FDA category B	–
Dapsone	50–150 mg/day	FDA category C	–
Topical antibiotics (treatment of secondary infection in atopic dermatitis)	Fusidic acid (preferred) Mupirocin 2% ointment	FDA category B	

Clinical features The initial presentation is severe pruritus (pre-bullous stage), followed by the development of urticarial papules and plaques over the abdomen, predominantly in the periumbilical region which later extends to other parts of the abdomen and extremities. The face and mucosae are spared. Later on, tense blisters develop.

Diagnostic evaluation The gold standard for diagnosis is direct immunofluorescence (DIF) of perilesional skin which shows deposition of linear C3 and/or IgG along the dermo-epidermal junction. Enzyme-linked immunosorbent assay (ELISA) is a non-invasive serological test with 94–98% specificity and 86–97% sensitivity which can be used for disease monitoring [1].

Treatment The management depends on the severity and is primarily aimed at the relief of pruritus, and prevention of blister formation (Table 3).

Maternal and Fetal Prognosis In mothers, the disease course follows exacerbations and remissions. It tends to flare characteristically during delivery or immediately postpartum resolving spontaneously within weeks to months. Lesions may reappear during menstruation and hormonal contraception. It may recur in future pregnancies in about 30–50% of cases and often presents at an earlier stage and with greater severity [1].

Fetal complications such as preterm labor, intrauterine growth retardation, and neonatal pemphigoid gestationis may occur, and the risk correlates with the disease severity. Neonatal pemphigoid gestationis is characterized by tense blisters due to the passive transfer of the antibodies and the management is conservative.

Intrahepatic Cholestasis of Pregnancy (ICP)

Etiology It is a hepatic disease, synonymously known as 'obstetric cholestasis' or 'jaundice of pregnancy', that arises due to an interplay of hormonal, environmental, and genetic factors. Mutation of *ABCB4* (*MDR3*), involved in the biliary secretion of phospholipids, has been theorized to have a role in a small population [1]. Other conditions associated with ICP include gallstones, hepatitis C infection, preeclampsia, and gestational diabetes [1]. The primary pathology is an inability to excrete bile salts from the liver, which results in elevated serum bile acid concentration and therefore, pruritus.

Clinical features It is characterized by pruritus over the abdomen, palms, and soles beginning in the second or third trimester without an associated rash, although dermatological manifestations vary from subtle excoriations to intensely pruritic nodules [3]. Jaundice presents 2–4 weeks after the pruritus.

Table 3 Treatment of Pemphigoid gestationis

Treatment of Pemphigoid gestationis	
Pre-bullous stage	Topical steroids + antihistamines If steroids are contraindicated, topical calcineurin inhibitors (no more than 5 g/day for 2–3 weeks)
Bullous	Short courses of systemic corticosteroids (prednisolone, usually started at a dose of 0.5–1 mg/kg/day) Taper if adequate response (no new blister formation for 2 weeks)
Refractory	Azathioprine 2 < mg/kg/day intravenous immunoglobulins (IVIG) 2 g/kg divided over 3 days every 4 weeks, dapsone 125 mg daily, plasma exchange, 2 doses of rituximab 1 g at a 2-week interval

Diagnostic evaluation Diagnosis is established when the level of non-fasting total serum bile acids is elevated (≥ 10 μmol/L) [1]. If the level of serum bile acid is normal and a strong clinical suspicion persists, blood work should be repeated weekly. A repeat measurement is recommended 4–6 weeks postpartum to ensure resolution as persistently elevated levels indicate alternative diagnoses. Liver enzymes are usually normal [3]. Alkaline phosphatase levels may be elevated.

Treatment The treatment of choice is ursodeoxycholic acid at 13–15 mg/kg in divided dosing 3–4 times daily and may be increased up to a maximum of 2000 mg/d [4]. Close obstetric surveillance is recommended with weekly fetal heart rate cardiotocographic monitoring from 34-week gestation onwards. Delivery should be considered at 36–37 weeks. Other management strategies include rifampicin, vitamin K administration, topical emollients with 1–2% menthol, and antihistamines [1, 4].

Maternal and fetal prognosis Pruritus usually resolves within days to weeks and maternal prognosis is good, although the condition may present in future pregnancies and with oral contraception. ICP poses a risk of preeclampsia and gestational diabetes in expectant mothers.

Preterm birth, meconium-stained amniotic fluid, neonatal respiratory distress syndrome, and intrauterine fetal death (with a concentration of bile acids > 100 μmol/L) are the fetal adversities reported [5].

Atopic Eruption of Pregnancy (AEP)

It is the most common dermatosis in pregnancy and accounts for 50% of patients [4]. AEP patients either experience an exacerbation of atopic dermatitis in pregnancy (20% cases) or manifest atopic changes for the first time or after a long remission (80% cases) in childhood. The symptoms usually start early in the first or second trimester.

Etiology Downregulation of cellular immunity and decreased production of Th1 cytokines (IL-2, interferon-γ, IL-12), and upregulation of humoral immunity leading to increased secretion of Th2 cytokines (IL-4, IL-10) [4] is responsible for aggravation of pre-existing atopic eczema. To summarise, the dominant Th2 immune response results in atopic manifestations.

Clinical features Onset is usually early in gestation with 75% of cases presenting before the third trimester [4]. Mainly two types of clinical scenarios have been described: P-type AEP and E-type AEP. E-type is seen more commonly and presents as widespread eczematous changes frequently involving atopic sites such as the face, neck, and flexures of the limbs. In P-type, small erythematous papules are disseminated on the trunk and limbs. Typical prurigo nodules occur which are mostly located on the shins and arms. A key feature is severe dryness and atopic 'minor' features such as a tendency to nonspecific hand/foot dermatitis, nipple eczema, cheilitis, recurrent conjunctivitis, orbital darkening, and itch when sweating [6]. Irrespective of the subtype, scratching poses a significant risk of secondary cutaneous infections.

Diagnosis It is a diagnosis of exclusion and is mainly established clinically. Most cases have a background of atopy. 'Minor' atopic features form an important clue to diagnosis. Elevated serum IgE levels are found in 20–70% [4].

Treatment Treatment involves topical emollients, antihistamines, and narrow-band ultraviolet B (NBUVB) phototherapy. In resistant cases, oral steroids and azathioprine may be used. Secondary bacterial infections with streptococci or staphylococci necessitate antibiotics.

Maternal and Fetal Prognosis Maternal prognosis is good even in severe cases as treatment results in rapid resolution of skin lesions. It commonly recurs in subsequent pregnancies. The fetal prognosis is unaltered, however, the infant has a risk of developing atopic features later.

Features of pregnancy-specific dermatoses have been summarized in Table 4.

Other Pruritic Conditions in Pregnancy

Several non-pregnancy-related conditions are also responsible for pruritus in pregnancy. However, only the relevant

Table 4 Pregnancy-specific dermatoses and their characteristics

Condition	Onset	Characteristic clinical features	Diagnostic modality of choice	Treatment	Recurrence in subsequent pregnancy	Fetal prognosis
Pruritic urticarial papules and plaques of pregnancy (PUPPP)	Third trimester or immediately postpartum	Polymorphic lesions with periumbilical sparing	Clinical	Topical corticosteroids + antihistamines + emollients	No	Good
Pemphigoid gestationis	Third trimester and postpartum	Urticarial papules and plaques in the periumbilical region in pre-bullous stage + Tense blisters	DIF showing linear IgG and/or C3 deposit at the dermo-epidermal junction	Topical corticosteroids + antihistamines, prednisolone, azathioprine, rituximab, IVIG, dapsone,	Yes, presents earlier and severe	Preterm labor, intrauterine growth retardation, and neonatal pemphigoid gestationis
Intrahepatic cholestasis of pregnancy	Second and third trimester	Pruritus over the abdomen, palms, and soles without rash and jaundice starting 2–4 weeks after the onset of pruritus	Clinical features + serum bile acid levels > 10 µmol/l	Ursodeoxycholic acid	Yes	Preterm birth, meconium-stained amniotic fluid, neonatal respiratory distress syndrome, and intrauterine fetal death
Atopic eruption of pregnancy	Early getation		Clinical	Topical corticosteroids + antihistamines, prednisolone, Narrow-band UVB	Yes	Good

conditions have been discussed as per the scope of this article.

Dermatological Causes

Psoriasis in Pregnancy

Pustular psoriasis of pregnancy, also known as 'impetigo herpetiformis,' is a variant of psoriasis that has the potential for unfavorable outcomes for the mother and fetus. Conventionally considered to be a variant of pustular psoriasis, it is now considered by several experts as the 'fifth dermatosis' of pregnancy [7].

Etiology Recent studies have demonstrated the role of IL-1 and IL-36, which cause neutrophil chemotaxis and pustule formation [7]. Keratinocytes, neutrophils, and monocytes, tumor necrosis factor-α (TNF-α), and IL-17α also have a role in the pathogenesis [8]. Mutations of an antagonist to IL-1 family receptors (*IL36RN*) have been reported [9]. Hypoparathyroidism, hypocalcemia, stress, and infections are the proposed triggers of PPP [7].

Clinical features PPP usually presents in the last trimester or the postpartum period and resolves after delivery. Many cases have a personal or family history of psoriasis. The lesions are sterile pustules atop erythematous plaques involving flexures such as axillae, and inframammary folds, which later progress to involve other sites. Tongue, buccal mucosae, and esophageal involvement in the form of circinate or erosive lesions may be seen [10]. Lesions occur in crops and are accompanied by several systemic features such as fatigue, fever, diarrhea, acute kidney injury, and delirium. Erythrocyte sedimentation rate (ESR) and white blood cell counts are frequently elevated. The rash may progress to erythroderma in severe cases causing complications like fluid and electrolyte imbalances, thermoregulation disturbances, and sepsis.

Diagnosis Diagnosis is made clinically in collaboration with dermatologists.

Treatment Supportive measures, topical and systemic therapy, and removal of the provoking factors are the key components of the management and often require in-patient management. Treatment has been provided in Table 5.

Maternal and fetal prognosis Prognosis is poor with severe and longstanding disease. Cardiac or renal failure may cause maternal mortality in severe cases. It tends to reappear in future pregnancies, during menstruation, and with hormonal contraception. Stillbirth, neonatal death, or fetal abnormalities due to placental insufficiency are possible fetal complications.

Chronic plaque psoriasis is the most common subtype and has varied presentations including palmoplantar hyperkeratosis, erythematous papules, and plaques with silvery scales over extensors, and scalp. IL-6, C-reactive protein, and tumor necrosis factor-α have been found in maternal blood and can affect fetal health by causing low birth weight, prematurity, and stillbirth. Maternal consequences of the inflammation are pre-eclampsia, gestational hypertension, gestational diabetes, cesarean delivery, and spontaneous abortion [11]. Treatment of chronic plaque psoriasis is given in Table 5.

Eczema

Pregnancy appears to influence the course of eczema in most; in some, it improves and in many, it worsens [12]. Majority of the cases are seen in the first and second trimesters. Pregnancy favors a type 2 T helper cell response, which causes deterioration of eczema during pregnancy. Eczema does not affect the maternal or fetal outcome unless complicated by a secondary infection with herpes simplex. Eczema herpeticum is a herpes simplex infection characterized by widespread monomorphic papulovesicular lesions that are frequently accompanied by high-grade fever, and lymphadenopathy [13]. During pregnancy, there is a higher likelihood of life-threatening complications such as septicemia, meningitis, and encephalitis. In severe cases, premature delivery, intrauterine growth restriction, and miscarriage are possible fetal outcomes. Treatment with 5 to 10 mg/kg of intravenous acyclovir, 3 times daily, for 5 to 7 days is the recommended treatment. The treatment of eczema is given in Table 6.

Urticaria

Urticaria frequently presents during pregnancy.[3] It may remit during the third trimester but usually reappears postpartum. The lesions are transient, well-demarcated, superficial pink or pale swellings of the dermis and are known as 'wheals.' Of particular concern are contact urticaria and delayed pressure urticarias. In contact urticaria, the symptoms occur within minutes of contact with the culprit substance such as antiseptics, and latex which are frequently used during pregnancy. Sustained pressure stimuli during delivery may trigger an episode of delayed pressure urticaria where wheals appear after sustained pressure after a delay of 30 min–9 h [14]. Cetirizine and loratadine are the treatments of choice [15]. The recommendations for using antihistamines as per the Pregnancy and Lactation Labeling Rule (PLLR) are given in Table 7. Urticaria does not affect pregnancy and its outcomes.

Infections and Infestations

Infections are common during pregnancy. Commonly encountered pruritic infections that are important for obstetric practice have been discussed in this article.

(1) *Scabies* This is a mite infestation that presents as itchy papules in the interdigital spaces of hands and feet, axillae, areola, navel, waistline, buttocks, perianal region, groins, and ankle. Comma-like, tortuous tunnels and nocturnal exacerbation of itching are the hallmark presentations of scabies. Vesicles, crusting, and excoriation marks may appear as secondary lesions. The treatment of choice is topical 5% permethrin with a contact period of 8–12 h [16]. Repeat application after 1 week is beneficial. As the mode of transmission involves direct contact, scabies-infested lactating women should express their milk if they are not receiving treatment. 5% permethrin is safe for lactating mothers; however, breastfeeding should be withheld during the 8 h of topical application. Concomitant treatment

Table 5 Treatment of psoriasis in pregnancy

Treatment of pustular psoriasis in pregnancy	
Supportive measures	Maintenance of adequate ambient temperature, fluid replenishment
Topical therapy	Serially diluted topical corticosteroids over several days, topical antibiotics for treatment of infections, bland creams or lotions
Systemic therapy	1st line: Steroids are the treatment of choice (prednisolone at the dose of 30–40 mg/day, higher doses up to 60–80 mg/day can be used) Cyclosporine 2–3 mg/kg body weight/day has been used without adverse effects Infliximab (TNF alpha inhibitor) 5 mg/kg intravenous infusion (limit use to first 30 weeks of gestation) Narrow-band ultraviolet B (NBUVB), mainly as an adjuvant therapy
Treatment of chronic plaque psoriasis	
Topical	Corticosteroids (First line)
Systemic	Cetirizine and Loratadine Biologics (TNF alpha inhibitors) in collaboration with dermatologists

of family members is essential and washing clothes and bed linens with hot water used in the previous 24 h is helpful. Itching tends to persist for up to several weeks in some cases emollients are helpful. It is beneficial to have patients machine wash with hot water any clothes and bed linens used in the previous 24 h.

(2) *Chickenpox* Chickenpox is caused by the varicella-zoster virus. After a fever of 1–2 days, the lesions present as morbilliform (measles-like) erythema, followed by the development of papules and vesicles on an erythematous base and spread centrifugally with eventual crusting and scab formation. Mucosal involvement presents as erosions and vesicles. Accompanied pruritus may be significant and the presence of different stages of skin lesions is a characteristic finding. The maternal complications include pneumonia and secondary bacterial infection. Congenital varicella syndrome is a feared complication that presents with skin scarring, multiple neurological problems, and limb hypoplasia. If the infection was acquired during delivery, neonatal varicella infection can occur. The diagnosis is made clinically although the most reliable way to confirm the diagnosis is by PCR of vesicle fluid or a scraping taken from the base of a blister [17]. Primary varicella infection during pregnancy should be treated with intravenous acyclovir 10 mg/kg every 8 h along with zoster immune globulin (ZIG) to reduce the risk of fetal transmission and the disease severity [17]. ZIG should be given to neonates whose mothers develop varicella between the period from 7 days before to 7 days after delivery. Continued breastfeeding is recommended.

(3) *Pityriasis rosea* Caused by Human herpes virus (HHV)-6 and HHV-7, it presents with a herald patch over the thigh, upper arm, trunk, or neck followed by numerous pruritic, scaly, erythematous, round or oval plaques that range from 2 to 5 cm. Diagnosis is clinical and collarette of scales, herald patch, and predominantly truncal distribution of the lesions are the clues to diagnosis. Lesions appear in crops and may last up to 3 months. Several studies have reported adverse outcomes such as preterm labor and spontaneous abortion, especially after an infection during the first trimester. Treatment involves acyclovir 400 mg three times per day for 1 week [18].

(4) *Human Immunodeficiency virus (HIV) infection* HIV infection is associated with pruritus which may be secondary to antiretroviral therapy (ART), xerosis or idiopathic. Xerosis in HIV-affected individuals occurs secondary to reduced lipid content, low CD4 counts, and immune dysregulation [19]. Treatment involves adequate moisturization. Another entity associated with HIV is pruritic papular eruption (PPE) of HIV which presents as numerous 2–5 mm vesicles and papules on extensors and heals with hyperpigmentation. It is seen in immunosuppressed individuals with a CD4 count of $< 100/mm^3$. The management is challenging and NBUVB is considered as the treatment of choice [19]. Drug reactions, eosinophilic folliculitis, seborrheic dermatitis, dermatophytosis, and systemic diseases are the other underlying causes that cause pruritus during HIV infection.

(5) *Zika virus* It is caused by an RNA virus that is acquired after the bite of Aedes mosquitoes. Although most infections are asymptomatic, itchy macular or papular rash, mild fever, arthralgia, non-purulent conjunctivitis, myalgia, headache, and edema have been reported in symptomatic cases [20]. Fetal complications occur due to the infection of the placenta which causes hypoper-

Table 6 Treatment of eczema in pregnancy

Antepartum	General measures: avoidance of soap during acute flare Topical therapy: topical steroids + emollients (first line) Systemic therapy: ciclosporin (preferred second line agent), Azathioprine (second line) Miscellaneous: narrow-band Ultraviolet B phototherapy (Safest second line therapy)
Postpartum	Topical steroids (mild, moderate, or potent Azathioprine Narrow-band Ultraviolet B phototherapy
Nipple dermatitis-	Moderate to low potency topical steroids and emollients to be applied after breast feeding and washed off thoroughly before the next feed

Table 7 Treatment of Urticaria in pregnancy and lactation

Pregnancy	
First line	Cetirizine (preferred) Loratadine (preferred)
Refractory	Omalizumab 150 or 300 mg every 4 weeks Systemic corticosteroids (use at the lowest possible dose < 20 mg/kg for the shortest time)

fusion, fetal loss, intrauterine growth restriction, and neonatal infection. The risk of fetal transmission is highest in the first trimester and occurs irrespective of the symptoms in the mother. PCR is the most reliable investigation that confirms ZIKV infection although serology with ELISA is also helpful. Treatment is supportive with monthly monitoring of fetal well-being by ultrasound.

(6) *Dermatophytosis* It is a superficial infection caused by keratinophilic fungi [21]. The lesions are pruritic, erythematous, circular plaques with central clearing and usually present in the flexural areas. Safe topical agents in pregnancy include clotrimazole, terbinafine, ciclopirox, naftifine, and oxiconazole [21]. Systemic therapy during pregnancy and breastfeeding is best avoided. It is a contagious condition that spreads through fomites and direct contact. Hence, treatment of affected family members is vital in the prevention of re-infection.

Systemic Diseases

Pruritus is a troublesome symptom associated with several systemic conditions such as biliary diseases (due to elevated bile salts), chronic renal disease (due to raised urea), iron deficiency anemia, polycythemia vera, hypothyroidism, and diabetes mellitus (due to xerosis) [22]. It presents with secondary changes such as excoriation marks, lichenification, and features of secondary infection resulting from scratching and rubbing as there are no primary lesions. The treatment of pruritus in systemic diseases is given in Table 8.

Neurological

Notalgia paresthetica presents with burning pruritus and lichenification between the scapulae and may involve the scalp and neighboring areas. It is thought to arise secondary to root entrapment of primary dorsal rami of the spinal nerves from T2 to T6. Treatment options include topical capsaicin 0.025% cream and 8% patch [23].

Post-herpetic neuralgia presents in a dermatomal distribution 3 months after the vesicular rash, predominantly with pain but may also present with pruritus. Treatment involves topical capsaicin 0.025% cream and antihistamines.

Table 8 Treatment of pruritus in systemic diseases

Systemic disease	Treatment of pruritus
Chronic kidney disease	NBUVB
Hepatobiliary	Ondansetron
Hypothyroidism	Thyroid hormone, Emollients
Polycythemia vera	Cytoreductive therapy and interferon alfa
Iron deficiency anemia	Iron supplementation
Paraneoplastic pruritus	Pregabalin

Psychological

Prurigo nodularis is an itchy condition characterized by multiple itchy eroded papules, nodules, excoriation, and lichenification which results from neural sensitization leading to an 'itch-scratch' cycle [24]. It is associated with several conditions, but several experts have noted an association with stress that leads to scratching causing stress relief. Treatment includes emollients, topical steroids, and antihistamines.

Psychogenic pruritus can be localized or generalized. The treatment involves targeting lesional, emotional, and psychological components and is mainly done by psychologists and psychiatrists.

Treatment of Pruritus in Pregnancy

As per the USFDA and Pregnancy Lactation labeling rule (PLLR), loratadine, cetirizine, levocetirizine, desloratadine, and fexofenadine can be considered during pregnancy [25]. During lactation, second-generation antihistamines are recommended [25]. As per PLLR, drugs have not been assigned a category and the guidelines are still in the process of being adopted. Treatment recommendations for the management of pruritus have been summarized as per US FDA pregnancy risk categories in Table 2.

Conclusion

It is important to consider the disease burden of pruritus in the gestational population as it is widely prevalent and troublesome. Awareness of the underlying causes, pregnancy-specific and others, and their treatment is the first step to broadening the perspective of obstetricians for better diagnosis, management, and timely referral to respective specialties.

Declarations

Conflict of interest Nil.

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