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Contents

Thrice Monthly Volume 9 Number 34 December 6, 2021

OPINION REVIEW

- 10392** Regulating monocyte infiltration and differentiation: Providing new therapies for colorectal cancer patients with COVID-19

Bai L, Yang W, Qian L, Cui JW

REVIEW

- 10400** Role of circular RNAs in gastrointestinal tumors and drug resistance

Xi SJ, Cai WQ, Wang QQ, Peng XC

MINIREVIEWS

- 10418** Liver injury associated with acute pancreatitis: The current status of clinical evaluation and involved mechanisms

Liu W, Du JJ, Li ZH, Zhang XY, Zuo HD

- 10430** Association between celiac disease and vitiligo: A review of the literature

Zhang JZ, Abudoureyimu D, Wang M, Yu SR, Kang XJ

- 10438** Role of immune escape in different digestive tumours

Du XZ, Wen B, Liu L, Wei YT, Zhao K

ORIGINAL ARTICLE

Basic Study

- 10451** Magnolol protects against acute gastrointestinal injury in sepsis by down-regulating regulated on activation, normal T-cell expressed and secreted

Mao SH, Feng DD, Wang X, Zhi YH, Lei S, Xing X, Jiang RL, Wu JN

Case Control Study

- 10464** Effect of Nephritis Rehabilitation Tablets combined with tacrolimus in treatment of idiopathic membranous nephropathy

Ly W, Wang MR, Zhang CZ, Sun XX, Yan ZZ, Hu XM, Wang TT

Retrospective Cohort Study

- 10472** Lamb's tripe extract and vitamin B₁₂ capsule plus celecoxib reverses intestinal metaplasia and atrophy: A retrospective cohort study

Wu SR, Liu J, Zhang LF, Wang N, Zhang LY, Wu Q, Liu JY, Shi YQ

- 10484** Clinical features and survival of patients with multiple primary malignancies

Wang XK, Zhou MH

Retrospective Study

- 10494** Thoracoscopic segmentectomy and lobectomy assisted by three-dimensional computed-tomography bronchography and angiography for the treatment of primary lung cancer
Wu YJ, Shi QT, Zhang Y, Wang YL
- 10507** Endoscopic ultrasound fine needle aspiration *vs* fine needle biopsy in solid lesions: A multi-center analysis
Moura DTH, McCarty TR, Jirapinyo P, Ribeiro IB, Farias GFA, Madruga-Neto AC, Ryou M, Thompson CC
- 10518** Resection of bilateral occipital lobe lesions during a single operation as a treatment for bilateral occipital lobe epilepsy
Lyu YE, Xu XF, Dai S, Feng M, Shen SP, Zhang GZ, Ju HY, Wang Y, Dong XB, Xu B
- 10530** Improving rehabilitation and quality of life after percutaneous transhepatic cholangiography drainage with a rapid rehabilitation model
Xia LL, Su T, Li Y, Mao JF, Zhang QH, Liu YY
- 10540** Combined lumbar muscle block and perioperative comprehensive patient-controlled intravenous analgesia with butorphanol in gynecological endoscopic surgery
Zhu RY, Xiang SQ, Chen DR
- 10549** Teicoplanin combined with conventional vancomycin therapy for the treatment of pulmonary methicillin-resistant *Staphylococcus aureus* and *Staphylococcus epidermidis* infections
Wu W, Liu M, Geng JJ, Wang M
- 10557** Application of narrative nursing in the families of children with biliary atresia: A retrospective study
Zhang LH, Meng HY, Wang R, Zhang YC, Sun J

Observational Study

- 10566** Comparative study for predictability of type 1 gastric variceal rebleeding after endoscopic variceal ligation: High-frequency intraluminal ultrasound study
Kim JH, Choe WH, Lee SY, Kwon SY, Sung IK, Park HS
- 10576** Effects of WeChat platform-based health management on health and self-management effectiveness of patients with severe chronic heart failure
Wang ZR, Zhou JW, Liu XP, Cai GJ, Zhang QH, Mao JF
- 10585** Early cardiopulmonary resuscitation on serum levels of myeloperoxidase, soluble ST2, and hypersensitive C-reactive protein in acute myocardial infarction patients
Hou M, Ren YP, Wang R, Lu LX

Prospective Study

- 10595** Remimazolam benzenesulfonate anesthesia effectiveness in cardiac surgery patients under general anesthesia
Tang F, Yi JM, Gong HY, Lu ZY, Chen J, Fang B, Chen C, Liu ZY

Randomized Clinical Trial

- 10604** Effects of lower body positive pressure treadmill on functional improvement in knee osteoarthritis: A randomized clinical trial study
Chen HX, Zhan YX, Ou HN, You YY, Li WY, Jiang SS, Zheng MF, Zhang LZ, Chen K, Chen QX

SYSTEMATIC REVIEWS

- 10616** Effects of hypoxia on bone metabolism and anemia in patients with chronic kidney disease
Kan C, Lu X, Zhang R

META-ANALYSIS

- 10626** Intracuff alkalinized lidocaine to prevent postoperative airway complications: A meta-analysis
Chen ZX, Shi Z, Wang B, Zhang Y

CASE REPORT

- 10638** Rarely fast progressive memory loss diagnosed as Creutzfeldt-Jakob disease: A case report
Xu YW, Wang JQ, Zhang W, Xu SC, Li YX
- 10645** Diagnosis, fetal risk and treatment of pemphigoid gestationis in pregnancy: A case report
Jiao HN, Ruan YP, Liu Y, Pan M, Zhong HP
- 10652** Histology transformation-mediated pathological atypism in small-cell lung cancer within the presence of chemotherapy: A case report
Ju Q, Wu YT, Zhang Y, Yang WH, Zhao CL, Zhang J
- 10659** Reversible congestive heart failure associated with hypocalcemia: A case report
Wang C, Dou LW, Wang TB, Guo Y
- 10666** Excimer laser coronary atherectomy for a severe calcified coronary ostium lesion: A case report
Hou FJ, Ma XT, Zhou YJ, Guan J
- 10671** Comprehensive management of malocclusion in maxillary fibrous dysplasia: A case report
Kaur H, Mohanty S, Kochhar GK, Iqbal S, Verma A, Bhasin R, Kochhar AS
- 10681** Intravascular papillary endothelial hyperplasia as a rare cause of cervicothoracic spinal cord compression: A case report
Gu HL, Zheng XQ, Zhan SQ, Chang YB
- 10689** Proximal true lumen collapse in a chronic type B aortic dissection patient: A case report
Zhang L, Guan WK, Wu HP, Li X, Lv KP, Zeng CL, Song HH, Ye QL
- 10696** Tigecycline sclerotherapy for recurrent pseudotumor in aseptic lymphocyte-dominant vasculitis-associated lesion after metal-on-metal total hip arthroplasty: A case report
Lin IH, Tsai CH

- 10702** Acute myocardial infarction induced by eosinophilic granulomatosis with polyangiitis: A case report
Jiang XD, Guo S, Zhang WM
- 10708** Aggressive natural killer cell leukemia with skin manifestation associated with hemophagocytic lymphohistiocytosis: A case report
Peng XH, Zhang LS, Li LJ, Guo XJ, Liu Y
- 10715** Chronic lymphocytic leukemia/small lymphocytic lymphoma complicated with skin Langerhans cell sarcoma: A case report
Li SY, Wang Y, Wang LH
- 10723** Severe mediastinitis and pericarditis after endobronchial ultrasound-guided transbronchial needle aspiration: A case report
Koh JS, Kim YJ, Kang DH, Lee JE, Lee SI
- 10728** Obturator hernia - a rare etiology of lateral thigh pain: A case report
Kim JY, Chang MC
- 10733** Tracheal tube misplacement in the thoracic cavity: A case report
Li KX, Luo YT, Zhou L, Huang JP, Liang P
- 10738** Peri-implant keratinized gingiva augmentation using xenogeneic collagen matrix and platelet-rich fibrin: A case report
Han CY, Wang DZ, Bai JF, Zhao LL, Song WZ

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Diagnosis, fetal risk and treatment of pemphigoid gestationis in pregnancy: A case report

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Abstract

BACKGROUND

Pemphigoid gestationis (PG) is a rare autoimmune blistering disease that usually presents in the second or third trimester, with an incidence of 1 per 50000 pregnancies. PG tends to recur with an earlier onset and a more severe course in subsequent pregnancies. Skin biopsy markers can be confirmed by direct immunofluorescence staining.

CASE SUMMARY

Our patient was diagnosed with PG at 8 mo of gestation with fresh bullous lesion marks on the abdomen and limbs. Termination of the pregnancy was performed by cesarean section at 37 + 4 wk of gestation. The patient delivered an infant weighing 3620 gm. The infant had urticaria-like and vesicular skin lesions and was diagnosed with PG. The patient was discharged on prednisolone and in a satisfactory condition. The infant was discharged after anti-inflammatory therapy for one week.

CONCLUSION

PG is a rarely reported disease, and 10% of newborns develop mild clinical symptoms consisting of urticaria-like or vesicular skin lesions. We intend to remind clinicians to consider this condition when a patient presents with such lesions so that treatment can be started early and neonatal morbidity can be taken into account.

Key Words: Pemphigoid gestationis; Pregnancy; Newborn; Pemphigus; Fluorescent antibody technique; Case report

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Core Tip: Pemphigoid gestationis (PG) is a rarely reported disease, and 10% of newborns develop mild clinical symptoms consisting of urticaria-like or vesicular skin lesions. Our patient was diagnosed with PG at 8 mo of gestation and was performed by cesarean section at 37 + 4 wk of gestation. The infant had urticaria-like and vesicular skin lesions and was diagnosed with PG. We intend to remind clinicians to consider this condition when a patient presents with such lesions so that treatment can be started early and neonatal morbidity can be taken into account.

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INTRODUCTION

Pemphigoid gestation (PG) is an autoimmune bullous disease that occurs during pregnancy or the puerperium[1]. The main clinical manifestations are tension blisters and bullae with urticaria-like plaques, accompanied by pruritus[2]. For approximately 14%-25% of patients in the postpartum period, menstrual periods and oral contraceptive drugs may induce or aggravate the disease[3]. PG can induce fetal growth restriction, premature delivery, and neonatal skin involvement. It has also been reported to cause low birth weight[4]. This disease is very rare, the clinical manifestation and laboratory examination results are similar to bullous pemphigoid, and the exact pathogenesis of PG has not been clarified.

CASE PRESENTATION

Chief complaints

A 30-year-old female presented with pruritic edematous erythema for 2 wk.

History of present illness

A 30-year-old female presented with a 2-wk history of pruritic edematous erythema on the trunk and extremities during her 32nd week of pregnancy with no inducing factors. These symptoms have never occurred before. She also had no history of drug or food allergies. Soon after, small bullae and vesicles developed at the periphery of the erythema; these healed readily with residual pigmentation (Figure 1).

History of past illness

The patient had no history of any previous disease.

Personal and family history

There was no family history of PG.

Physical examination

No mucosal lesions were observed. The Nikolsky sign was negative.

Laboratory examinations

Laboratory findings were normal, including hematocrit, urine analysis, hepatic and renal function, electrolytes, glycemia, anti-streptolysin and C-reactive protein.

Imaging examinations

Obstetric ultrasonography indicated no abnormality in the fetus.



Figure 1 Pemphigoid gestationis on the abdomen and limbs.

FINAL DIAGNOSIS

A clinical diagnosis of PG was made. This was supported by histology that showed a dermoepidermal blister with conspicuous eosinophils within the blister and in the papillary dermis. Further investigation identified that the skin biopsy showed subepidermal blister with eosinophils and lymphocytes within the cavity and in the superficial dermis (Figure 2). Direct immunofluorescence (DIF) showed linear deposits of C3 along the basement membrane zone in the absence of IgG (Figure 3). Indirect immunofluorescence (IIF) was negative. Anti-BP180 was positive (90 normal range < 20 IU/mL), and anti-BP230 was negative. Thus, a diagnosis of PG was made.

TREATMENT

Prednisone was administered orally at a dose of 25 mg. Four weeks later, the prednisone was tapered to 20 mg gradually with no new lesions appearing. Many red spots and pigmentation were left on the body and limbs. During the pregnancy, regular prenatal visits were conducted to help to monitor the health of the patient and her baby.

OUTCOME AND FOLLOW-UP

A multidisciplinary (obstetrics, dermatology, pathology, and neonatology) discussion was initiated during the patient's late pregnancy (37 wk of pregnancy) to determine the timing and method of delivery. For this patient with obstetrical factors (scarred uterus), a cesarean section at 37 + 4 wk of gestation was recommended. All the decisions were fully communicated with the patient and her family members. The Apgar score of the infant was 10 points, and the weight of the infant was 3620 g. After the placenta was delivered, a pathology specimen and umbilical cord blood was collected for examination. Prednisone was administered orally at a dose of 20 mg with no postoperative symptoms.

After examining the newborn, we found that the infant had vesicles and pustules on the palms and soles and erosions on the face and trunk (Figure 4). The skin biopsy showed another layer of epithelium over the normal epithelium with eosinophil and lymphocyte infiltration (Figure 5). DIF showed linear deposits of C3 along the basement membrane zone, similar to that observed in the mother (Figure 6). Anti-BP180 was positive (100 normal range < 20 IU/mL). Anti-BP230 was negative.

Intensive monitoring and anti-infection treatment with penicillium legume intravenous injection for the infant were given. With local use of astringent and bactericidal drugs, the infant's rash gradually improved after 1 wk (Figure 7).

DISCUSSION

PG can be diagnosed in several ways: clinical symptoms, histological findings, DIF or IIF, and enzyme-linked immunosorbent assay. In our patient, DIF showed a linear

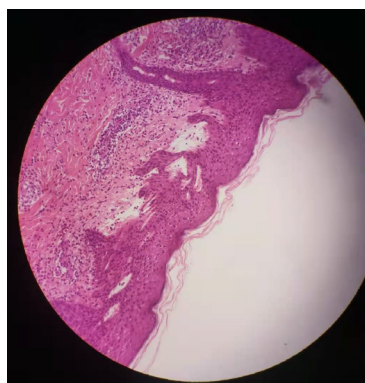


Figure 2 Skin with a sub-epidermal blister with eosinophils and lymphocytes within the cavity and in superficial dermis of pemphigoid gestationis in pregnancy (hematoxylin and eosin 100×).

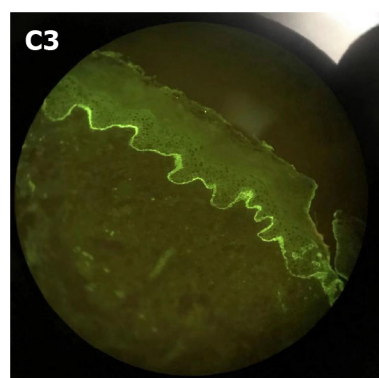


Figure 3 Linear deposition of C3 along the basement membrane zone with the absence of IgG (direct immunofluorescence) of pemphigoid gestationis in pregnancy.

deposit of C3 along the basement membrane zone in the absence of IgG. In a retrospective study of 25 cases of PG, fourteen patients presented the same DIF pattern as our patient[5]. Linear C3 deposits were observed in 100% of patients, and linear IgG deposits were observed in 25%-50% of patients, which indicated that C3 was pathognomonic to the disease[3]. This is the most important and commonly used PG detection method[6].

Treatment, which prevents the formation of herpes and controls itching symptoms depends on the stage and severity of the disease[7]. Topical glucocorticoids and orally administered antihistamine drugs can be used in mild cases. Oral corticosteroids (prednisone 0.5-1 mg/kg/d) or even intravenous gamma globulin may be used in severe cases[8]. PG is usually in remission for weeks to months after delivery, with a few cases extending to 2 years after delivery.

Most pregnant women with PG can deliver a healthy newborn after full-term pregnancy, *via* natural delivery or cesarean section for obstetric indications[9]. Due to the passive transfer of IgG1 antibody from mother to fetus, 10% of newborns with PG develop clinical symptoms, including urticaria or vesicular skin lesions, and have an increased risk of premature delivery and low birth weight[10]. There is no evidence of an association between medication regimens and fetal outcomes, and adverse pregnancy outcomes appear to be more closely associated with higher antibody titers in maternal serum and neonatal cord blood[11].

In this patient, multidisciplinary (obstetrics, dermatology, pathology, and neonatology) discussion was initiated during the patient's late pregnancy (37 wk of pregnancy) to determine the timing and method of delivery. For this patient, who had obstetrical factors, a cesarean section at 37 + 4 wk of gestation pregnancy was recommended.

The newborn had vesicles and pustules on the palms and soles and erosions on the face and trunk. Her oral mucosa was not involved, and the skin lesions subsided rapidly without any proliferative changes. Therefore, the clinical manifestations of skin lesions in the infant were significantly different from those in the mothers.



Figure 4 The first day after birth of the infant: Urticaria-like and vesicular skin lesions in neonatal pemphigoid gestationis.

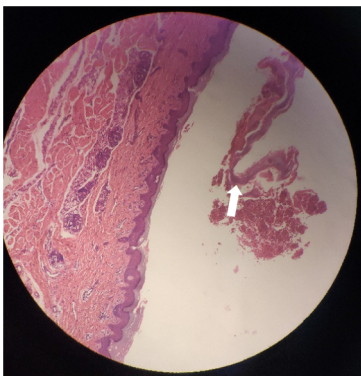


Figure 5 Skin biopsy of the infant showed another layer of epithelium over the normal epithelium with eosinophils and lymphocytes infiltration.

Additionally, anti-BP180 was positive (100 normal range < 20 IU/mL) and significantly higher than that of the mother. The diagnosis was confirmed by referencing the clinical history, histopathological examination and immunofluorescence examination of the mother and the infant. Neonatal pemphigoid is a self-healing disease, and most infants can resolve the skin lesions within 1 mo, so the treatment is mainly symptomatic treatment to prevent secondary infection of skin lesions. Short-term topical use of glucocorticoids may be considered for those with more frequent rashes. In this case, no special treatment was given to the infant, and the skin lesion subsided rapidly.

CONCLUSION

PG is a rarely reported disease, and 10% of newborns develop mild clinical symptoms consisting of urticaria-like or vesicular skin lesions[12]. We intend to remind clinicians to consider this condition when a patient presents with such lesions so that treatment

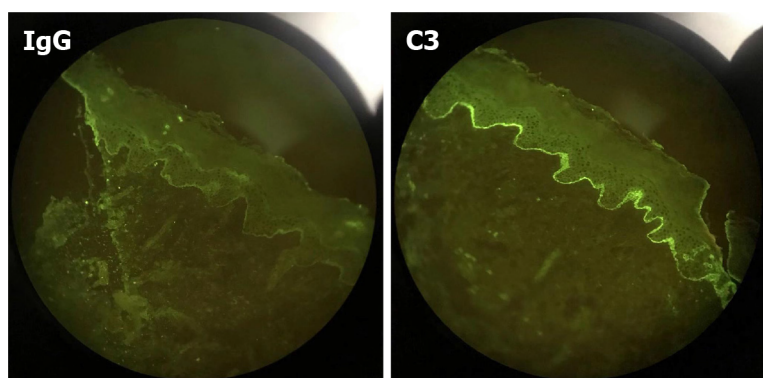


Figure 6 Direct immunofluorescence of the infant showed linear deposits of C3 along the basement membrane zone similar to her mother.



Figure 7 The seventh day after birth of the infant.

can be started early and neonatal morbidity can be taken into account.

In the management of pregnant women with PG, a multidisciplinary team should be assembled quickly to develop a treatment plan; obstetricians, dermatologists, pediatricians, and pathologists should collaborate in the multidisciplinary approach to treating maternal, fetal, and neonatal complications.

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