Pruritus in Pregnancy and Its Management

Mark A. Bechtel, MD

KEYWORDS

• Pruritus • Pemphigoid gestationis • Polymorphic eruption of pregnancy
• Intrahepatic cholestasis of pregnancy • Atopic eruption of pregnancy

KEY POINTS

- Pemphigoid gestationis often involves the periumbilical region.
- Polymorphic eruption of pregnancy starts in the striae gravidarum.
- Intrahepatic cholestasis of pregnancy has no primary skin lesions, but may produce intense itching of the palms.
- Atopic eruption of pregnancy has the earliest onset during pregnancy, usually the first or second trimester.
- Pemphigoid gestationis has been associated with maternal autoimmune thyroid disease, and intrahepatic cholestasis of pregnancy with maternal hepatobiliary disease, gall stones, and hepatitis C.

Pruritus is the most common dermatologic complaint during pregnancy. It has been reported in 14% to 20% of pregnancies.[1] Most cases of pruritus are mild but some can be severe, interfering with sleep, producing mood changes, and affecting quality of life. The etiologic factors of pruritus during pregnancy can be directly related to pregnancy-specific dermatoses, allergic hypersensitivity reactions, and systemic diseases. Additional important causes of pruritus include atopic dermatitis, psoriasis, lichen sclerosus, lichen planus, vulvovaginal candidiasis, allergic and irritant contact dermatitis, and scabies.[2]

An underlying systemic cause of pruritus is important to consider if there is no specific cutaneous eruption. Considerations should include hepatic and renal disease, poorly controlled diabetes, and an underlying malignancy, such as lymphoma. Intense itching of the palms during the third trimester without a specific eruption may be an indication of intrahepatic cholestasis of pregnancy (ICP).[3] Pruritus during pregnancy in the setting of jaundice may indicate a more serious condition, such as hepatitis, acute fatty liver of pregnancy, severe ICP, or hyperbilirubinemia states.[1]

Many physiologic changes occur during pregnancy that can be exacerbate itching. There is abdominal stretching, edema of the legs, and xerosis. Important complex immunologic mechanisms at the maternal-fetal interface occur during pregnancy. The T helper (Th)-2-type profile predominates, whereas Th1 is downregulated. Important Th2 cytokines include interleukin (IL)-4, IL-5, IL-6. IL-10, IL-13, and transforming growth factor (TGF)-beta. Regulatory T cells, which suppress antigen-specific immune responses, are elevated during pregnancy. The change in the cytokine profile may exacerbate atopic dermatitis but improve psoriasis.[4] There are also fluctuating estrogen and progesterone levels.

Pregnancy-specific dermatoses are an important cause of pruritus during pregnancy and include ICP, atopic eruption of pregnancy (AEP), pemphigoid gestationis (PG), and polymorphic eruption of pregnancy (PEP). Some of the

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Division of Dermatology, Ohio State University College of Medicine, 540 Officenter Place, #240, Gahanna, OH 43230, USA
E-mail address: dermdocmab@aol.com

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disorders were reclassified, which can lead to confusion[5] (**Table 1**). Many of the pregnancy-specific dermatoses have distinct presentations of their rash and pruritus, as well as time of onset during pregnancy, which can help in diagnosis (**Fig. 1**, **Table 2**). **Box 1** presents skin rashes in genitals that can cause itch in pregnancy that are nonpregnancy associated.

PREGNANCY-SPECIFIC DERMATOSES

Polymorphic Eruption of Pregnancy

PEP was previously known as pruritic and urticarial papules and plaques of pregnancy (PUPPP syndrome). PEP is one of the most common dermatoses of pregnancy and occurs in approximately 1 in 200 pregnancies.[3] It typically presents in primigravidas late in the third trimester or immediately postpartum. Risk factors include multiple gestation pregnancies and increased maternal weight gain.[6] The pathophysiology of PEP is unknown but may be related to abdominal distention, immunologic factors, fetal cell microchimerism, and hormonal factors.[3] The onset of the eruption typically occurs late in the third trimester at the peak of stretching of abdominal skin. Many patients have multiple gestational pregnancies or excessive maternal weight gain, which maximizes abdominal stretching. A direct relationship with damage of the collagen fibers due to distension and overstretching of the skin is suspected.[7] This is supported by the onset of PEP within the striae gravidarum. One study showed that pregnant women with PEP had a significant reduction in serum cortisol levels, but normal levels of ß-human chorionic gonadotropin.[8] The rash initially presents in the striae distensae (**Fig. 2**) and then spreads to the trunk and proximal thighs. Typically, the rash spares the umbilicus, in contrast to PG. Clinically, the cutaneous lesions may be urticarial papules, eczematoid patches, targetoid lesions, vesicles, and bullae.[6]

Neonates do not develop skin lesions and the maternal and fetal prognosis is not affected.[9] Diagnosis of PEP is based on clinical presentation and history. The cutaneous histopathology is nonspecific and laboratory tests are unremarkable.

Treatment of PEP is symptomatic. Low-potency to mid-potency topical steroids are first-line treatment, along with bland emollients.[10] Oral antihistamines, such as chlorpheniramine and diphenhydramine, are first-line agents for pruritus. Loratadine remains the first choice and cetirizine the second choice among second-generation antihistamines.[11] Severe intractable pruritus can be treated with short courses of systemic steroids or ultraviolet B phototherapy.[8] Folic acid levels should be monitored during phototherapy and avoidance of high heat is advisable.[11] Prednisone is the preferred systemic steroid because placental enzymes limit passage to the embryo.[11] In some cases of PEP unresponsive to other treatments, early delivery may be a treatment of last resort.[12]

Table 1
Diagnostic features of the pregnancy-specific dermatoses

Condition	Onset	Clinical Features	Diagnosis	Treatment
ICP	Late second or third trimester	Pruritus, especially palms No primary skin lesions	Total bile acids >11 μmol/L	Ursodeoxy cholic acid 15 mg/kg/d or 1 gm
PEP	Third trimester or postpartum	Urticarial papules, plaques, vesicles, target lesions Initially involves striae gravidarum, spares umbilicus	No specific laboratory or skin biopsy findings, negative DIF	Topical or oral corticosteroids, antihistamines
PG	Second or third trimester or postpartum	Urticarial papules and bullae Umbilicus usually involved	Linear C_3 deposition along DEJ on DIF antibodies to BP 180	Topical or oral corticosteroids, antihistamines
AEP	First and second trimesters	Widespread eczematous excoriated papules, plaques, prurigo nodularis	Clinical appearance, elevated IgE	Emollients, topical steroids, light therapy, antihistamines

Abbreviations: BP, bullous pemphigoid; DEJ, dermal epidermal junction; DIF, direct immunofluorescence.