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# Pemphigoid Gestationis - literature review

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**Abstract:**

**Introduction and Objective**: Pemphigoid gestationis (PG) is a rare, self-limiting autoimmune disorder, typically appearing in the second or third trimester of pregnancy. It can lead to postpartum exacerbations, and studies suggest an increased risk of preterm birth and the development of Graves' disease in the mother. The aim of this article is to gather the latest information on pemphigoid gestationis.

**Review Methods**: A review of studies available in the PubMed database was conducted using the keywords "pemphigoid gestationis" and "dermatoses of pregnancy" in order to find publications from the last 8 years.

**Brief Description of the State of Knowledge**: Pemphigoid gestationis (PG) is characterized by the production of IgG1 antibodies against bullous pemphigoid antigen 180 (BP180).

The primary symptom of PG is intense itching, which often occurs before skin lesions develop. Diagnosis is typically confirmed through direct immunofluorescence (DIF) of perilesional skin. Treatment focuses on relieving itching and preventing new blister formation, usually starting with topical corticosteroids for mild cases, while systemic corticosteroids are reserved for more severe presentations. Recent research indicates that biologic therapies may also offer effective management options for PG.

**Summary**: The authors highlight the need for further research due to the limited sample sizes in existing studies, as well as the potential complications during pregnancy and long-term health effects.

**Keywords:** pemphigoid gestationis, skin lesions, dermatoses of pregnancy

## Introduction

Pemphigoid gestationis (PG) is a relatively rare subepidermal bullous dermatosis with an autoimmune basis, and in most cases, it is self-limiting [1]. Historically, the disease was referred to as herpes gestationis due to the blistering nature of the lesions; however, because it is not associated with the Herpes virus, its name was changed to the current one [2]. PG typically begins in the second or third trimester of pregnancy, although rarer cases have been reported in the first trimester and the postpartum period [3]. The condition tends to recur in subsequent pregnancies, occurring in at least 30% of cases, often with a more severe course [2]. Although the disease generally follows a self-limiting course, postpartum exacerbations are common, and some studies suggest that PG is associated with risks such as preterm birth and the development of Graves' disease in the mother [3].

## Objective

The aim of this article is to gather the latest information regarding pemphigoid gestationis.

## Methods

A review of studies available on the PubMed platform (https://pubmed.ncbi.nlm.nih.gov/) was conducted, including articles with free full-text access that used the keywords "pemphigoid gestationis" and "dermatoses of pregnancy." The scope of the review was limited to publications from the last 8 years.

## Epidemiology

The available literature offers limited information on the incidence of pemphigoid gestationis (PG). Population studies conducted in France, Kuwait, Iran, and Germany estimate that the disease occurs at a rate of approximately 0.5 to 2.0 cases per million women [2,3,4]. According to Stefaniak et al. [5], PG occurs in 1 out of 2,000 to 60,000 pregnancies, and its prevalence correlates with the presence of human leukocyte antigen (HLA) haplotypes DR3 and DR4 in the population. It is worth noting that PG is the third most common bullous dermatosis in pregnancy [6].

According to several authors, PG most frequently appears in the second or third trimester of pregnancy [1,2,6].

The number of studies providing detailed data on this topic is limited, especially newer publications or those that assessed larger patient groups, likely due to the rarity of the condition. The largest such analysis was conducted by Jenkins et al. [7], involving 117 women with PG. The researchers found that the disease manifests primarily in the second and third trimesters of pregnancy (around 33% of cases in each trimester), with approximately 15% of cases occurring in the first trimester and postpartum period [7]. A similar analysis was performed around the same time by Vaughan Jones et al. [8], although the study sample was smaller, consisting of only 15 patients, more than half of whom developed the disease in the third trimester, while nearly 25% in the second trimester. Another analysis was conducted by Ambros-Rudolph et al. [9], where the study group was again small, consisting of 21 patients, one-third of whom developed symptoms in the second trimester, with the remainder in the third trimester. The most recent analysis was carried out by Al-Saif et al. [6], who studied 32 cases of women with PG, with 84% developing the disease in the second or third trimester, and the percentages for both trimesters being similar, with a slight predominance of the second trimester. Considering the above data, it is clear that PG occurs primarily in the second or third trimester of pregnancy, although there is no consensus on which trimester is most common. These discrepancies are primarily attributed to the small sample sizes in most studies, favoring the findings of Jenkins et al. [7].

PG has a relatively high recurrence rate in subsequent pregnancies, estimated at approximately 30-50%. All researchers agree that each recurrence tends to be more severe [2,4]. It is important to note that the reasons for recurrence or absence in subsequent pregnancies remain unclear. So far, various studies have ruled out the significance of maternal-fetal HLA-DR matching and changes in sexual partners in this process [4,6,7,8].

**Pathogenesis**

The pathogenesis of pemphigoid gestationis (PG) appears to be well-studied and closely resembles that of bullous pemphigoid (BP), which has led some researchers to consider PG as a variant of BP [1,3]. The development of PG is associated with the production of IgG1 antibodies targeting bullous pemphigoid antigen 180 (BP180) in the maternal body [10]. The immune response likely originates in the placenta due to abnormal expression of major histocompatibility complex (MHC) class II antigens, leading to the presentation of the BP180 antigen to the maternal immune system [11,12,13]. The antibodies produced in the maternal body bind to BP180 and BP230 in hemidesmosomes, resulting in complement activation, granulocyte chemotaxis, and subsequent degranulation, which ultimately causes damage to the basement membrane of the skin, leading to blister formation [14,15,16].

Previous studies have also shown that the presence of HLA-DR3 and HLA-DR4 haplotypes in women plays a key role in the development of the disease, with HLA-DR3 appearing to have a stronger association. However, the co-occurrence of both haplotypes is frequently observed [2].

**Clinical Presentation**

The primary symptom of pemphigoid gestationis (PG) is intense itching, which often precedes the onset of skin lesions [5,6]. The skin lesions are polymorphic, progressing through various stages, including papules, plaques, and vesicles, eventually developing into blisters on an erythematous base [2,5].

These lesions typically first appear around the umbilical area before spreading to the rest of the body [2,5]. The face and mucous membranes are usually spared, although there have been reported cases of their involvement [2,4,17]. In very rare cases, itching may be the sole symptom of PG, significantly complicating the diagnosis [4].

**Diagnosis**

The diagnosis of pemphigoid gestationis (PG) is based on clinical presentation, histopathological findings, and the results of direct immunofluorescence (DIF) testing [3]. The histopathological features depend on the stage of the disease. In the urticarial stage, edema of the superficial and deep dermis with a perivascular infiltrate consisting of lymphocytes, histiocytes, and eosinophils can be observed. In the bullous stage, subepidermal blisters filled with eosinophils and a mixed perivascular infiltrate are characteristic [1,3,5]. A biopsy result alone is not sufficient to confirm the diagnosis but is sometimes performed to exclude other dermatoses [5]. The gold standard for diagnosing PG is DIF of perilesional skin, which reveals linear deposition of C3 complement (in 100% of cases) and IgG autoantibodies (in 25-50% of cases) along the basement membrane zone [1,3,18]. In some cases, DIF results remain positive even years after the disease has resolved [1].

Other diagnostic methods include indirect immunofluorescence (IIF), which detects disease-specific antibodies in 30-100% of cases [19,20,21,22]. The ELISA test is also useful for identifying BP180 IgG antibodies, with sensitivity and specificity exceeding 90%, making it a valuable diagnostic tool for PG [2]. ELISA testing may eventually replace DIF, as it can also monitor disease activity, which correlates with antibody titers [1]. However, it should be noted that these newer diagnostic methods require specialized equipment and trained personnel, which may limit their availability in some parts of the world [23,24].

**Treatment**

The primary goals of PG treatment are to reduce itching and prevent the formation of new blisters [1]. A literature review conducted by Genovese et al. [25] indicated that the most commonly used treatments for PG include topical glucocorticosteroids (for mild disease) combined with systemic corticosteroids (in more severe cases) and antihistamines. This treatment approach was fully effective in over 80% of patients.

Non-fluorinated topical glucocorticosteroids are preferred because they cause fewer systemic side effects when absorbed through damaged skin [26]. In cases where glucocorticosteroids are contraindicated, calcineurin inhibitors may be used as an alternative therapy [27].

For systemic treatment, prednisone [25] or prednisolone [28] is most commonly used. The dosage is determined based on the severity and progression of the disease, with an effort to minimize the duration of systemic treatment during pregnancy to reduce its negative effects on the fetus [29]. When the standard treatment fails to achieve the desired outcomes, other options include intravenous immunoglobulins (IVIG) [29,30], azathioprine [5], and dapsone [28]. The first and third drugs are classified as category C by the FDA, while the second is category D. However, all have relatively safe profiles concerning fetal health [5]. It is important to conduct proper diagnostics (particularly for methemoglobinemia) and supplement vitamin E when using dapsone [28].

The treatment approach for postpartum patients differs slightly from that for pregnant women [5]. First-line treatment typically includes potent topical glucocorticosteroids and oral antihistamines. Second-line treatment consists of systemic glucocorticosteroids, followed by dapsone, and then azathioprine/rituximab/IVIG [25].

There have also been reports of rituximab as a preventive measure for PG, though it is recommended to allow at least one year between the last dose of the drug and the onset of a new pregnancy [31].

Recent studies [32,33] have demonstrated the high effectiveness of biologic agents in treating PG, including IVIG, omalizumab, dupilumab, and the previously mentioned rituximab. These analyses have also shown a low risk of fetal complications, though their limitation is the small number of cases reviewed [32].

**Prognosis and Complications**

The prognosis for both mother and child in most cases is favorable [1]. However, it is important to note the relatively high risk of disease recurrence, as previously mentioned. PG has been associated with Graves' disease, which occurs in nearly 10% of women who have had PG (compared to a prevalence of 0.4% in the general population) [2]. This correlation is at least partially explained by the HLA-DR3 and HLA-DR4 haplotypes typical of PG patients [2].

The prognosis for the fetus is generally good, but PG can influence the course of pregnancy [3]. Potential complications include preterm birth and intrauterine growth restriction (IUGR) [2]. These complications are most likely related to the course of the disease itself [3], although the impact of the treatment cannot be completely ruled out [2].

Approximately 10% of newborns from mothers with PG develop transient symptoms similar to the disease, resulting from the transfer of maternal autoantibodies to the child [34]. In most cases, these symptoms resolve spontaneously within a month as the antibodies disappear, and only symptomatic treatment is necessary [34]. In cases of severe skin lesions in the newborn, short-term topical steroid therapy may be considered [34].

**Summary**

Pemphigoid gestationis (PG) is an uncommon autoimmune disorder that is generally self-limiting and typically manifests during the second or third trimester of pregnancy. Research indicates that it is linked to an elevated risk of preterm delivery and the onset of Graves' disease in mothers. The condition arises from the production of IgG1 antibodies that target bullous pemphigoid antigen 180 (BP180).

The main symptom of PG is severe itching, which often precedes the appearance of skin lesions. These lesions evolve through various stages, ultimately resulting in blisters set against an erythematous background. Diagnosis relies on clinical evaluation, histopathological examination, and direct immunofluorescence (DIF) testing. The gold standard for diagnosis is DIF of perilesional skin. Additional diagnostic methods include indirect immunofluorescence (IIF) and the ELISA test.

The treatment of PG aims to alleviate itching and prevent the emergence of new blisters. For mild cases, topical corticosteroids are usually the first-line option, while systemic corticosteroids are reserved for more severe manifestations.

In the postpartum period, the approach to management may include the use of stronger topical corticosteroids and oral antihistamines.
Recent studies have suggested that biologic therapies could be effective in managing PG. While the prognosis for the fetus is largely positive, PG may complicate the course of pregnancy, with potential risks such as preterm birth and intrauterine growth restriction.
This article aims to summarize existing knowledge about pemphigoid gestationis (PG) and emphasizes the necessity for additional research, given the small sample sizes in previous studies. Despite its rarity, the occurrence of PG and its potential complications during pregnancy, along with long-term health effects, indicate a clear need for more extensive studies. Future research should focus on exploring preventive therapies and assessing the efficacy of emerging treatment options.

**Author's contribution:**
Conceptualization: K.W., A.M.
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formal analysis: K.W., A.M., J.W., A.W.
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data curation: K.W., A.M., J.W., A.W., N.S., E.Ga.
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writing - review and editing: K.W., A.M., J.W., A.W., J.D., W.C., P.S., E.Gw., N.S., E.Ga.
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