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Dermatoses of pregnancy: Nomenclature, misnomers, and myths

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Abstract The most recent reclassification of dermatoses of pregnancy includes polymorphic eruption of pregnancy, atopic eruption of pregnancy, and pemphigoid gestationis; intrahepatic cholestasis of pregnancy, strictly not a dermatosis, was included in specific dermatoses of pregnancy for working purposes. Another dermatosis, pustular psoriasis of pregnancy, could be included for similar reasons. The nomenclature of these pregnancy-specific eruptions has been revised several times, generating potential confusion among practitioners. Clouding the picture further are misnomers that have been used to describe dermatoses of pregnancy. In addition, several cutaneous conditions that are associated with, but not specific to, pregnancy, have been misunderstood, which has resulted in certain myths among patients and physicians. In this contribution, we describe how the nomenclature of each dermatosis of pregnancy has evolved to fit the current classification scheme. We then identify several misnomers that have generated confusion within the scheme. Finally, we debunk several myths that have developed around cutaneous conditions outside of this scheme, in both mother and newborn.

Introduction

The four well-established dermatoses of pregnancy are polymorphic eruption of pregnancy (PEP), atopic eruption of pregnancy (AEP), pemphigoid gestationis (PG), and intrahepatic cholestasis of pregnancy (ICP) (Table 1).[1] A potential fifth dermatosis, pustular psoriasis of pregnancy (PPP), was formerly part of the original categorization of the dermatoses of pregnancy but was dropped from the scheme by Holmes and Black in 1983 (Table 2)[2]; however, some authors still consider PPP as another disease within the current scheme.[1,3,4] The nomenclature and classification system of these pregnancy-specific eruptions, particularly PEP, AEP, and PPP, have been revised several times, generating potential confusion among practitioners. Generating further confusion are several misnomers, historic and modern, that have been used to describe dermatoses of pregnancy.

Many factors contribute to the paucity of large prospective studies on dermatoses of pregnancy. This lack of solid data on the nature of pregnancy dermatoses has increased confusion

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Table 1 Classification schemes of specific dermatoses of pregnancy

Author	Year	Evolution of the classification scheme of specific dermatoses of pregnancy
Holmes and Black	1983	PG, PEP, PP, PFP
Shornick	1998	PG, PEP, PP *, ICP
Ambros-Rudolph et al.	2006	AEP †, PG, PEP, ICP

AEP, atopic eruption of pregnancy; *ICP*, intrahepatic cholestasis of pregnancy; *PEP*, polymorphic eruption of pregnancy; *PG*, pemphigoid gestationis; *PP*, prurigo of pregnancy.

* PFP was categorized under PP.

† PP and PFP were categorized under AEP.

and allowed certain myths to be propagated among patients and physicians.

More research is needed in common skin problems, such as *striae gravidarum*, and controversial topics, such as the effect of lactation in reducing the risk of infantile atopic dermatitis. In this contribution, we describe how the original nomenclature of each dermatosis of pregnancy has been modified to fit the current classification scheme. We then identify several misnomers that have generated confusion within the scheme. Finally, we present the historical perspectives of myths that have developed around cutaneous conditions that are not included in specific dermatoses.

Dermatoses of pregnancy: Nomenclature and classification

To better understand the current nomenclature, it is useful to decipher how and why the names have evolved (Table 2). In the following sections, we shall trace the evolution of terminology from the early literature on dermatoses of pregnancy, which centered on morphologic description—urticarial eruptions and papular and prurigo-type eruptions, as previously highlighted in this issue.[5]

Urticarial eruptions

In 1931, Gross first described PEP (synonymously known as pruritic urticarial papules and plaques of pregnancy) as *erythema multiforme of pregnancy*[6] to reflect the targetoid lesions observed in pregnant patients. Approximately 30 years later, the condition was renamed *toxemic rash of pregnancy* and was further characterized as erythematous urticarial papules and plaques associated with severe pruritus, which were found on the abdomen, thighs, and buttocks.[7] In 1968, Nurse renamed the condition *late onset prurigo of pregnancy* to underscore how the eruption tends to occur in the third trimester of pregnancy.[8]

Shortly thereafter, in 1979, a case series was published of seven pregnant patients with late-onset, erythematous papules and urticarial plaques, which generally responded to topical and oral corticosteroids.[9] Unlike previous authors, these authors obtained biopsies and laboratory data from these women. Based on this additional information and slight differences on skin examination between his series of seven women and those diagnosed with toxemic rash of pregnancy (in whom urticarial lesions were less frequent and crusting was more common), the group renamed this eruption *pruritic urticarial papules and plaques of pregnancy* (PUPPP).[9]

Although we now understand that toxemic rash of pregnancy and PUPPP describe the same condition, *PUPPP* has become the accepted term and is used predominantly in the United States and Australia. A synonym for PUPPP is *polymorphic eruption of pregnancy* (most commonly used outside of the United States), first introduced by Holmes and Black in 1983 to better describe the wide variety of observed morphologies, including eczematous lesions, which can include excoriated papules, plaques, and crust (22% of patients), targetoid lesions (6%), polycyclic erythema (6%), and vesicles (17%).[2,10] Although it would be favorable to use only one of these terms to describe this dermatosis, both terms are used widely and interchangeably.

Table 2 Nomenclature of specific dermatoses of pregnancy

Current classification	Historic synonyms
Polymorphic eruption of pregnancy (PEP)	• Pruritic urticarial papules and plaques of pregnancy * • Toxic erythema of pregnancy • Late onset prurigo of pregnancy • (Bourne's) toxemic rash of pregnancy • Erythema multiforme of pregnancy • Linear IgM dermatosis of pregnancy †
Atopic eruption of pregnancy (AEP)	• Prurigo of pregnancy • Prurigo gestationis (of Besnier) • (Nurse's) early-onset prurigo of pregnancy • Papular dermatitis of pregnancy • Pruritic folliculitis of pregnancy • Eczema in pregnancy • Linear IgM dermatosis of pregnancy †
Pemphigoid gestationis (PG)	• Herpes gestationis
Intrahepatic cholestasis of pregnancy (ICP)	• Cholestasis of pregnancy • Pruritus/prurigo gravidarum • Obstetric cholestasis/hepatosis • (Idiopathic) jaundice of pregnancy • Hepatosis gestationalis • Icterus gravidarum
Pustular psoriasis of pregnancy (PPP)	• Impetigo herpetiformis • Generalized pustular psoriasis in pregnancy

IgM, immunoglobulin M.

* Pruritic urticarial papules and plaques of pregnancy (PUPPP) is still currently used in the United States as a synonym of PEP.

† Linear IgM dermatosis of pregnancy has been categorized under PP, of the current categorization AEP, as well as under PEP.

Papular and prurigo-type eruptions

In 1904, a study categorized all of the dermatoses of pregnancy that lacked blisters as *prurigo gestationis* (later renamed *prurigo gestationis of Besnier*).[11,12] In 1962, another study described *papular dermatitis of pregnancy* (PDP), which is now recognized as a more widespread form of prurigo gestationis. *Papular dermatitis of pregnancy* was reported as a generalized pruritic papular eruption that occurred throughout pregnancy and was associated with a high fetal mortality rate (30%) and hormonal abnormalities (elevated serum and urine human β-chorionic gonadotropin levels and reduced serum estradiol and hydrocortisone levels).[13] Shortly after this original report, Nurse introduced the term *early-onset prurigo of pregnancy* to describe 40 patients who developed eruptions during pregnancy that lacked blisters but presented differently from PDP, specifically in the number and types of papules, likelihood of recurrence, and response to steroids.[8]

Another pruritic dermatosis, *linear immunoglobulin M (IgM) dermatosis of pregnancy*, was introduced in 1988.[14] The authors described a patient in the third trimester of pregnancy who developed red follicular papules on the abdomen and extremities; direct immunofluorescence exam from obtained skin biopsies showed dense linear IgM deposits at the dermal-epidermal junction[14]; however, most authors consider it currently as a "ghost entity" included in the spectrum of prurigo of pregnancy (reclassified under AEP).

Subsequently, Holmes and Black reevaluated the patients previously described by Besnier, Nurse, and Spangler and concluded that there was not enough clinical or biochemical evidence to classify these conditions as separate.[2] As a result, they used the term *prurigo of pregnancy* (PP) to describe prurigo gestationis, PDP, and early-onset prurigo of pregnancy under one umbrella term.[2] In addition, after the observation that linear IgM deposits at the dermoepidermal junction occurred in healthy pregnant women and as a nonspecific finding in several cutaneous conditions (urticaria, leukocytoclastic vasculitis, pigmented purpuric dermatosis, and others) the dermatosis has been classified under PP by some authors and PEP by others.[15–17] After the terminology of PP was coined, PDP was completely removed from this classification, when a large prospective study of 200 women with dermatoses of pregnancy found no cases that were consistent with Spangler's definition of PDP.[18] In particular, the authors did not observe the hormonal abnormalities previously described by Spangler and reported an overall fetal mortality <10% associated with any dermatosis of pregnancy (compared with the 30% risk of fetal mortality associated with PDP quoted by Spangler).[18]

In 2006, 505 pregnant patients who developed generalized pruritic skin conditions were studied, and the authors placed PP under the new umbrella entity of *atopic eruption of pregnancy*.[19] This broader category includes also pruritic folliculitis of pregnancy (PFP) and eczema in pregnancy. The group found considerable clinical and histopathologic overlap between the three conditions[19]; however, several PP patients in the study satisfied only minor criteria of atopy.[20] As noted by several authors, not all PP and PFP patients satisfy criteria for atopy.[20–22] Of note, PFP was first used in 1981 by Zoberman and Farmer[23] to classify six patients with widespread pruritic folliculitis and later used in a classification scheme proposed by Holmes and Black in 1983 (Table 1). Most recently, the authors that introduced AEP suggested that only the papular, not the acneform, PFP presentations should be classified under AEP. They suggested using the term *acneform eruption* for cases of acneform PFP.[24] Unfortunately, there are no data at present on acneform PFP, which makes consensus on this nomenclature challenging.

Pemphigoid gestationis

Like PEP and AEP, pemphigoid gestationis is a modern classification that exists after improvement of our understanding of the disease pathogenesis. John Laws Milton (1820–1898) originally named the dermatosis *herpes gestationis* (HG) in 1872 to describe the herpetic-like blisters observed during pregnancy. After more recent advances found substantial clinical and immunologic similarities between HG and the pemphigoid group of blistering diseases, the disease was reclassified as PG.[2,19] Further support for the term *PG* stems from the understanding that the dermatosis is not related to or associated with any active or previous herpes infection.[25]

Intrahepatic cholestasis of pregnancy

The most recent reclassification of ICP remains largely unchanged from prior nomenclature. Intrahepatic cholestasis of pregnancy, a liver disease of pregnancy associated with raised serum bile acids and increased rates of adverse fetal outcomes, was first described by Johann Friedrich Ahlfeld (1843–1929) in 1883.[26] Since its original description by Ahfeld as recurrent jaundice in pregnancy, the name *ICP* is still used today to describe the condition; however, several other names have been used over the years to describe ICP, including *jaundice in pregnancy, idiopathic jaundice of pregnancy, obstetric hepatosis, hepatosis gestationalis,* and *obstetric cholestasis*.[26]

In 1998, Shornick added *intrahepatic cholestasis of pregnancy* to the contemporary classification scheme.[27] Although ICP lacks primary lesions, it was added to specific dermatoses for working purposes, which also promotes its consideration in physicians' differential diagnoses of excoriated papules in pregnant women.[27] Inclusion of ICP in specific dermatoses of pregnancy helps raise awareness of the condition and reduces misdiagnosis, especially because the condition is associated with significant fetal risks.[28]

Pustular psoriasis of pregnancy

In addition to PG, PEP, ICP, and AEP, a fifth dermatosis, *pustular psoriasis of pregnancy* (synonymous with *impetigo herpetiformis*), has been proposed as a specific dermatosis

of pregnancy. *Impetigo herpetiformis* was first described in 1872 by Ferdinand von Hebra (1816–1880), who reported a pustular eruption occurring in five pregnant and puerperal women, four of whom died.[29] Moritz Kaposi (1837–1902) described six patients with a similar clinical presentation in 1883.[30,31] Since these reports, several patients have been reported to develop *impetigo herpetiformis*, typically in the second half of pregnancy with resolution after delivery.[32–35] The dermatosis has been associated with significant fetal risk, including stillbirth, neonatal death, and fetal abnormalities.[36,37]

The term *impetigo herpetiformis* was replaced with PPP after several patients with *impetigo herpetiformis* were noted to have a family history of pustular psoriasis.[37] Considerable clinical and pathologic overlap was also observed between the two diseases.[38–40] In fact, additional evidence suggests that PPP is a variant of generalized pustular psoriasis, rather than a distinct clinical entity[38,41,42]; therefore, its inclusion in the classification scheme of dermatoses of pregnancy has created controversy.

Although we are in agreement that PPP is likely a variant of generalized pustular psoriasis, we believe that it may be included in specific dermatoses of pregnancy for working purposes, because delayed or missed diagnosis may pose significant, life-threatening risk to mother and fetus.[43] In this way, a justification for including PPP in the classification is no different from inclusion of ICP.

Misnomers

Further adding to the confusion of the nomenclature for cutaneous conditions in pregnancy are several historic and modern misnomers (Table 3). As a result, we enumerate four of the most widely used misnomers.

As described earlier, PG was originally termed *herpes gestationis*; however, because there is no relationship between herpes gestationis and infection with the herpes simplex virus, HG is a misnomer for the condition. Similarly, PPP was previously described as impetigo herpetiformis, which is a double misnomer incorrectly assuming a relationship both between bacterial impetigo and the herpes simplex virus with this dermatosis of pregnancy. These misnomers contribute to confusion for patients and clinicians.

Misnomers have also been used to describe lesions associated with but not specific to pregnancy. *Molluscum fibrosum gravidarum*, which corresponds to acrochordons that develop or grow during pregnancy, is not related to viral molluscum. Similarly, *granuloma gravidarum*, synonymous with pyogenic granuloma, does not appropriately describe the vascular growth underlying this skin condition.

Table 3 Misnomers used to describe dermatoses of pregnancy

Dermatosis	Why the dermatosis is classified as a misnomer
Herpes gestationis	• No relationship with herpes simplex virus.
Impetigo herpetiformis	• No relationship with bacterial impetigo or the herpes simplex virus.
Molluscum fibrosum gravidarum	• No relationship with viral molluscum.
Granuloma gravidarum	• *Granuloma gravidarum* does not describe the vascular growth underlying this skin condition.

Debunking myths about pregnancy and lactation

Next we elaborate on myths that have developed around cutaneous conditions during pregnancy and lactation. We also discuss the effects of maternal diet during pregnancy and breastfeeding on the risk of atopic dermatitis in the infant.

Myth 1: There are established therapies for stretch mark prevention

Striae distensae occur in up to 86% of pregnant women, a phenomenon termed *striae gravidarum* (SG).[44] The development of severe SG has been found to have a significant negative psychologic impact in pregnant women.[45] Rapid fluctuations in weight and obesity have been associated with the development of *striae distensae*[44]; however, previous studies have not found significant correlations between SG and maternal weight gain during pregnancy, baseline maternal body mass index, or infant birth weight.[44,46,47] As a result, much of the focus for SG prophylaxis has shifted away from lifestyle modifications and toward topical preparations. A review of topical therapies for the prevention of SG, including centella, almond oil, hyaluronic acid, tretinoin, cocoa butter, and olive oil, was recently published.[48] This review included 17 studies on the efficacy of topical preparations for SG prevention, and the authors concluded that there was evidence (although limited) to suggest that centella, hyaluronic acid, and massage with bitter almond oil may prevent SG; cocoa butter and olive oil were not associated with the prevention of SG. Also, tretinoin has not been sufficiently studied during pregnancy to draw conclusions.[48–50] A Cochrane review with stricter inclusion criteria than this review (randomized and quasirandomized controlled trials with a comparator group) included 800 women from 6 studies and did not find evidence to support the use of any topical preparation for the prevention of SG.[51]

Myth 2: Breastfeeding reduces the severity of postpartum pemphigoid gestationis

PG typically develops in the second or third trimester, and approximately 75% of patients experience a flare of disease at

delivery or postpartum.[19,52] Mean duration of these exacerbations is approximately 28 weeks, but postpartum disease has been reported to last for years in some rare cases[53,54]; furthermore, the initial onset of PG may occur in the postpartum period (usually within hours after delivery) in 14-25% of patients.[53,55] Breastfeeding has previously been suggested as a means of hastening resolution of postpartum PG.[54]

In a 1983 report of patients who developed PG, the average postpartum duration was shorter in those who breastfed. Bullous lesions resolved on average by 5 weeks, versus 35 weeks in nonbreastfeeders. The urticarial eruption resolved sooner in breastfeeding women, at 24 weeks, versus nonbreastfeeders at 68 weeks.[54] The study had a small sample size of 25 women, 13 of whom breastfed, and did not allow for disease severity in the analysis. In 1990, another study also found that breastfeeding might help reduce duration of postpartum PG[56]; however, no recent studies have evaluated the effects of breastfeeding on PG. Although breastfeeding has a multitude of other benefits, we do not believe that there is enough evidence to indicate a reduction in the duration of postpartum PG.

Myth 3: Breastfeeding can prevent infantile atopic dermatitis

Breastfeeding can modestly reduce the incidence of infantile atopic dermatitis (AD) in a select group of high-risk infants.[57] High-risk infants are defined as those who have a first-degree relative with AD. In these children, there is strong evidence to suggest that breastfeeding during the first 4 months of life can reduce risk of AD by up to 33%.[58–60] This risk is not altered whether the child is exclusively breastfed for the first 6 months or whether the infant's diet is supplemented with solid foods or formula; however, the use of hydrolyzed formulas rather than intact cow's milk may delay or prevent risk of developing AD in these infants.[59–61] In contrast to high-risk infants, there is no risk reduction for the development of AD with breastfeeding in infants without risk factors for AD.[57]

Myth 4: Maternal diet may have an effect on the development of infantile atopic dermatitis

Maternal dietary modifications in pregnancy and lactation have also been studied in relation to the development of infantile AD. A Cochrane review involving 952 patients found no protective effect of maternal dietary antigen avoidance (cow's milk, eggs, peanuts, fish, and chocolate) during pregnancy on the development of AD in the first 18 months of life of high-risk infants.[62] In fact, antigen avoidance may be associated with an increased risk of preterm birth and decreased mean birth weight.[63,64] With regard to antigen avoidance during breastfeeding, two trials were unable to find any protective effect on the incidence of AD during the first 18 months of life.[65,66]

Conclusions

An evolving understanding of clinical and histopathologic features of dermatoses of pregnancy over the years has led to several classifications and revised definitions of these conditions. Even today, the classification scheme is debated by dermatologists and obstetricians. We believe that creating a clear classification scheme for dermatoses of pregnancy and avoiding the use of misnomers are vital to improving the diagnosis and management of these diseases.

Several myths about dermatologic disease in pregnancy and lactation have also developed due to a lack of substantial evidence. By analyzing the available data, we addressed some of these myths, including *striae gravidarum* prevention and the effects of breastfeeding on postpartum pemphigoid gestationis and prevention of infantile atopic dermatitis. Also, we discussed the role of maternal diet during pregnancy in the development of infantile atopic dermatitis. Although performing research in pregnancy is challenging due to ethical and other constraints, it is imperative to perform further studies to address gaps in knowledge.

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