

Clinical Characterization and Outcomes of Dupilumab for Pemphigoid Gestationis: A Case Series of Five Patients and Literature Review

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Treatment of pemphigoid gestationis (PG) mainly consists of systemic and potent topical corticosteroids. This retrospective case series describes patients with severe PG treated with dupilumab, including serological follow-up, and reviews all published cases. PG was confirmed by direct immunofluorescence (linear C3 and/or IgG deposition along the basement membrane zone) and serology (IgG epidermal staining on salt-split skin or anti-BP180 NC16A IgG ELISA). Pruritus, quality of life and anti-BP180 NC16A IgG and anti-BP180 NC16A IgE were monitored longitudinally. In 5 pregnant women, dupilumab was initiated with a 600 mg loading dose followed by 300 mg biweekly. Baseline pruritus was severe (NRS 8–10), with high DLQI scores (18–30). Blistering ceased within 1 week in 2 patients and within 4 weeks in 1. Two patients required weekly dosing for disease control. Systemic corticosteroids were discontinued in 3 patients and tapered to ≤10 mg/day in 2 before delivery. In one case, anti-BP180 IgG paralleled clinical improvement. Anti-BP180 IgE remained negative. All patients delivered healthy infants. In 10 reported cases, pruritus improved rapidly and systemic corticosteroids were tapered. These findings suggest dupilumab may be a promising corticosteroid-sparing option for PG, with rapid clinical response and a favourable safety profile, despite off-label use in pregnancy.

Keywords Autoimmune disease; Biomarkers; Immunobullous disease; Patients.

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Pemphigoid gestationis (PG) is a rare autoimmune subepidermal blistering disease of pregnancy, characterized by intensely pruritic urticarial plaques that may evolve into vesicles or bullae. PG most commonly arises in the second or third trimester of

SIGNIFICANCE

Pemphigoid gestationis (PG) is a rare pregnancy-related skin disease that can cause intense itching and blistering. Treatment usually relies on corticosteroids, but these are not always effective and may have disadvantages for mother and child. We describe 5 pregnant women with severe PG who were treated with dupilumab, a targeted anti-inflammatory medicine. Symptoms improved rapidly, corticosteroids could be reduced or stopped, and all babies were born healthy. Together with 10 previously reported cases, our findings suggest that dupilumab may offer a promising additional treatment option for severe PG, although it is not yet officially approved for use in pregnancy.

pregnancy, although cases have been reported at any stage of gestation. Multigravid pregnant women are more susceptible to earlier onset and tend to experience a longer disease duration before remission. Although PG occasionally resolves spontaneously within 6 months postpartum, chronic disease or relapses may occur during subsequent pregnancies. Recent large-scale propensity-matched cohort data further demonstrated that PG is associated with an increased risk of adverse pregnancy outcomes, highlighting the importance of close obstetric follow-up (1, 2).

PG is hypothesized to involve an aberrant maternal immune response in early pregnancy. During the first trimester, the antigen of the BP180 protein is expressed by placental cells, including trophoblast and amniochorion stromal cells. An activation of the maternal immune system possibly leads to the generation of IgG autoantibodies targeting placental BP180, which subsequently cross-react with BP180 in maternal skin. This immune reaction within the skin initiates an inflammatory cascade, including eosinophil recruitment and degranulation, ultimately resulting in blister formation (3). Consequently, the immunological profile of PG is similar to that of bullous pemphigoid (BP), a more prevalent autoimmune blistering disease in the elderly (4).

Treatment options for PG are largely limited to systemic and potent topical corticosteroids. However, prolonged systemic corticosteroid use during pregnancy may be associated with maternal and fetal risks, including gestational diabetes, pregnancy induced hypertension and impaired fetal growth (5–7). Other treatments, including intravenous immunoglobulin, azathioprine, dapsone and cyclosporine, have also been reported, although supporting evidence is limited to case reports and small case series (5). Given the shared Th2-skewed immunopathogenesis of BP and PG, targeted type-2 immunomodulation has emerged as a potential therapeutic strategy. Dupilumab, a monoclonal antibody targeting the interleukin-4 receptor α subunit and thereby inhibiting IL-4 and IL-13 signalling, has shown clinical benefit in BP and has been reported in a limited number of cases with PG (8–16). Here, we describe 5 patients with severe, refractory PG treated with dupilumab, including longitudinal monitoring of circulating autoantibodies against BP180, and provide a review of all reported cases with PG treated with dupilumab to date.

MATERIALS AND METHODS

All consecutive patients with PG treated with dupilumab between September 2023 and January 2026 at the UMCG Center of Expertise for Blistering Diseases, University Medical Center Groningen, a national referral centre in the Netherlands, were included and retrospectively reviewed. The diagnosis of PG was based on compatible clinical features arising during pregnancy, together with (i) positive direct immunofluorescence (DIF) showing linear C3 and/or IgG deposition along the basement membrane zone of a skin biopsy specimen, and (ii) positive serologic test of IgG staining on the epidermal side of salt-split skin (SSS) by indirect immunofluorescence (IIF); or a positive enzyme-linked immunosorbent assay (ELISA) for BP180 NC16A IgG (17).

All patients received dupilumab according to the standard dosing regimen for atopic dermatitis: 600 mg subcutaneously on day 1, followed by 300 mg subcutaneously every 2 weeks. Dosing intervals were adjusted according to clinical response. Disease activity was evaluated at each visit. Pruritus was evaluated using the Numerical Rating Scale (NRS) (18), and quality of life was measured using the Dermatology Life Quality Index (DLQI) (19). Anti-BP180 NC16A IgG titres (>30 considered positive) and anti-BP180 IgE titres were monitored longitudinally (17, 20).

Patients were followed by a dermatologist and were also monitored by an obstetrician throughout pregnancy, including assessment of fetal growth. Placental histopathological examination was performed according to the Amsterdam criteria in 1 patient (21).

RESULTS

Patient characteristics and diagnostic findings

The clinical and diagnostic findings of our 5 patients with PG are summarized in **Table I**. All patients reported severe pruritus (NRS range: 7–10). DLQI was assessed in 4 patients (range: 18–30), indicating a very large to extremely large impact on quality of life. Three patients reported similar symptoms during previous pregnancies; however, a confirmed diagnosis of PG had been established in only one of them. DIF revealed linear n-serrated deposition of IgG along the basement membrane zone in all patients, with additional linear C3c in 3 cases, and IgM and IgA, each in 1 case. IIF on SSS showed IgG binding to the epidermal side in 4 patients, accompanied by IgA in 1 case, whereas 1 patient had negative findings. Circulating anti-BP180 NC16A IgG antibodies were positive in all cases (range: 54–115 U/mL). Anti-BP180 IgE titres were negative in all patients.

Treatment and neonatal outcomes

Treatment and neonatal outcomes of dupilumab treatment in the current case series are summarized in **Table II** (cases 1–5). Clinical photographs illustrating the marked improvement in skin lesions before and after dupilumab treatment are shown in **Fig. 1**. Longitudinal changes in NRS pruritus and DLQI are shown in **Fig. 2A** and **B**, respectively. Dupilumab was initiated between 17+5 and 31+6 weeks' gestation because of insufficient response to systemic corticosteroids in 4 patients (maximum doses 30–60 mg/day) and to potent topical corticosteroids in 1 patient. To exemplify the clinical response, in case 2, cessation of blistering occurred as early as day 2, with pruritus decreasing to NRS 4, further improving to NRS 0 after 5 weeks. At week 11, DLQI had improved from 26 to 10. In case 4, blistering ceased within 1 week, with pruritus decreasing to NRS 4 and resolving by delivery. In case 1, blistering ceased within 4 weeks, with pruritus improving to NRS 6 and further decreasing to NRS 4 after 11 weeks. DLQI improved from 30 to 16 at week 4, and to 4 at week 11. In 2 cases (3 and 5), the dupilumab dosing interval was shortened to weekly administration, resulting in cessation of blistering at week 12 in case 3 and week 5 in case 5. Pruritus improved to NRS 4 in case 3 and NRS 2 in case 5. All patients delivered between 36+0 and 39+6 weeks' gestation; 2 deliveries were by caesarean section, and all infants were healthy at birth without skin blistering.

Fig. 2C illustrates longitudinal changes in anti-BP180 NC16A IgG titres by ELISA during dupilumab treatment. In case 1, titres declined from 74 U/mL at initiation (30 weeks' gestation) to 68 U/mL after 4 weeks. In case 2, titres increased from 73 U/mL at 18 weeks' gestation to 132 U/mL at delivery

Table I. Clinical characteristics and diagnostic findings of 5 patients with pemphigoid gestationis

Patient no./ Age (years)	Gravida para	Prior PG	Clinical status before dupilumab initiation			Diagnostic findings		
			Clinical features	NRS pruritus	DLQI	DIF	IIF	ELISA BP180 NC16A IgG (U/mL)
1/37	G5P3	Yes	Multiple annular, erythematous urticarial plaques with bullae on the trunk, legs and arms	10	30	Linear n-serrated IgG 2+, IgM +	IgG 2+roof	54
2/37	G2P1	Similar symptoms, no diagnosis	Widespread annular, urticarial, erythematous plaques and bullae	10	26	Linear n-serrated IgG +/2+, C3c 3+	Neg.	77
3/29	G3P1	Similar symptoms diagnosis of PUPPP	Widespread pink-violaceous and erythematous maculae and plaques with erosions and excoriations, most pronounced on the trunk and legs, and excoriated papules on the cheeks	7	NR	Linear n-serrated IgG +, C3c 2+	IgG+roof	73
4/30	G1P0	No	Urticarial papules and plaques on the abdomen and legs, including bullae on the hands and feet	8	26	Linear n-serrated IgG +/-, IgA +/-	IgG 2+, IgA 2+roof	115
5/31	G1P0	No	Disseminated erythematous papules and plaques, with bullae on the soles	8	18	Linear n-serrated IgG 2+, C3c 3+	IgG 2+roof	105

BP: bullous pemphigoid; C: complement; DIF: direct immunofluorescence microscopy; DLQI: Dermatology Life Quality Index; ELISA: enzyme-linked immunosorbent assay; Ig: Immunoglobulin; IIF: indirect immunofluorescence; NR: not reported; NRS: numeric rating scale; PUPPP: pruritic urticarial papules and plaques of pregnancy.

despite clinical improvement, and subsequently declined postpartum. In case 3, titres rose from 73 U/mL 2 weeks before initiation to 104 U/mL at 30 weeks' gestation. In cases 4 and 5, titres declined from 115 to 68 U/mL postpartum and from 105 to 94 U/mL after 4 weeks of treatment, respectively. Anti-BP180 IgE by ELISA was negative at baseline and during follow-up in all samples.

In case 2, placental weight was 545 g (normal for gestational age) with <5% abnormal parenchyma. Microscopic placental examination showed lymphohistiocytic infiltrates within the villi, compatible with high-grade chronic villitis, of unknown aetiology. Multifocal chronic histiocytic intervillitis, low-grade fetal vascular malperfusion, and areas of chorangiosis were also observed. No maternal vascular malperfusion or chorioamnionitis were found. Parenchymal maturation was appropriate for gestational age.

Table II also summarizes the treatment outcomes of ten previously published dupilumab-treated cases of PG (cases 6–15) (8–16). Dupilumab was initiated during pregnancy in 9 out of 10 cases, most often in the second or third trimester; in one case, treatment was started postpartum (15). Clinical response was generally rapid, with marked improvement of complete cessation of blistering within 6 weeks. Pruritus improved rapidly in all reported cases, often within days to weeks after initiation of dupilumab. Follow-up of BP180 antibody titres was reported in 4 cases and showed variable patterns, with decreasing titres in 2 cases (8, 11) and increasing or persistently elevated titres in 2 others (13,

14) despite clinical improvement. In all 9 cases in which dupilumab was initiated during pregnancy, systemic corticosteroids could be tapered (8–14, 16).

Mild postpartum flares were reported in 2 cases (12, 14), and dupilumab was discontinued postpartum in most patients. Delivery occurred between 34 and 39+6 weeks' gestation. One preterm delivery followed preterm premature rupture of membranes (8), and one pregnancy was complicated by pre-eclampsia requiring caesarean section (16). Overall, caesarean section was performed in 5 cases (8–10, 14, 16). All reported neonates were healthy at birth.

DISCUSSION

We report a series of 5 pregnant women with severe, refractory PG with a high impact of pruritus treated with dupilumab. A rapid clinical improvement was seen with cessation of new blister formation within 1 week in 2 patients and within 4 weeks in a third, with marked reductions in pruritus from NRS 8–10 to NRS 4. In 2 patients, escalation to weekly administration of dupilumab was required to achieve full disease control, resulting in subsequent cessation of blistering and improvement in pruritus to NRS 4 and NRS 2, respectively. Dupilumab enabled discontinuation of systemic corticosteroids in 3 patients and tapering to 10 mg daily or less before delivery in the remaining 2. Dupilumab was well tolerated, and all infants were born healthy.

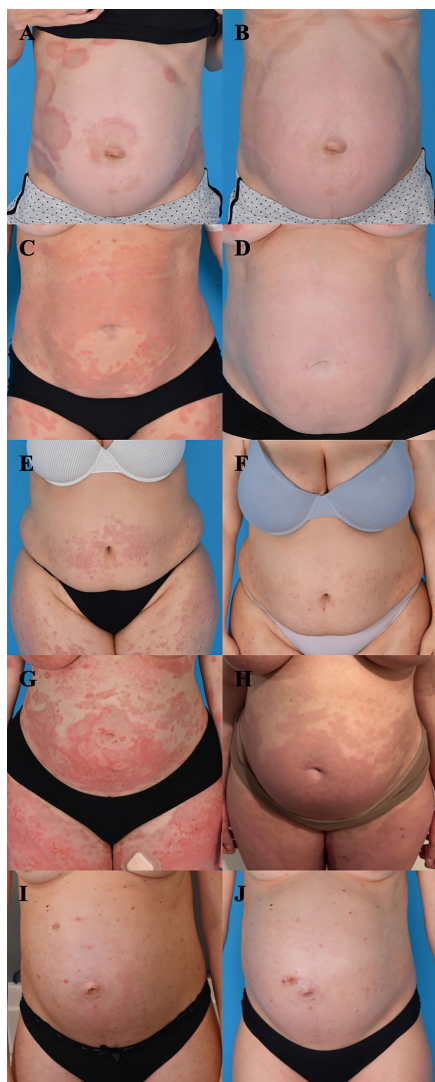


Fig. 1. Patients with pemphigoid gestationis before and after treatment with dupilumab.

A. Case 1 (baseline). Multiple annular erythematous plaques on the trunk, arms and legs. B. Case 1 (week 4). Complete resolution of inflammatory skin lesions. C. Case 2 (baseline). Widespread annular urticarial erythematous plaques on the trunk. D. Case 1 (week 4). Complete resolution of inflammatory skin lesions. E. Case 3 (baseline). Pink-violaceous and erythematous macules and plaques with erosions and excoriations on the trunk and legs. F. Case 3 (week 8). Excoriated erythematous papules on the trunk and arms. Widespread annular plaques with peripheral activity persisted predominantly on the lower legs, with an intact vesicle on the right pretibial area. G. Case 4 (baseline). Urticarial papules and plaques on the abdomen and legs, with bullae on the hands and feet. H. Case 4 (week 2). Marked improvement with fading erythema and no new bullae formation. I. Case 5 (baseline). Disseminated erythematous papules and plaques on the trunk, with bullae on the soles. J. Case 5 (week 4). Several lenticular, erythematous macules on the trunk without vesicles or bullae.

When considered together with the 10 previously published cases, a total of 15 dupilumab-treated PG patients have now been reported. Across all these cases, dupilumab was generally associated with rapid clinical improvement, marked reduction in pruritus, and a corticosteroid-sparing effect during pregnancy. Clinical improvement was typically observed within the

first weeks after initiation, and the postpartum disease course was mostly stable, with only mild flares reported in a minority of cases. Only 3 pregnancies were preterm and 1 pregnancy was complicated by pre-eclampsia.

Given that PG has been associated with an increased risk of adverse pregnancy outcomes, including fetal growth restriction, preterm delivery, and hypertensive pregnancy disorders (7, 22, 23), the pregnancy outcomes observed in this dupilumab-treated PG cohort appear comparable to those previously reported for PG. However, the limited number of cases limits firm conclusions regarding the effect of dupilumab on pregnancy outcomes in PG.

Limited data are available on pregnancy in patients treated with dupilumab for other indications. A recent systematic review and meta-analysis evaluating 115 pregnancies exposed to dupilumab in women with AD found a weighted prevalence of spontaneous pregnancy loss of 18.9%, consistent with background population rates (24).

In the only placenta available for histopathological examination, placental weight and parenchymal maturation were appropriate for gestational age, and no maternal vascular malperfusion or chorioamnionitis was observed. These findings do not support clear placental insufficiency in this case. Chronic villitis was observed, which has been associated with fetal growth restriction but may also occur in uncomplicated pregnancies. Focal fetal vascular malperfusion was also present, likely secondary to inflammation (21). Chronic intervillitis, which may reflect a maternal immune response, can occur together with villitis, while chorangiosis, a nonspecific marker of chronic fetal hypoxia, may represent a reactive change to these inflammatory processes. Villitis has previously been described in PG (25), and earlier reports suggested maternal immune activation at the villous stroma based on HLA-DR upregulation (26). These findings may reflect maternal immune activation but should be interpreted with caution because placental histopathology was available for only one patient.

Beyond clinical observations, mechanistic and preclinical data provide additional context regarding potential safety during pregnancy. In general, maternal immune adaptation is characterized by a shift from a Th1-dominant to a Th2-skewed cytokine profile, particularly at the maternal-fetal interface, with increased production of IL-4 and IL-10 to facilitate fetal tolerance (27). Although dupilumab inhibits IL-4 and IL-13 signalling, current data on placental transfer and safety are reassuring. Dupilumab, an IgG4 monoclonal antibody, crosses the placenta via the neonatal Fc receptor (FcRn), which is expressed on placental syncytiotrophoblasts. Given that FcRn expression is minimal or absent during the first trimester, significant transplacental transfer early in pregnancy is unlikely,

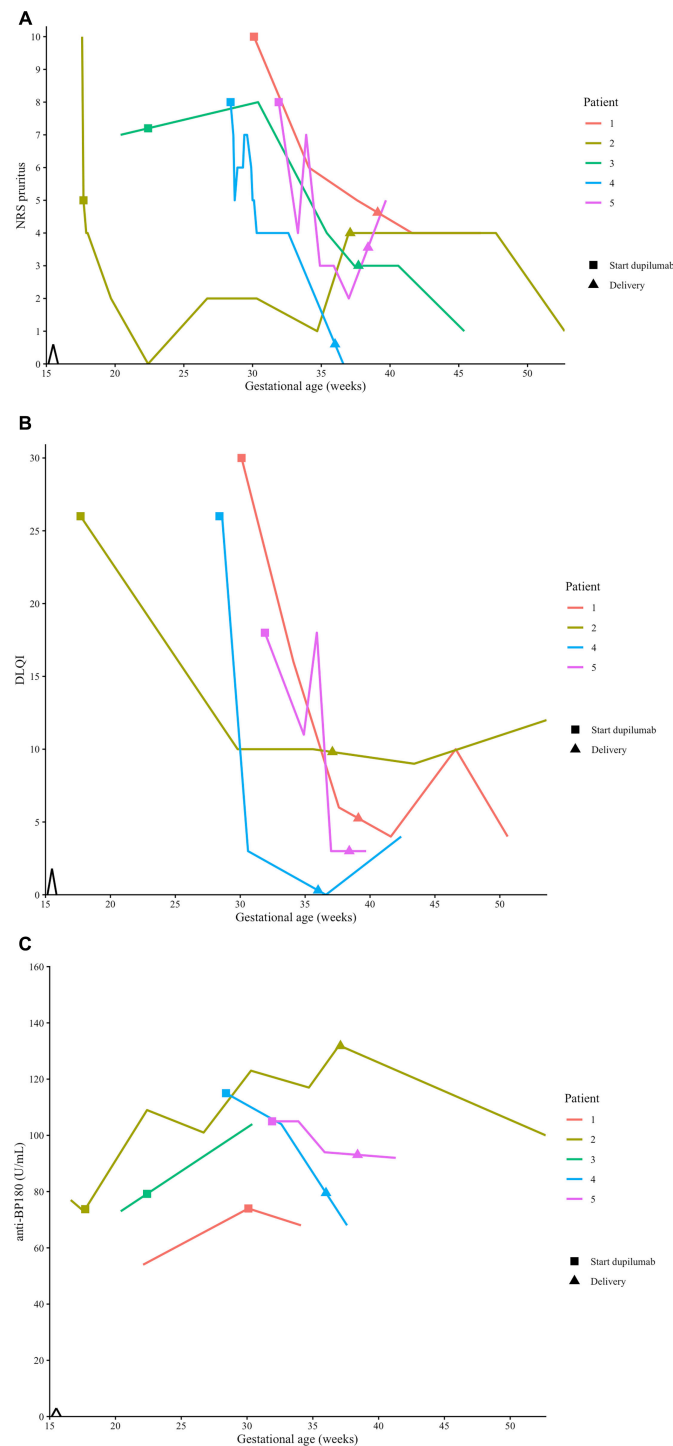


Fig. 2. A. Longitudinal changes in NRS pruritus scores. B. Longitudinal changes in DLQI scores. C. BP180-titres monitoring during treatment with dupilumab.

and teratogenic risk may be considered low. Since PG typically manifests in the second or third trimester, treatment generally begins after the critical period of organogenesis. Furthermore, pre- and postnatal toxicity studies in monkeys have shown no adverse effects on offspring development with anti-IL-4R α inhibition (28). Taken together, clinical and preclinical data suggest that dupilumab may be a safe treatment for PG.

Anti-BP180 IgG antibody monitoring during PG disease course and dupilumab treatment showed variable responses (Fig. 2C), with in one case a decline of antibody titres following clinical improvement. This variable pattern is consistent with previous dupilumab-treated PG reports, where clinical improvement has been observed both with decreasing and with persistently elevated or increasing BP180 titres (8, 11, 13, 14).

Table II. Treatment outcomes of dupilumab in pemphigoid gestationis: current case series (1–5) and previously reported cases (6–15)

Patient no./ Age (years)	Author	GA at PG onset (wk)	Oral CS max dose	GA at dupilumab initiation (wk+d)	Treatment response after start dupilumab	Oral CS tapered after start dupilumab	Oral CS at delivery (mg/day)	Treatment response postpartum	Treatment after delivery	GA at delivery, neonatal outcome (wk+d)
1/37	Meijer et al.	14	30 mg/day	30+1	Wk 4: no new blisters; NRS 10→6; DLQI 30→16. Wk 11: NRS 4; DLQI 4	Yes	-	No new blisters Day 12 pp: NRS pruritus 4	Dupilumab dose interval extended from 7 wk pp and stop at 28 wk pp. Persistent pruritus requiring restart dupilumab 10 months later	39+6, healthy infant
2/37	Meijer et al.	13	60 mg/day	17+5	Day 2: no new blisters; NRS 10→4; wk 5: NRS 0. Wk 11: DLQI 26→10.	Yes	-	Day 2 pp: new blisters Wk 1 pp: no new blisters, NRS pruritus 4	PP topical corticosteroids for mild flare; dupilumab dose interval extended from 16 wk pp. Interval every 5 weeks last follow-up 47 wk pp	37+1, C-section, healthy infant
3/29	Meijer et al.	8	-	22+3	Wk 8: new blisters; NRS 8. After dupilumab q1w, at wk 12: no new blisters; NRS 8→4. At delivery: NRS 3	-	-	No new blisters, NRS pruritus 1	Dupilumab stop at 4 wk pp	37+5, healthy infant
4/30	Meijer et al.	26	45 mg/day	28+3	Week 1: no new blisters; NRS 8→4. At delivery: NRS 0	Yes	7.5 mg	Day 4 pp: NRS 0 Day 6 pp: new blisters Wk 6 pp: no new blisters, NRS pruritus 0	PP topical corticosteroids for mild flare. Oral CS stop at wk 7 pp; dupilumab dose interval extended from 13 wk pp to q3w	36+0, C-section, healthy infant
5/31	Meijer et al.	26	40 mg/day	31+6	Wk 3: new lesions, no new blisters; NRS 8→3. After dupilumab q1w, at wk 5: no new blisters; NRS 2	Yes	10 mg	Day 1 pp: new blisters	Day 1 pp: oral CS 20 mg tapered to 10 mg until 4 wk; Dupilumab 1qw until last follow-up	38+3, healthy infant
6/37	Riquelme-Mc Loughlin et al. (6)	20	0.5 mg/kg/day	30	Wk 3: No blisters and stop dupilumab	Yes	NR	No new blisters	No dupilumab Oral CS NR	34.4, PPROM, C-section, healthy infant
7/36	Chen et al. (7)	18	40 mg/day	22	Wk 4: considerable improvement of rash, blisters, pruritus	Yes	-	Wk4 pp: urticarial plaques legs resolved	Dupilumab until wk 4 pp; discontinuation not reported	39, C-section, healthy infant
8/28	Liu et al. (8)	22	1 mg/kg/day	25	Day 5: no lesions Wk 4 itch relieved	Yes	10 mg	NR	NR	36.2, C-section, healthy infant
9/28	Alvarez Martinez et al. (9)	27	0.5 mg/kg	6 wk pre delivery	Itch rapidly controlled Wk 6: no lesions	Yes	NR	NR	Wk 6 pp: dupilumab stop	NR, healthy infant
10/39	Thomas. et al. (10)	23.5	60 mg/day	30.5	Wk 2: no lesions and no itch	Yes	100 mg stress-dose	Wk 2 pp: new lesions and itch Wk 12 pp: no lesions	Oral CS 2.5 mg Q2D. Wk 2 pp: oral CS 10 mg tapering in 2 wk. Wk 12 pp: dupilumab stop	37+3, healthy infant
11/39	Thomas et al. (11)	24	40 mg/day	25+5	Wk 4: no lesions	Yes	No	Wk 5: no recurrence	Wk 5 pp: dupilumab stop	38, healthy infant

(Continued)

Table II. Treatment outcomes of dupilumab in pemphigoid gestationis: current case series (1–5) and previously reported cases (6–15) (Continued)

Patient no./ Age (years)	Author	GA at PG onset (wk)	Oral CS max dose	GA at dupilumab initiation (wk+d)	Treatment response after start dupilumab	Oral CS tapered after start dupilumab	Oral CS at delivery (mg/day)	Treatment response postpartum	Treatment after delivery	GA at delivery, neonatal outcome (wk+d)
12/38	Phong et al. (12)	19	40 mg/day	29	Wk 3: no blisters. Wk 6: lesions resolved	Yes	No	No new lesions, moderate itch.	Wk 6 pp: dupilumab stop	39, healthy infant
13/38	Calvo Arbeloa et al. (13)	6	1.5 mg/kg/day	14	Wk 1: less lesions, less itch. Month 1: few lesions, no itch	Yes	NR	Day 2 pp: mild flare Month 2: stable	Day 2 pp: oral CS Month 2 pp: dupilumab stop	37, scheduled C-section
14/32	Sun et al. (14)	26	60 mg/day	After delivery	NR	NR	NR	Stable lesions	Month 9: dupilumab stop Month 19: no relapse	NR
15/31	Ryan et al. (15)	25	40 mg/day	34	Rapid resolution of blisters and itch	Yes	NR	Wk 4: no relapse	Wk 4 pp: dupilumab stop	37+6, pre-eclampsia, C-section, healthy infant

CS: corticosteroids; C-section: caesarean section; d: day(s); GA: gestational age; NR: not reported; NRS: numeric rating scale; pp: postpartum; PPRM: preterm prematurely rupture of membranes; PUPPP: Pruritic Urticarial Papules and Plaques of Pregnancy; Q2D: every other day; wk: week(s); y: years.

Dupilumab appears to be a well-tolerated, potentially safe and effective therapeutic option for severe PG, demonstrating rapid clinical improvement and corticosteroid-sparing potential during pregnancy. With the addition of the present series, 15 cases of dupilumab-treated PG have now been documented, substantially expanding the currently available evidence on dupilumab treatment in PG. Placental examination revealed inflammatory and vascular abnormalities, suggesting that placental involvement may represent an underexplored aspect of PG pathophysiology and warranting systematic investigation in future studies.

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IRB approval status: The Central ethics Review Board non-WMO studies (CTC UMCG) has approved.

Conflict of interest: J.M. Meijer reports Argenx (Advisory Boards, Consultations), Sanofi (Consultations, Symposium presentations), all contracts are reviewed and signed by the Board of Directors and all fees were paid to the institution; E.J. Leeman reports no conflict of interest; L.M. Prens reports no conflicts of interest; J.R. Prins reports no conflicts of interest; S.J. Gordijn reports no conflicts of interest; Mirthe H. Schoots reports no conflicts of interest; Maria C. Bolling reports no conflicts of interest; Gilles F.H. Diercks reports no conflicts of interest; J. Bremer reports no conflicts of interest; B. Horváth reports fees from Janssen-Cilag (Advisory Boards, Educational grants, Consultations, Investigator Initiative Studies), AbbVie (Advisory Boards, Educational grants, Consultations, Investigator Initiative Studies), Novartis

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