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ORIGINAL RESEARCH

Clinical Characteristics of Postpartum Women With Hypoxia: A Retrospective Analysis of 92 Cases

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Purpose: Postpartum hypoxia is a significant concern among clinicians due to its association with fatal diseases such as amniotic fluid embolism. This study analyzed the clinical characteristics of patients with different etiologies of postpartum hypoxia.

Patients and Methods: Ninety-two postpartum cases with hypoxia, defined as oxygen saturation (SpO_2) < 95% within 48 h postpartum without supplemental oxygen inhalation, and 100 normal gravidas were enrolled. All patients with postpartum hypoxia underwent 24 h vital sign monitoring and relevant examinations, including hematological tests, chest computed tomography (CT) scans, or CT pulmonary angiography, to determine the cause of hypoxia and received appropriate treatments. All patients were followed up for 1 month.

Results: Compared with normal gravidas, the patients with postpartum hypoxia had a higher occurrence rate of complications, including gestational hypertension (26.09% vs 8.00%), eclampsia (20.65% vs 4.00%), and a lower level of albumin (29.09 ± 0.57 vs 32.74 ± 0.94), thus tended to have longer hospitalization days (7.98 ± 0.40 vs 4.90 ± 0.16), with all $P < 0.05$. In all 92 cases, the most common cause of postpartum hypoxia was partial atelectasis with pleural effusion (65/92), followed by pulmonary edema (18/92), pneumonia (9/92), pulmonary embolism (6/92), and asthma (4/92). Among the five groups, patients with pneumonia had the longest hospital stay, whereas most patients with partial atelectasis accompanied by pleural effusion were asymptomatic. From the 1-month follow-up, all patients had a favorable prognosis with no apparent symptoms. Among those who underwent re-examination (27/92), no apparent imaging abnormalities were detected.

Conclusion: Postpartum hypoxia, which occurs more commonly in patients with gestational hypertension, is often caused by partial atelectasis with pleural effusion or pulmonary edema. The patient's prognosis was generally satisfactory after treatment.

Plain Language Summary: After giving birth, some women experience low oxygen levels, a condition known as postpartum hypoxia. This can be caused by various medical issues, such as fluid buildup in the lungs (pulmonary edema), collapsed lung areas (atelectasis), infections like pneumonia, or even blood clots in the lungs (pulmonary embolism). Detecting and treating this condition early is important to ensure the mother's recovery.

In this study, we analyzed the medical records of 92 women who developed postpartum hypoxia at a hospital in China. We compared these women to 100 healthy postpartum women to understand the causes and risk factors of this condition. The study found that the most common causes of postpartum hypoxia were partial lung collapse with fluid buildup (pleural effusion) and pulmonary edema. Many affected women also had pregnancy-related high blood pressure (gestational hypertension) or low protein levels in their blood (hypoproteinemia).

Doctors treated patients based on the cause of their hypoxia. Treatments included oxygen therapy, medications to remove excess fluid, antibiotics for infections, and blood thinners for clots. Most women recovered well within a short period.

This study highlights the importance of monitoring oxygen levels in postpartum women, especially those with risk factors like high blood pressure or low protein levels. The study also suggests that unnecessary imaging tests, which may expose breastfeeding infants to radiation, can sometimes be avoided with careful monitoring and treatment. More research is needed to improve guidelines for managing postpartum hypoxia.

Keywords: postpartum hypoxia, partial atelectasis, pulmonary edema, prognosis

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Introduction

Hypoxia is a common and critical condition in respiratory medicine and an important clinical manifestation of respiratory failure.[1] Hypoxia refers to insufficient oxygen content in the blood, characterized by an arterial oxygen partial pressure lower than the normal reference range (<80 mmHg).[2] Finger pulse oximeters are widely used as alternative methods for detecting peripheral blood oxygen saturation (SpO_2) in clinical practice due to their simplicity, noninvasiveness, and high accuracy.[3] However, pulse oximeters may have limited accuracy under certain conditions, such as motion artifacts, poor perfusion, and altered hemoglobin states.[3] Generally, $SpO_2 < 95\%$ is considered as hypoxemia.[4] In clinical practice, many respiratory diseases cause hypoxia, also known as respiratory failure. These diseases can be categorized into chronic conditions, such as chronic obstructive pulmonary disease and asthma, and acute conditions, such as severe pneumonia and pulmonary embolism (PE).[5,6] These conditions often indicate that the patient's condition is severe, requiring timely treatment.

In postpartum women, hypoxia may be associated with fatal diseases such as amniotic fluid embolism, which has an incidence rate of 1 in 40,000 to 1 in 120,000 deliveries and causes significant clinical concern due to its occult nature and high mortality.[7–9] Additionally, physiological changes during pregnancy, such as increased circulating blood volume, can predispose women to pathological conditions like pulmonary edema, pleural effusion, and atelectasis, which can contribute to hypoxia.[10,11] Therefore, timely identification and treatment of these causes are crucial. However, due to the particularity of the postpartum period, such as the high false-positive rate of D-dimer and the impact of contrast agents on breastfeeding, obstetricians should weigh the benefits and risks before choosing the appropriate examination method when treating patients with postpartum hypoxia.[12,13] For example, computed tomography (CT) pulmonary angiography may be avoided due to radiation exposure concerns in breastfeeding infants.

Given the complexity and potential urgency of postpartum hypoxia, a retrospective analysis allows us to leverage existing data to identify patterns and risk factors that may not be immediately apparent in prospective studies. Thus, this study retrospectively analyzed the clinical features of 92 patients with postpartum hypoxia, including etiology, clinical symptoms, imaging examinations, and laboratory tests. This study aims to provide obstetricians with a better understanding of the characteristics of postpartum hypoxia, thereby optimizing diagnosis and avoiding unnecessary investigations or treatments.

Materials and Methods

Subjects

The medical records of 40,564 pregnant women who gave birth in the obstetrics department of Xi'an People's Hospital (Xi'an No.4 hospital) between November 1, 2021, and October 31, 2024, were collected. Among these, 1705 patients who sought consultation from the respiratory department for various respiratory symptoms were identified. We included 92 patients with hypoxia (defined as $SpO_2 < 95\%$ within 48 h postpartum without supplemental oxygen inhalation, measured continuously by finger pulse oximeters) that was detected at least three times to exclude transient episodes. Additionally, 100 healthy participants were randomly selected for the control group, matched for age, gestational age, and delivery mode to ensure comparability.

Monitoring and Diagnosis

All patients with postpartum hypoxia underwent continuous or intermittent SpO_2 monitoring after operation until the SpO_2 rose to normal ($\geq 95\%$). To confirm the diagnosis and identify the underlying causes, patients underwent a series of examinations, such as hematological tests, chest CT scans, and CT pulmonary angiography, when clinically indicated. The specific choice of examination was guided by the clinical symptoms, signs, and suspected etiology (e.g pulmonary embolism, pneumonia, pleural effusion).

Main Treatment Approaches

All patients diagnosed with postpartum hypoxia received continuous nasal cannula or mask oxygen therapy (2–5 L/min) immediately to maintain adequate oxygenation, in addition to other relevant treatments based on the underlying cause. Treatments were tailored based on identified causes.

- Pulmonary Edema: Patients received protein supplementation (intravenous human serum albumin, 5 g/day) and diuretics (furosemide, 10 mg/day) for 2–3 days.
- Atelectasis with Pleural Effusion: Patients were encouraged to avoid prolonged bed rest. Patients with bilateral lower lung atelectasis and pleural effusion received protein supplementation (intravenous human serum albumin, 5 g/day) and diuretics (furosemide, 10 mg/day) if the area involved was > 30%.
- Pulmonary Embolism: Patients were treated with low molecular weight heparin calcium.
- Pneumonia: Antibiotics were administered to treat bacterial pneumonia. For pneumonia caused by COVID-19, treatment included antiviral therapy (Paxlovid, 5 days) combined with anti-inflammatory medicine (methylprednisolone, 40 mg/d, 3–5 days). Diagnostic confirmation was based on clinical symptoms, laboratory tests (e.g Polymerase chain reaction (PCR) for COVID-19), and imaging findings.

Oxygen therapy was discontinued when clinical symptoms improved and SpO_2 stabilized at ≥ 95% without supplemental oxygen.

Follow-up

All patients with postpartum hypoxia were followed up for 1 month after discharge. Assessments were conducted through phone interviews and in-person visits, and the main symptoms of all patients, such as cough and chest tightness, were recorded. Additionally, 27 patients were reexamined using chest CT or CT pulmonary angiography. Patient rights were safeguarded and privacy was protected throughout the study in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants after a detailed explanation of the study purpose, procedures, potential risks and benefits, and the right to withdraw at any time.

Statistical Analysis

Microsoft Excel and SPSS (version 26.0; IBM Corp., Armonk, NY, USA) were used for data collection and analysis. Count data, for example, the number of patients with underlying diseases, were displayed as numbers and percentages (%), and the chi-square test was used to compare variables among the three groups. If the expected count in any cell was ≥1 but <5, a continuity-adjusted chi-square test was applied, while Fisher's exact test was used for expected counts <1. Continuous variables, such as gestational age, were expressed as mean ± standard and analyzed using Student's *t*-test or one-way ANOVA for normally distributed data. The Mann–Whitney *U*-test was used for non-normally distributed variables. Statistical significance was set at $P < 0.05$ (two-tailed). Appropriate statistical tests were selected based on data type (categorical vs continuous) and the distribution characteristics to ensure robust analysis.

Results

Population Characteristics

This study enrolled 92 patients with postpartum hypoxia and 100 normal gravidas who were admitted during the same period. Compared to normal gravidas, patients with postpartum hypoxia had a lower gestational age (37.38 ± 0.34 vs 39.21 ± 0.15), a higher occurrence rate of complications, including gestational hypertension (26.09% vs 8.00%) and eclampsia (20.65% vs 4.00%), and a lower level of albumin (ALB, 29.09 ± 0.57 vs 32.74 ± 0.94), thus tended to have longer hospitalization days (7.98 ± 0.40 vs 4.90 ± 0.16) (all $P < 0.05$) (Table 1 and Figure 1). Additionally, before delivery, the patients in the postpartum hypoxia group usually had a higher expression of D-Dimer (2.45 ± 0.37 vs.1.25 ± 0.14) than the normal controls ($P < 0.05$). However, no statistical difference was observed between patients with postpartum hypoxia and normal cases in terms of age, ratio of cesarean section to vaginal delivery, number of

Table 1 Baseline Demographic and Clinical Characteristics of the Patients With Postpartum Hypoxia and Normal Puerperants

Characteristics	Postpartum Hypoxia (n=92, %)	Normal (n=100, %)	P value
Age	31.23±0.42	31.21±0.38	>0.05
Gestational weeks	37.38±0.34	39.21±0.15	<0.00*
Multiparous	6 (6.52)	2 (2.00)	>0.05
First pregnancy	86 (93.48)	87 (87.00)	>0.05
Multiple pregnancy	6 (6.52)	13 (13.00)	>0.05
Mode of delivery			
Cesarean section	90 (97.83)	98 (98.00)	>0.05
Vaginal deliver	2 (2.17)	2 (2.00)	>0.05
Underlying disease			
Gestational hypertension	24 (26.09)	8 (8.00)	<0.01*
Eclampsia	19 (20.65)	4 (4.00)	<0.00*
GDM	6 (6.52)	14 (14.00)	>0.05
Thyroid dysfunction	7 (7.61)	14 (14.00)	>0.05
Hospitalization days	7.98±0.40	4.90±0.16	<0.00*
Biochemical index (prenatal)			
WBC (*10^9/L)	9.49±0.70	9.07±0.54	>0.05
Neutrophil count (*10^9/L)	7.61±0.66	6.88±0.52	>0.05
CRP (mg/L)	5.89±1.45	3.12±0.66	>0.05
ALB (g/L)	29.09±0.57	32.74±0.94	<0.01*
Creatinine (μmol/L)	54.33±2.91	54.10±1.03	>0.05
D-Dimer (μmol/L)	2.45±0.37	1.25±0.14	<0.05*
Biochemical index (within 1 day postpartum)			
WBC (*10^9/L)	14.21±1.34	12.33±0.93	>0.05
Neutrophil count (*10^9/L)	12.06±1.42	10.25±0.94	>0.05
CRP (mg/L)	66.70±12.91	99.18±11.64	>0.05
D-dimer (μmol/L)	3.91±0.69	3.50±0.80	>0.05

Note: *A two-tailed P<0.05 means statistically significance.
Abbreviations: GDM, Gestational diabetes mellitus; WBC, White blood cell; CRP, C-reactive protein; ALB, Albumin.

parturitions, multifetal pregnancies, other associated comorbidities such as gestational diabetes mellitus (GDM), thyroid dysfunction, and laboratory indicators including white blood cells (WBC), neutrophils, C-reactive protein (CRP), and creatinine (prenatal and/or within 1 day postpartum). All statistical tests were adjusted for multiple comparisons to account for the increased likelihood of Type I errors.

Laboratory examinations in Different Groups With Postpartum Hypoxia

We further divided patients with postpartum hypoxia into five groups according to the different causes of the disease. Etiological analysis revealed that, in all 92 cases with postpartum hypoxia, the main causes were partial atelectasis accompanied by pleural effusion (65/92), followed by pulmonary edema (18/92), pneumonia (9/92), PE (6/92), asthma (4/92) (Figure 2 and Table 2). Ten cases presented with partial atelectasis, pleural effusion, and pulmonary edema simultaneously. Patients with partial atelectasis accompanied by pleural effusion were largely asymptomatic (53/65, 81.54%). In contrast, patients with pulmonary edema, pneumonia, or asthma often experienced coughing (9/18, 50%; 7/9, 77.78%; 3/4, 74%) and chest tightness (16/18, 88.89%; 5/9, 55.56%; 4/4,100%). Additionally, chest tightness was a common symptom in patients with PE (5/6, 83.33%) ($P < 0.05$). Fever was also prevalent among patients with pneumonia (6/9, 66.67%) ($P < 0.05$). Regarding the length of hospital stay, patients with pneumonia had a significantly longer hospital stay than the other four groups (Figure 1). Additionally, in a subset of patients with pulmonary edema, B-type natriuretic peptide (BNP) levels decreased significantly within 48 h postpartum (Figure 3). However, among patients with postpartum hypoxia, no clear associations were observed between etiologies and differential expressions of

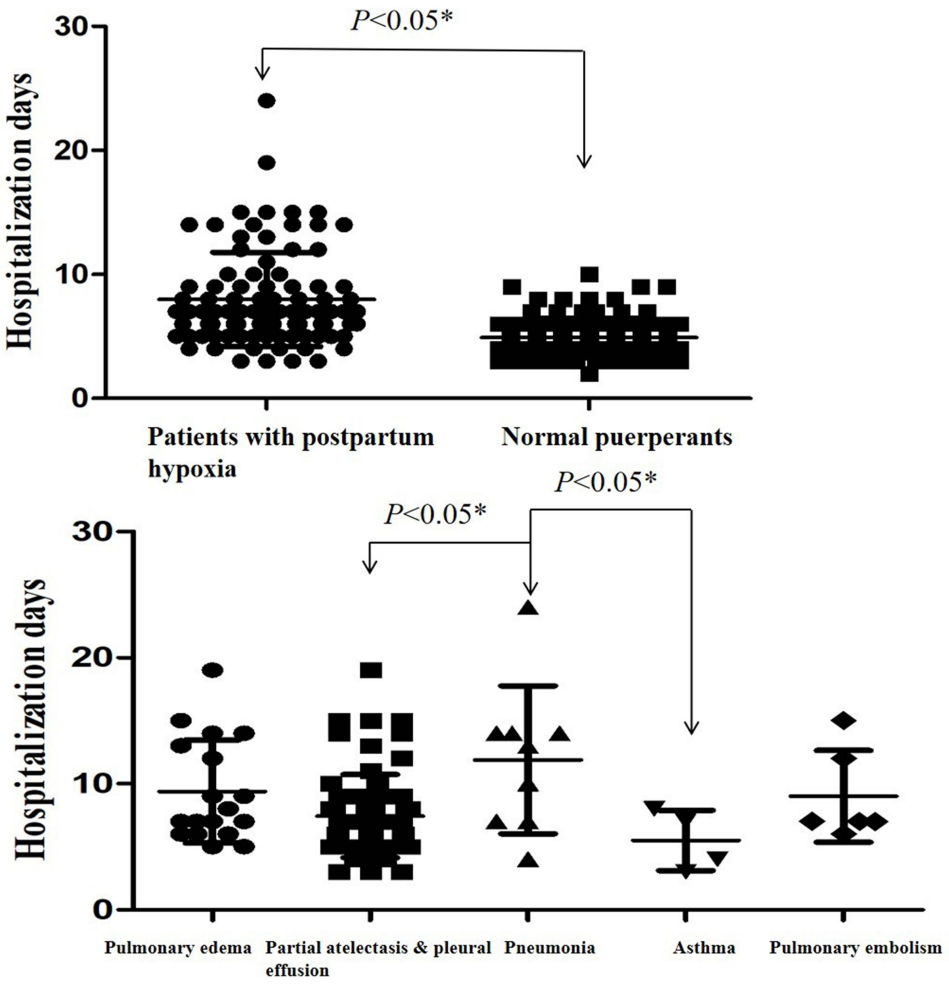

Figure 1 Comparison of hospitalization days between patients with postpartum hypoxia and normal postpartum women, as well as among patients with postpartum hypoxia due to different causes. The symbol * represents a statistically significant difference.

common laboratory examination indices, including WBC, neutrophil, CRP, ALB, Creatinine (prenatal and within 1 day postpartum), and D-dimer (prenatal).

Follow-up (1 Month)

From the 1-month follow-up, based on patient self-reports, all patients had a favorable prognosis with no obvious symptoms or signs. Among the 92 patients, 27 underwent re-examination via chest CT or CT pulmonary angiography (CTPA) after 1 month. Of these, 12 showed normal results, whereas 15 exhibited minor residual abnormalities.

Discussion

This study explored the common causes of postpartum hypoxia and analyzed the clinical characteristics of patients with various etiological factors, including symptom presentation, laboratory findings, and treatment outcomes. From the data, partial atelectasis, often occurring on the dorsal side of the lower lung, along with pleural effusion followed by pulmonary edema, were the main causes of postpartum hypoxia in our cohort. Patients with hypoxia caused by these two etiologies tend to have a high incidence of gestational hypertension with or without eclampsia and hypoproteinemia. However, no statistically significant differences were found in the expression of laboratory indicators frequently used in clinics, such as D-dimer, WBC count, and CRP, and their dynamic changes. Atelectasis occurring on the dorsal side of the lower lung, accompanied by pleural effusion and pulmonary edema in pregnancy, can arise from different etiologies. Multiple factors, such as increased circulating blood volume, reduced plasma protein concentrations, pregnancy-induced

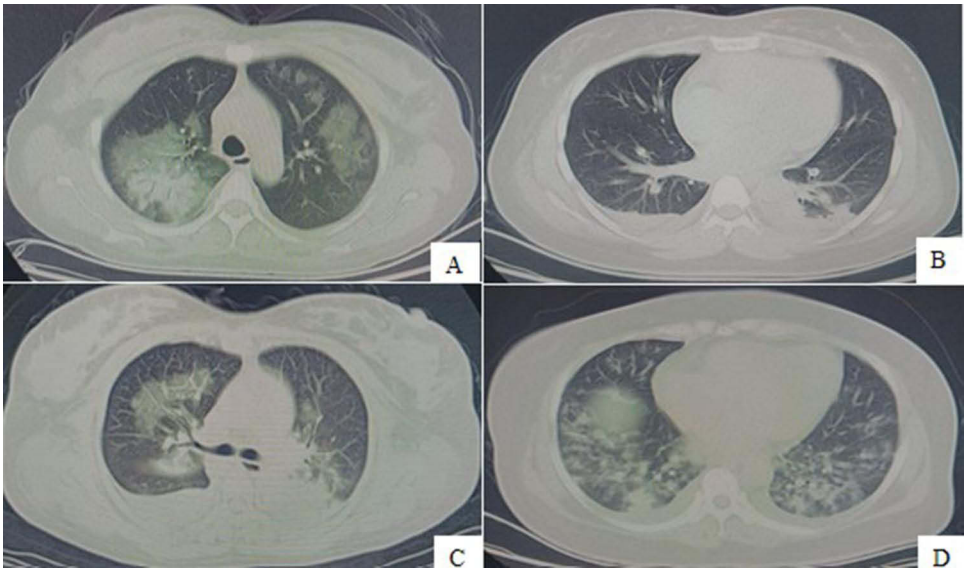

Figure 2 A 28-year-old parturient with a bigeminal pregnancy and severe hypertension during pregnancy experienced significant shortness of breath. After delivery, her oxygen saturation (SpO_2) was 89% without supplemental oxygen, and chest computed tomography (CT) revealed pulmonary edema (**A**); A 32-year-old parturient was asymptomatic, with an SpO_2 of 92% without supplemental oxygen after delivery. Chest CT showed mild atelectasis in both lower lungs with minimal pleural effusion (**B**); A 30-year-old parturient reported chest tightness and shortness of breath. Her SpO_2 was 90% without supplemental oxygen after delivery, and chest CT revealed concurrent partial atelectasis, pleural effusion, and pulmonary edema (**C**); Another 30-year-old parturient developed postpartum fever and cough, with an SpO_2 of 91% without supplemental oxygen. Chest CT indicated pneumonia (**D**).

hypertension, decreased vascular resistance, and relative cardiac insufficiency, make pregnant women susceptible to these two complications.[14] Patients with pulmonary edema have more clinical symptoms than those with partial atelectasis and often require medication and longer hospital stays. In a study by O'Dwyer SL et al, the main symptoms in patients with pulmonary edema during pregnancy and puerperium were chest pain (4/53), paroxysmal nocturnal dyspnea (5/53), and orthopnea (17/53).[15] The positivity rate for these symptoms was lower than that in this study population. The drugs used in their study included magnesium sulfate (26/53), dihydralazine (17/53), nifedipine (10/53), and labetalol (2/53); however, most patients with pulmonary edema did not receive any of these drugs. In this study, the most commonly used antihypertensive drug was labetalol (100 mg, q8). For most postpartum patients, due to a significant decrease in circulating blood volume, combined treatments with short-term protein supplementation and diuretics can result in a marked improvement in localized pulmonary edema, atelectasis, and pleural effusion (often within 48 h). Potential explanations for these discrepancies may include differences in study populations, diagnostic criteria, or treatment protocols. O'Dwyer et al included patients with pulmonary edema during pregnancy and puerperium, whereas this study focused specifically on postpartum hypoxia. Additionally, variations in the severity of pulmonary edema and timing of symptom assessment may have contributed to the observed differences.

Additionally, pregnant and postpartum women are at increased risk of venous thromboembolism (VTE), including deep vein thrombosis (DVT) and PE, due to a combination of hypercoagulability, progesterone-induced venous stasis, local trauma to pelvic veins during delivery, and prolonged immobility or bed rest, particularly in the postpartum period,[16–18] having a 5 to 10-fold higher risk than non-pregnant women of comparable age. Relevant statistics show that the absolute risk of VTE is 0.1% antepartum and 0.05% postpartum, while the actual day-to-day risk increases 15 to 35-fold postpartum, with the highest risk in the first 6 weeks, and pregnancy-associated PE is approximately 1 in 1000–3000.[19] For example, a retrospective study conducted by Galambosi et al found that VTE had an incidence rate of approximately 1.8 in 1000 deliveries at 0–180 days postpartum, and the incidence risk was the highest during the first week postpartum: 37-fold compared to non-pregnant women, declining to two-fold immediately after that.[20] As PE is often related to hypoxia and is considered a life-threatening, serious complication in pregnant and postpartum women, postpartum hypoxia attracts attention from both obstetricians and respiratory physicians.[21] However, the necessity for CTPA in patients with postpartum hypoxia remains unclear. Radiopharmaceuticals are secreted into the breast, resulting

Table 2 Baseline Demographic and Clinical Characteristics of the Postpartum Hypoxia Patients With Different Causes

Characteristics	Partial Atelectasis along with Pleural Effusion (n=65)	Pulmonary Edema (n=18)	Pneumonia (n=9)	Pulmonary Embolism (n=6)	Asthma (n=4)	P value
Age	30.33±3.27	31.44±4.20	31.00±3.24	29.17±1.72	33.75±3.86	>0.05
Gestational weeks	35.49±4.14	37.59±2.99	37.21±2.71	37.51±4.72	35.50±5.40	>0.05
Multiparous	7 (10.77)	5 (27.78)	0 (0.00)	0 (0.00)	0 (0.00)	>0.05
First pregnancy	61 (93.85)	18 (100.00)	8 (88.89)	5 (83.33)	4 (100.00)	>0.05
Multiple pregnancy	4 (6.15)	0 (0.00)	1 (11.11)	1 (16.67)	0 (0.00)	>0.05
Cesarean section	63 (96.92)	18 (100.00)	9 (100.00)	6 (100.00)	4 (100.00)	>0.05
Underlying disease						
Gestational hypertension	20 (30.77)	8 (44.44)	0 (0.00)	1 (16.67)	0 (0.00)	>0.05
Eclampsia	14 (21.54)	6 (33.33)	0 (0.00)	1 (16.67)	0 (0.00)	>0.05
Gestational diabetes mellitus	2 (3.08)	2 (11.11)	1 (11.11)	0 (0.00)	1 (25.00)	>0.05
Thyroid dysfunction	6 (9.23)	2 (11.11)	0 (0.00)	1 (16.67)	0 (0.00)	>0.05
Hospitalization days	7.43±3.30	9.39±4.09	11.89±5.86	9.00±3.63	5.50±2.38	<0.01*
Clinical symptoms						
Asymptomatic	53 (81.54)	2(11.11)	0 (0.00)	0 (0.00)	0 (0.00)	<0.01*
Cough	10 (15.38)	9 (50.00)	7 (77.78)	0 (0.00)	3 (75.00)	<0.01*
Fever	3 (46.15)	1 (5.56)	6 (66.67)	0 (0.00)	0 (0.00)	<0.01*
Chest tightness	8 (12.31)	16 (88.89)	5 (55.56)	5 (83.33)	4 (100.00)	<0.01*
Biochemical index (prenatal)						
WBC (*10^9/L)	7.62±1.08	9.70±2.91	11.94±2.76	10.33±5.19	10.09±2.26	>0.05
Neutrophil count (*10^9/L)	5.57±1.08	7.77±2.81	7.25±3.03	7.78±3.15	8.49±1.70	>0.05
CRP (mg/L)	3.83±2.96	3.58±2.13	12.33±6.32	4.92±1.35	10.42±3.72	>0.05
ALB (g/L)	29.23±3.65	29.13±3.47	26.55±4.03	29.68±3.26	29.48±1.71	>0.05
Creatinine (μmol/L)	53.45±6.35	53.17±8.11	53.18±4.57	55.69±6.24	49.25±2.89	>0.05
D-Dimer (μmol/L)	2.64±1.02	2.99±1.20	2.36±0.87	2.46±1.80	2.04±0.98	>0.05
Biochemical index (within 1 day postpartum)						
WBC (*10^9/L)	12.04±2.99	16.12±6.52	14.77±3.93	14.94±9.54	11.83±2.44	>0.05
Neutrophil count (*10^9/L)	10.04±2.56	13.78±7.25	10.08±5.25	13.18±4.93	9.94±1.67	>0.05
CRP (mg/L)	54.99±12.66	68.90±22.35	126.24±46.84	40.12±12.48	61.62±14.44	<0.05*
D-dimer (μmol/L)	4.00±2.26	4.35±2.41	2.47±0.54	4.77±3.62	3.51±1.34	>0.05

Note: *A two-tailed P<0.05 means statistically significance.
Abbreviations: WBC, White blood cell; CRP, C-reactive protein; ALB, Albumin.

in radiation exposure in infants who ingest milk. Radiologists recommend avoiding the administration of radiopharmaceuticals due to potential radiation exposure in breastfeeding infants.[22] Therefore, based on expert opinion, we recommend prophylactic low-molecular-weight heparin (LMWH) for patients with stable vital signs and no obvious clinical symptoms. Instead, CTPA examination is suggested to be completed if the SpO_2 still does not rise satisfactorily within 48–72 h. This approach aims to balance the risk of VTE with the potential harm caused by radiation exposure during imaging studies. However, further research is needed to validate this recommendation and to develop more accurate and comprehensive clinical guidelines for the management of postpartum hypoxia. Moreover, for patients with postpartum hypoxia caused by other causes, such as pneumonia and asthma, inquiring about disease history, symptom analysis, physical examination, and infection marker detection is necessary.

To our knowledge, this is the first reported study to focus on patients with postpartum hypoxia and analyze the clinical characteristics of patients with different etiological factors. However, this study has some shortcomings. First, not all pregnant women, especially asymptomatic women, require routine vital sign testing before delivery. Therefore, the exact timing for the occurrence of hypoxia remains uncertain for all patients and is only detected in symptomatic patients or through routine electrocardiographic monitoring after a cesarean section. Hypoxia may also occur during pregnancy in some patients. Our future research will aim to include more perinatal women to obtain more comprehensive and accurate clinical data. Second, our study was limited to a 1-month follow-up period. This timeframe may not capture the full spectrum of the long-term health implications for affected patients. Conditions such as gestational diabetes increase the

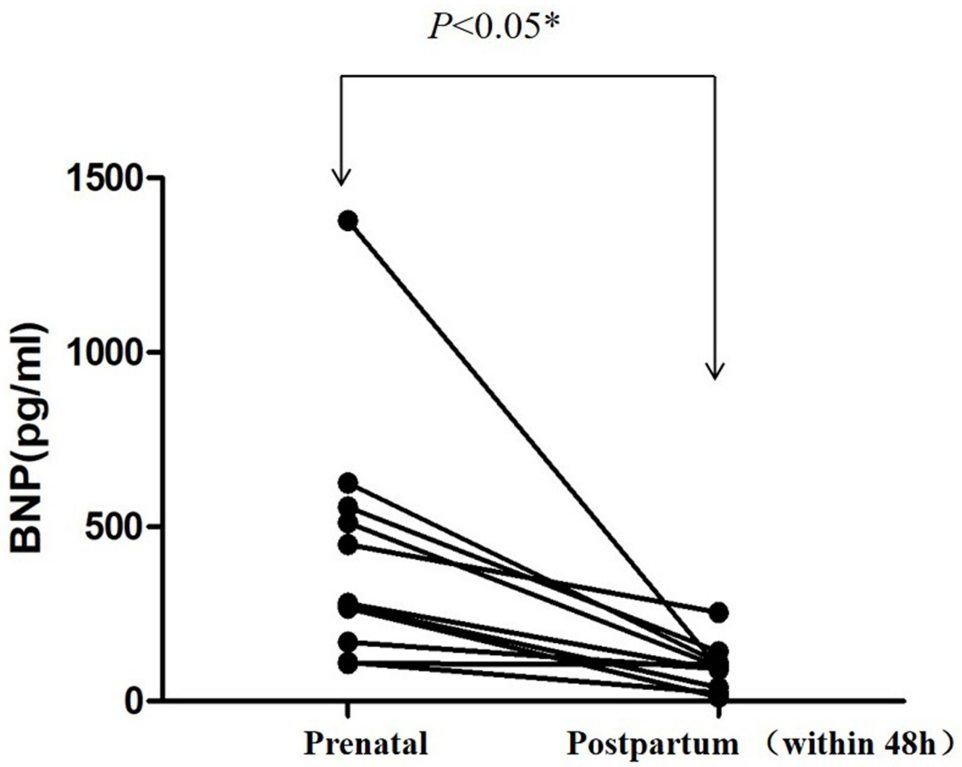

Figure 3 Changes in B-type natriuretic peptide (BNP) levels before delivery and within 48 h postpartum in 11 puerperal patients with pulmonary edema. The symbol *represents a statistically significant difference.

long-term risk of developing diabetes mellitus,[23] highlighting the potential for chronic sequelae following acute postpartum complications. Future studies should consider extended follow-up periods to better understand the chronic effects of postpartum hypoxia and to inform long-term management strategies. Third, this study had a retrospective design, and its population was selected from a single-center cohort, which may cause potential biases in the data and limit the generalizability of our findings.

Conclusions

This study identified partial atelectasis with pleural effusion and pulmonary edema as the main causes of postpartum hypoxia, which commonly occurs in patients with gestational hypertension and hypoproteinemia. The prognosis is always satisfactory after treatment.

Abbreviations

SpO_2, oxygen saturation; COVID-19, Corona Virus Disease 2019; WBC, white blood cells; PLT, platelet; VTE, venous thromboembolism; DVT, deep vein thrombosis; PE, pulmonary embolism; GDM, gestational diabetes mellitus; PCR, polymerase chain reaction; BNP, B-type natriuretic peptide; CRP, C- reactive protein; ALB, Albumin; LMWH, low-molecular-weight heparin; SPSS, statistical package for the social sciences; ANOVA, analysis of variance.

Data Sharing Statement

The datasets used and/or analyzed in the current study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The protocols were approved by the Ethics Committee of Xi'an People's Hospital (Xi'an No.4 hospital), and informed consent was obtained from the patients or their guardians. All procedures involving human participants were performed in accordance with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Consent for Publication

Written informed consent was obtained from all patients for the publication of any potentially identifying images and clinical details of this study.

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Disclosure

The authors report no potential competing interest in this work.

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