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The Specific Dermatoses of Pregnancy

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ABSTRACT:

Introduction and purpose: Pregnancy-specific dermatoses are a group of pruritic skin disorders that arise exclusively during pregnancy or the postpartum period. They encompass four primary conditions: atopic eruption of pregnancy (AEP), polymorphic eruption of pregnancy (PEP), intrahepatic cholestasis of pregnancy (ICP), and pemphigoid gestationis (PG). This review aims to provide a comprehensive analysis of their prevalence, clinical characteristics, underlying mechanisms, therapeutic strategies, and potential fetal risks.

Results: These dermatoses often present with overlapping clinical features, making accurate differential diagnosis crucial. While some pose minimal risk to the fetus, others, such as ICP and PG, are associated with significant complications, including preterm birth and stillbirth.

The underlying pathophysiology remains incompletely understood, with hormonal, immunological, genetic, and environmental factors playing key roles. Treatment strategies focus primarily on symptomatic relief, with topical corticosteroids and oral antihistamines being the mainstay of therapy, while ursodeoxycholic acid is the treatment of choice for ICP.

Conclusions: A multidisciplinary approach is essential for optimal maternal and fetal outcomes. Further research is needed to better understand the mechanisms and develop more effective treatment options for these conditions.

Keywords: pregnancy-specific dermatoses, atopic eruption of pregnancy, polymorphic eruption of pregnancy, intrahepatic cholestasis of pregnancy, pemphigoid gestationis

Introduction

There are several classifications of skin conditions that can occur during pregnancy. The most general classification categorizes these conditions into three main groups. The first group includes physiological changes resulting from hormonal fluctuations, such as hyperpigmentation, melasma, striae gravidarum, hirsutism, postpartum telogen effluvium, and vascular alterations like palmar erythema and hemorrhoids. The second group consists of pre-existing dermatological conditions that may be exacerbated by pregnancy. The third group comprises pregnancy-specific dermatoses [1].

This review focuses on pregnancy-specific dermatoses, a group of pruritic skin disorders that occur exclusively during pregnancy or the postpartum period. These conditions include four major entities: atopic eruption of pregnancy, polymorphic eruption of pregnancy, intrahepatic cholestasis of pregnancy, and pemphigoid gestationis. Due to overlapping clinical presentations, accurate differential diagnosis is essential. While some of these dermatoses pose minimal risk to the fetus, others are associated with significant complications. The pathophysiology of pregnancy-specific dermatoses remains incompletely understood, underscoring the need for further investigation [2]. This review aims to provide a comprehensive analysis of their

prevalence, clinical characteristics, underlying mechanisms, therapeutic strategies, and potential fetal risks.

1. Atopic eruption of pregnancy (AEP)

The concept of atopic eruption of pregnancy (AEP) was first introduced in 2005 by Christina M. Ambros-Rudolph et al., based on a retrospective study analyzing data from 505 pregnant women. The authors reviewed cases with concurrent clinical manifestations of EP (eczema in pregnancy), PP (prurigo of pregnancy) and PF (pruritic folliculitis of pregnancy) and proposed the term AEP to describe this complex clinical picture [3]. AEP is the most commonly diagnosed dermatosis during pregnancy, accounting for half of all cases of pregnancy dermatoses. In 75 % of cases, the symptoms of this condition appear before the start of the third trimester, which distinguishes it from other pregnancy-specific dermatoses that occur later in pregnancy. Approximately 80% of patients with AEP experience a de novo onset of atopic lesions during pregnancy, while the remaining 20% develop an exacerbation of pre-existing atopic dermatitis (AD) features [4]. AEP is often an idiopathic condition. However, individuals with a family history of AD have an increased risk of developing it [5].

The disease manifests in two clinical forms: the E-type (eczematous) and the P-type (papular/pruriginous). The E-type, accounting for 67% of AEP cases, is the more prevalent form and is characterized by eczematous lesions predominantly distributed on the face, neck, upper chest, flexural areas of the extremities. Patients often have a personal or family history of conditions such as atopy, asthma, or seasonal allergies. In contrast, the P-type, observed in the remaining cases, is characterized by concentrated pruritic erythematous papules disseminated predominantly on the trunk and extensor surfaces of the extremities. These two forms frequently coexist, resulting in generalized lesions across multiple regions of the body [3,6].

During pregnancy, immunological changes occur to prevent fetal rejection, including a shift from Th1- to Th2-mediated immunity. This shift results in a decrease in Th1 cells and their associated cytokines (e.g. IL-12, interferon gamma) and an increase in Th2 cells and their cytokines (e.g. IL-4, IL-10). Since AD is a Th2-driven condition, this adjustment can lead to an atopic exacerbation during pregnancy and manifestation of AEP [3].

The diagnosis of this dermatosis is primarily based on clinical presentation, although a skin biopsy may occasionally be performed to exclude other conditions [7]. Histopathological findings can reveal epidermal changes such as spongiosis, epidermal hyperplasia, and parakeratosis, but these are not specific to the disease [8]. Immunofluorescence test results are

negative, while laboratory tests may reveal elevated serum IgE levels, which were reported in 71% of AEP patients in the original study [3].

Treatment of AEP is symptomatic and tailored to the severity of the ongoing symptoms. In mild to moderate cases, the use of emollients containing 3-10% urea, topical antipruritic agents (1%-2% menthol, camphor, polidocanol), topical corticosteroids and oral H1 antihistamines (chlorpheniramine, diphenhydramine, loratadine) is recommended. In more severe cases, short-term oral corticosteroids such as prednisolone or prednisone (20 to 30 mg/day) may be used, although their use is contraindicated in the first trimester due to the risk of orofacial cleft in newborns. Narrowband UVB phototherapy can be used in the first trimester as an alternative to corticosteroids and may also be used in cases of recurrent symptoms after previous oral steroid treatment. In very severe and refractory cases, immunosuppressive agents such as cyclosporine or azathioprine may be considered, taking into account potential risks and benefits [9].

2. Polymorphic eruption of pregnancy (PEP)

Polymorphic eruption of pregnancy (PEP) also known as pruritic urticarial papules and plaques of pregnancy (PUPPP) is a benign, self-limited pruritic inflammatory skin condition that was first described by Lewley in 1979 [10,11]. The disease occurs in approximately 1:160 pregnancies and typically presents in the third trimester (especially in the last few weeks) or immediately postpartum. Risk factors include first pregnancy, excessive maternal weight gain, and multiple gestations [12].

The classic presentation involves the development of pruritic urticarial papules within the striae, typically sparing the periumbilical area. These papules gradually coalesce to form erythematous plaques. As the disease progresses, a variety of lesions may appear, including papulovesicles, microvesicles, urticarial plaques, annular or polycyclic wheals and occasionally small bullae. Lesions tend to spread to areas such as the buttocks, proximal thighs and back, with less frequent involvement of more distant sites such as the arms, legs, face and hands [13].

The exact pathogenesis of the condition remains unclear, but several hypotheses have been proposed. One theory suggests that it may result from an inflammatory response triggered by significant weight gain during the later stages of pregnancy, leading to excessive stretching of the abdominal skin and damage to the connective tissue in the striae, following the exposure of collagenic antigens. Hormonal factors are also thought to play a role, with studies reporting reduced serum cortisol levels in PEP patients. Additionally, multiple pregnancies are associated with higher levels of estrogen and progesterone. Some research has identified the presence of

progesterone receptors on keratinocytes in the skin lesions of affected patients, further supporting the hormonal hypothesis. Another theory posits that a substance released by the aging placenta could stimulate fibroblast proliferation. Lastly, it has been suggested that fetal cells may migrate to maternal skin and trigger an inflammatory reaction, adding another potential mechanism to explain the condition [13,14].

There are no specific histopathological changes associated with PEP. However, findings may include superficial or deep perivascular dermatitis with lymphohistiocytic vasculitis. Variable papillary edema and nuclear dust may also be observed. Eosinophils are present in 60-100% of cases, while focal spongiosis and parakeratosis are noted in approximately 33% of cases. Further epidermal changes include acanthosis, hyperkeratosis and intraepidermal vesicles. Importantly, no definitive histopathological feature distinguishes PEP from AEP [11]. Skin biopsy and direct immunofluorescence (DIF) are recommended when it is necessary to differentiate PEP from pemphigoid gestationis, due to the similarity between the urticarial phase of PEP and the clinical presentation of pemphigoid gestationis. However, the diagnosis of PEP is primarily based on the patient's history and clinical examination [15].

The condition poses no risk to either the mother or the fetus. It is self-limiting, with recurrence being very rare. Furthermore, skin lesions typically resolve within 7-10 days after delivery. Treatment primarily focuses on relieving pruritus, with options including emollients, antipruritic topical medications, moderately potent corticosteroids, or low doses of antihistamines (such as chlorpheniramine, loratadine, and cetirizine) [14,15]. In more severe cases, short-term oral prednisolone can be administered - 20-30 mg/day during pregnancy and up to 40 mg/day during breastfeeding. Additionally, UVB therapy has shown positive results in some cases [14].

3. Intrahepatic cholestasis of pregnancy (ICP)

Intrahepatic cholestasis of pregnancy (ICP) is known by several other names, including: cholestasis of pregnancy, obstetric cholestasis, recurrent jaundice of pregnancy, idiopathic jaundice of pregnancy, pruritus gravidarum, and icterus gravidarum [13]. This is the most common liver disorder associated with pregnancy, typically emerging in the third trimester and resolving after childbirth. It is marked by intense itching accompanied by elevated serum bile acid levels. The condition has a recurrence rate of 45-90% in subsequent pregnancies, often presenting in a more severe form [16,17]. The prevalence of this condition is estimated to range from 0.32% to 5.6% worldwide. The occurrence varies depending on ethnic groups,

geographical regions, seasons (more common in winter), as well as genetic and environmental factors [18]. Risk factors include multiple pregnancy, hepatitis C infection, advanced maternal age, in vitro fertilization, hepatobiliary disease, and a history of ICP. Risk factors for severe disease are smoking, a history of ICP in a previous pregnancy, a prior cholecystectomy, and a pre-existing diabetes mellitus [17,18]. The highest prevalence has been recorded in Southern Africa and Northern Europe. Interestingly, among the Mapuche Indians, cholestasis has been reported in over 27% of pregnancies, which is an exceptionally high rate compared to other populations [18].

The condition is primarily characterised by pruritus, which typically manifests after 30 weeks of gestation. The itching often starts on the palms and soles, but can spread to the whole body. It worsens at night and affects the psychological well-being of women, possibly leading to insomnia and even depression [16]. The severity of pruritus tends to increase as pregnancy progresses. A distinctive feature of ICP is the absence of primary skin lesions, which helps to distinguish it from other pregnancy-related dermatoses [18]. Secondary skin lesions such as excoriations, erosions and scabs develop as a result of scratching [7]. Notably, serum bile acid levels may not be elevated at the onset of pruritus and typically rise about three weeks after the first signs of skin symptoms [18]. Jaundice occurs in 10-15% of cases and usually develops within four weeks of the onset of pruritus. Other symptoms include nausea, vomiting, abdominal pain, dark urine and steatorrhoea, which results from fat malabsorption. Symptoms of ICP usually resolve within 2-3 weeks postpartum [16].

The exact pathogenesis of ICP remains unclear but is believed to result from a combination of genetic, environmental, and endocrine factors. Hormones, especially the metabolites of estrogen and progesterone interfere with the passage of bile acids through the hepatocytes. This may explain why ICP is more common in multiple pregnancies and later in pregnancy when hormone levels are higher. Genetic predisposition has been linked to mutations in the ABCB4 and ATP8B1 genes, which have been identified in some affected women. These mutations may impair bile acid transport and metabolism, increasing susceptibility to the condition. Additionally, environmental and dietary factors such as low selenium intake and vitamin D deficiency have been suggested as possible contributors, as there is a higher prevalence of ICP in the winter season, although their exact role is still debated [13].

Diagnosis is based on the presence of characteristic cholestatic pruritus and elevated bile acids >10 μmol/L. Mildly elevated liver enzymes such as AST, ALT and ALP may also be

present. Diagnosis is aided by the resolution of symptoms within 2-3 weeks postpartum. In the diagnostic process, it is necessary to exclude other potential causes that may lead to cholestasis and liver dysfunction [19]. The first-line treatment involves administering ursodeoxycholic acid (UDCA) orally at a dose of 300 mg 2–3 times per day or 10–16 mg/kg/day. UDCA reduces the concentration of bile acids in the blood, which helps alleviate itching. In 60% of patients, itching is significantly reduced, and in 40% it disappears completely. UDCA is safe for the fetus and well tolerated by patients. Since ICP can lead to a decrease in vitamin K levels, some specialists recommend vitamin K supplementation at a dose of 10 mg to prevent postpartum haemorrhage [16,18]. The disease is associated with many fetal complications such as preterm delivery, meconium staining of amniotic fluid, fetal distress, fetal bradycardia. The most severe complication is stillbirth, which occurs more frequently when serum bile acid levels exceed 40 μmol/L. It is believed that bile acids or toxic metabolites contribute to these fetal complications, but further research is needed to better understand the underlying mechanisms [19,20].

4. Pemphigoid gestationis (PG)

Pemphigoid gestationis (PG) is a rare autoimmune blistering disease that occurs specifically during pregnancy. It was previously known as herpes gestationis because of the similarity of the skin lesions. However, as there is no association with any active or prior herpes virus infection and the clinical and immunological features of the condition are similar to those of the pemphigoid group of diseases, the name was changed to pemphigoid gestationis [3,21]. The dermatosis typically develops during the second or third trimester of pregnancy, though there have been reports of cases occurring in the first trimester or postpartum. Its prevalence is approximately 1 in 60,000 pregnancies, and it is observed worldwide without any significant differences across ethnicities. Recurrences in subsequent pregnancies are reported in 33–55% of cases, often with an earlier onset and more severe symptoms [22].

The initial symptom of PG is intense pruritus, followed by the development of skin lesions. Early lesions present as urticarial papules and annular plaques, which progress to vesicles and tense bullae on an erythematous base. Dermatosis typically originates in the periumbilical region and later spreads to the rest of the abdomen and, in some cases, to the extremities, chest and back. The face and mucous membranes are usually spared. Vesicles and bullae may rupture, resulting in crust-covered erosions. The Nikolsky phenomenon (types I and II) may be positive. Systemic symptoms such as fever, fatigue, chills and shivering may occur. Psychological distress, particularly related to severe pruritus, has also been reported in some patients. The

condition is self-limiting, with skin lesions typically resolving within weeks to months postpartum. A remission phase often occurs in late pregnancy and in some cases an acute exacerbation of symptoms occurs immediately after delivery. However, this usually resolves spontaneously within four weeks. Persistent skin lesions lasting for several years are very rarely observed [22,23].

Studies have indicated a connection between the presence of MHC class II HLA antigens, specifically DR3 and DR4 in affected women, and the development of PG. The pathogenesis of the disease is related to the production of antibodies against collagen XVII (BP-180 or bullous pemphigoid antigen 2), which is also an autoantigen in bullous pemphigoid. BP-180 is an intercellular junction protein that connects the epidermis to the dermis. This protein is also found in fetal membranes and placental tissues of affected women and stimulates an immune response. The course of the disease results in the production of IgG class antibodies that mainly attack the epitopes in NC16A, the largest domain of the BP180. This is followed by a cross-reaction in which the autoantibodies bind to the maternal skin proteins, leading to tissue damage and blister formation [24,25].

The diagnosis of PG is based on clinicals symptoms, histology and direct immunofluorescence, which is a gold standard for establishing the diagnosis. DIF always shows linear C3 deposition and sometimes IgG deposits along the dermal-epidermal junction [24]. The histopathology of the skin is not specific for PG and varies depending on the severity and stage of the disease. In urticarial lesions, it may reveal perivascular infiltration consisting of lymphocytes, histiocytes, and eosinophils, along with edema in the upper and mid-dermis. To avoid skin biopsy, other complement-binding tests can be performed to detect circulating autoantibodies. These include indirect immunofluorescence (IIF) and enzyme-linked immunosorbent assay (ELISA). IIF identifies IgG autoantibodies directed against the basement membrane of the skin in 30%-100% of cases, while ELISA reveals circulating IgG antibodies against BP180 with a 94%–98% specificity and a 86%–97% sensitivity. Additionally, ELISA can be used to monitor the activity of the disease [22].

The treatment mainly focuses on relieving itching through the use of steroids and second-generation antihistamines, such as cetirizine, levocetirizine, and loratadine, which are considered safe during pregnancy. The use of mild or moderate topical corticosteroids is recommended during pregnancy. If these are not effective, potent or very potent topical corticosteroids may be used for a short duration. For more severe cases of PG, oral prednisolone,

up to 0.5 mg/kg/day, can be administered for a brief period. In some situations, plasmapheresis, immunoadsorption, and intravenous immunoglobulin G infusion can be applied. Although tetracycline, cyclophosphamide, azathioprine, dapsone, and rituximab have been used in cases of persistent skin lesions after delivery, these treatments are not recommended during pregnancy due to their potential toxicity to the fetus [25]. In PG, there is an increased risk of preterm birth and infants can be small-for-gestational-age. Due to the transfer of antibodies from the mother to the fetus, some newborns may develop mild blistering, which resolves untreated within a few days or weeks without any lasting consequences for the infant. Women with PG are more likely to develop autoimmune diseases such as Graves' disease, thyroiditis, and pernicious anemia. This is related to the presence of HLA-DR3 and DR4 in both PG and these autoimmune conditions [21].

Conclusions

Pregnancy-specific dermatoses can manifest at various stages of gestation, with diverse clinical presentations and distribution patterns, yet they share common features. The predominant symptom associated with these conditions is pruritus. The etiopathogenesis of pregnancy-related dermatoses is multifactorial, involving hormonal, genetic, environmental, and immunological factors. The management strategy is mainly aimed at symptomatic relief while prioritizing fetal safety. The primary therapeutic modalities include the administration of topical corticosteroids and oral antihistamines. In the case of ICP, ursodeoxycholic acid remains the treatment of choice. A multidisciplinary approach is crucial for accurate diagnosis and individualized management, ultimately optimizing maternal and fetal outcomes. Further research is warranted to enhance the understanding of these conditions and to develop more effective treatment strategies.

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Author's contribution:

Conceptualization, PP; methodology, PP; software, PP; check, PP; formal analysis, PP; investigation, PP; resources, PP; data curation, PP; writing - rough preparation, PP; writing - review and editing, PP; visualization, PP; supervision, PP; project administration, PP; All authors have read and agreed with the published version of the manuscript.

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References:

[1] Snarskaya ES, Olisova OY, Makatsariya AD, Kochergin NG, Radetskaya L, Bitsadze V, et al. Skin pathologies in pregnancy. J Perinat Med 2019;47:371–80. https://doi.org/10.1515/JPM-2018-0338.

[2] de Lourdes Ribeiro de Carvalho M, Magalhães GM, Leite HV. Update on specific dermatoses of pregnancy. Rev Assoc Med Bras (1992) 2023;69. https://doi.org/10.1590/1806-9282.2023S109.

[3] Ambros-Rudolph CM, Müllegger RR, Vaughan-Jones SA, Kerl H, Black MM. The specific dermatoses of pregnancy revisited and reclassified: Results of a retrospective two-center study on 505 pregnant patients. J Am Acad Dermatol 2006;54:395–404. https://doi.org/10.1016/J.JAAD.2005.12.012.

[4] Balakirski G, Novak N. Atopic dermatitis and pregnancy. J Allergy Clin Immunol 2022;149:1185–94. https://doi.org/10.1016/J.JACI.2022.01.010.

[5] Vaughan Jones SA, Hern S, Nelson-Piercy C, Seed PT, Black MM. A prospective study of 200 women with dermatoses of pregnancy correlating clinical findings with hormonal and immunopathological profiles. Br J Dermatol 1999;141:71–81. https://doi.org/10.1046/J.1365-2133.1999.02923.X.

[6] Himeles JR, Pomeranz MK. Recognizing, Diagnosing, and Managing Pregnancy Dermatoses. Obstetrics and Gynecology 2022;140:679–95. https://doi.org/10.1097/AOG.0000000000004938.

[7] Himeles JR, Pomeranz MK. Recognizing, Diagnosing, and Managing Pregnancy Dermatoses. Obstetrics and Gynecology 2022;140:679–95. https://doi.org/10.1097/AOG.0000000000004938.

[8] Massone C, Cerroni L, Heidrun N, Brunasso AMG, Nunzi E, Gulia A, et al. Histopathological diagnosis of atopic eruption of pregnancy and polymorphic eruption of pregnancy: a study on 41 cases. Am J Dermatopathol 2014;36:812–21. https://doi.org/10.1097/DAD.0000000000000067.

[9] Roth MM, Cristodor P, Kroumpouzos G. Prurigo, pruritic folliculitis, and atopic eruption of pregnancy: Facts and controversies. Clin Dermatol 2016;34:392–400. https://doi.org/10.1016/J.CLINDERMATOL.2016.02.012.

[10] Lawley TJ, Hertz K, Wade T, Ackerman A, Katz S. Pruritic urticarial papules and plaques of pregnancy. JAMA 1979:1696–9.

[11] Taylor D, Pappo E, Aronson IK. Polymorphic eruption of pregnancy. Clin Dermatol 2016;34:383–91. https://doi.org/10.1016/J.CLINDERMATOL.2016.02.011.

[12] Ambros-Rudolph CM. Dermatoses of pregnancy - clues to diagnosis, fetal risk and therapy. Ann Dermatol 2011;23:265–75. https://doi.org/10.5021/AD.2011.23.3.265.
[13] Roth MM. Pregnancy dermatoses: diagnosis, management, and controversies. Am J Clin Dermatol 2011;12:25–41. https://doi.org/10.2165/11532010-000000000-00000.
[14] Petropoulou H, Georgala S, Katsambas AD. Polymorphic eruption of pregnancy. Int J Dermatol 2006;45:642–8. https://doi.org/10.1111/J.1365-4632.2006.02715.X.
[15] Zejnullahu VA, Zejnullahu VA. Polymorphic eruption of pregnancy. Dermatol Reports 2022;15. https://doi.org/10.4081/DR.2022.9546.
[16] Piechota J, Jelski W. Intrahepatic Cholestasis in Pregnancy: Review of the Literature. J Clin Med 2020;9. https://doi.org/10.3390/JCM9051361.
[17] Mashburn S, Schleckman E, Cackovic P, Shellhaas C, Rood KM, Ma'ayeh M. Intrahepatic cholestasis of pregnancy: risk factors for severe disease. J Matern Fetal Neonatal Med 2022;35:8566–70. https://doi.org/10.1080/14767058.2021.1988924.
[18] Smith DD, Rood KM. Intrahepatic Cholestasis of Pregnancy. Clin Obstet Gynecol 2020;63:134–51. https://doi.org/10.1097/GRF.0000000000000495.
[19] Pusl T, Beuers U. Intrahepatic cholestasis of pregnancy. Orphanet J Rare Dis 2007;2. https://doi.org/10.1186/1750-1172-2-26.
[20] Williamson C, Geenes V. Intrahepatic cholestasis of pregnancy. Obstetrics and Gynecology 2014;124:120–33. https://doi.org/10.1097/AOG.0000000000000346.
[21] Intong LRA, Murrell DF. Pemphigoid gestationis: pathogenesis and clinical features. Dermatol Clin 2011;29:447–52. https://doi.org/10.1016/J.DET.2011.03.002.
[22] Sävervall C, Sand FL, Thomsen SF. Pemphigoid gestationis: current perspectives. Clin Cosmet Investig Dermatol 2017;10:441–9. https://doi.org/10.2147/CCID.S128144.
[23] Lipozenčić J, Ljubojevic S, Bukvić-Mokos Z. Pemphigoid gestationis. Clin Dermatol 2012;30:51–5. https://doi.org/10.1016/J.CLINDERMATOL.2011.03.009.
[24] Cohen S, Strowd LC, Pichardo RO. Pemphigoid gestationis: a case series and review of the literature. J Dermatolog Treat 2018;29:815–8. https://doi.org/10.1080/09546634.2018.1459034.
[25] Huilaja L, Mäkikallio K, Tasanen K. Gestational pemphigoid. Orphanet J Rare Dis 2014;9. https://doi.org/10.1186/S13023-014-0136-2.