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Research Article

A Complex High-Risk Pregnancy with Second Trimester Superimposed Preeclampsia Following Vanishing Twin Syndrome in a Patient with Previous Severe Preeclampsia and Cesarean Section: A Case Report

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Abstract: Hypertensive disorders of pregnancy remain a major cause of maternal and perinatal morbidity and mortality. Superimposed preeclampsia is particularly challenging because it occurs in women with pre-existing chronic hypertension and is associated with increased risk of severe maternal and fetal complications. Vanishing twin syndrome may further increase obstetric risk, especially when associated with abnormal placentation and maternal vascular dysfunction. This case report describes a 35-year-old woman, gravida 5 para 3 abortus 1, at 21 weeks of gestation, who was admitted with severe nausea and vomiting. The pregnancy was complicated by hyperemesis gravidarum without dehydration, superimposed preeclampsia, previous severe preeclampsia, previous cesarean section, vanishing twin syndrome, and mild hypokalemia. Laboratory evaluation showed proteinuria +1, anemia, and hypokalemia with serum potassium of 3.1 mmol/L. The patient was managed with intravenous fluid therapy, antiemetics, gastric protection, potassium correction, supplementation, antibiotics, and close maternal-fetal monitoring. This case highlights the importance of early identification, multidisciplinary surveillance, and individualized management in high-risk pregnancies with overlapping maternal and placental risk factors.

Keywords: High-Risk Pregnancy; Hyperemesis Gravidarum; Hypokalemia; Superimposed Preeclampsia; Vanishing Twin Syndrome.

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1. Introduction

Hypertensive disorders of pregnancy remain major causes of maternal and perinatal morbidity and mortality worldwide. Preeclampsia is a complex multisystem disorder characterized by abnormal placentation, endothelial dysfunction, and an exaggerated maternal inflammatory response. It affects approximately 2-8% of pregnancies and contributes to adverse outcomes including preterm birth, fetal growth restriction, and maternal organ dysfunction (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; Phipps et al., 2016).

Superimposed preeclampsia, defined as preeclampsia developing in a woman with chronic hypertension, is clinically challenging because it is associated with increased maternal and perinatal risk. A previous history of preeclampsia is an important risk factor for recurrence and warrants intensified antenatal surveillance. Patients with chronic hypertension, previous severe preeclampsia, and a prior cesarean delivery require individualized assessment because maternal vascular risk and obstetric considerations coexist (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019). Vanishing twin syndrome (VTS) is the spontaneous loss and subsequent resorption of one conceptus from an initially recognized multiple gestation. Although early VTS is often regarded as a self-limited event, available studies associate it with adverse

outcomes in the surviving singleton, particularly preterm birth, lower birth weight, and fetal growth abnormalities. The mechanisms remain uncertain and may reflect abnormal early embryonic or placental development (National Institute for Health and Care Excellence (NICE, 2019; Seong et al. 2020).

The coexistence of VTS and superimposed preeclampsia presents a distinctive clinical challenge because both may involve abnormal placental development and impaired maternal vascular adaptation; however, a direct causal relationship has not been established. Additional complications such as hyperemesis gravidarum and electrolyte imbalance may further complicate clinical decision-making. Hyperemesis gravidarum can be accompanied by nutritional deficiencies, dehydration, ketonuria, and electrolyte disturbances, including hypokalemia, requiring careful monitoring and correction (Phipps et al., 2016; Batsry & Yinon, 2022; Royal College of Obstetricians and Gynaecologists [RCOG], 2024; Committee on Practice Bulletins–Obstetrics, 2018).

This case report describes a complex high-risk pregnancy in a G5P3A1 patient at 21 weeks of gestation with a history of severe preeclampsia and previous cesarean section who developed second-trimester superimposed preeclampsia following vanishing twin syndrome. The aim of this report is to highlight the diagnostic challenges, clinical considerations, and importance of multidisciplinary surveillance in managing pregnancies with multiple overlapping maternal and obstetric risk factors.

A 35-year-old woman, gravida 5 para 3 abortus 1 (G5P3A1), was admitted to Siloam Sentosa Hospital on May 25, 2026, at 21 weeks of gestation with complaints of severe nausea and vomiting. The patient was diagnosed with a high-risk pregnancy complicated by hyperemesis gravidarum (HEG) without dehydration, superimposed preeclampsia, previous severe preeclampsia, one previous cesarean section, vanishing twin syndrome, and mild hypokalemia. The patient was conscious and fully oriented upon admission, with adequate communication. She reported nausea and vomiting more than 10 times per day, characterized by watery vomitus without blood. Approximately five hours prior to admission, the patient experienced a transient loss of consciousness at around 12:00 PM, lasting approximately 10 minutes, without associated seizure activity. There was no history of headache, visual disturbances, epigastric pain, chest pain, or palpitations.

The patient reported a history of fever one day prior to admission, which had resolved at presentation. She also complained of productive cough for two days with whitish sputum, without dyspnea, nasal congestion, or activity-related worsening. Gastrointestinal and urinary functions were otherwise normal.

The patient had a significant obstetric history. Her first pregnancy was complicated by severe preeclampsia, resulting in vaginal delivery at 28 weeks of gestation; the neonate subsequently died. Her second pregnancy was also complicated by severe preeclampsia and resulted in vaginal delivery at 32 weeks of gestation with a surviving neonate. Her third pregnancy was delivered by cesarean section at 40 weeks of gestation due to oligohydramnios, with a living child. The fourth pregnancy ended in abortion in 2022. She had no history of diabetes mellitus, cardiac disease, asthma, renal disease, or autoimmune disorders. The current pregnancy was initially documented as a twin gestation. On April 20, 2026, ultrasonography at Primaya Hospital Bekasi Timur demonstrated two fetuses with markedly discordant development.

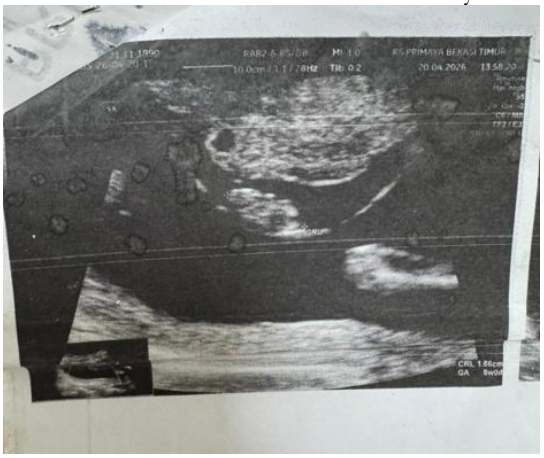

Figure 1. Smaller embryonic structure at approximately 8 weeks of gestation

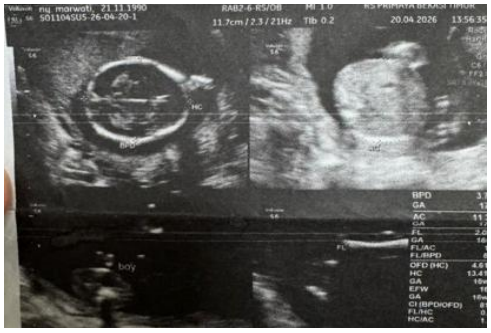

Figure 2. Biometric assessment of the larger fetus.

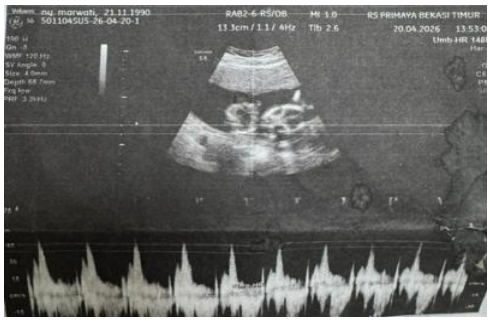

Figure 3. Fetal heart rate assessment of the larger fetus.

Figure 3. Initial ultrasonography performed at Primaya Hospital Bekasi Timur on April 20, 2026. (A) The smaller embryonic structure measured a crown-rump length of 1.66 cm, corresponding to approximately 8 weeks of gestation. (B) The larger fetus demonstrated biometric measurements corresponding to approximately 16 weeks of gestation, with an estimated fetal weight of 166 g. (C) Cardiac activity of the larger fetus was recorded at 148 beats per minute.

Fetal evaluation showed discordant gestational development, with fetus A measuring approximately 8 weeks of gestation based on crown-rump length (CRL 1.66 cm), while fetus B measured approximately 16 weeks and 4 days based on biometric parameters, including biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC), femur length (FL), and estimated fetal weight (EFW 166 g). Due to uncertainty regarding the viability and status of the twin pregnancy, the patient was referred to a fetomaternal specialist at Cipto Mangunkusumo Hospital (RSCM) for further evaluation.

On May 13, 2026, follow-up ultrasonography at RSCM confirmed a singleton viable intrauterine pregnancy at 18 weeks and 6 days of gestation, with normal fetal activity. Biometric measurements included BPD 4.62 cm, HC 17.43 cm, AC 14.72 cm, FL 3.15 cm, HL 2.99 cm, and estimated fetal weight of 321 g, with an expected date of delivery of October 8, 2026. The findings were consistent with a diagnosis of vanishing twin syndrome, in which one fetus from an initially documented twin gestation was no longer visualized and a single viable fetus remained. The follow-up fetomaternal ultrasonographic findings are shown in Figure 4.

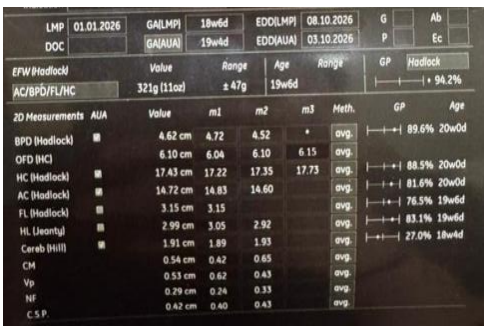

Figure 4. Fetal biometry at 18 weeks and 6 days of gestation

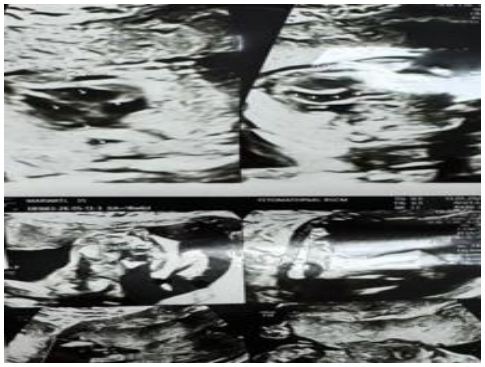

Figure 5. Selected anatomical views of the surviving fetus.

Figure 5. Follow-up fetomaternal ultrasonography performed at 18 weeks and 6 days of gestation showing a singleton viable fetus, fetal biometric measurements, and selected anatomical views, including the four-chamber cardiac view, left ventricular outflow tract, palate, foot, urinary bladder, and stomach.

On admission to Siloam Sentosa Hospital, physical examination revealed a body weight of 59 kg. Obstetric examination showed uterine fundal height corresponding to gestational age. Leopold maneuver was not performed due to gestational age below 24 weeks. Linea nigra, striae gravidarum, and a Pfannenstiel surgical scar from previous cesarean delivery were noted.

During hospitalization, serial blood pressure monitoring demonstrated fluctuating hypertension, with several systolic blood pressure measurements reaching the severe range. The serial maternal blood pressure measurements are presented in Figure 5.

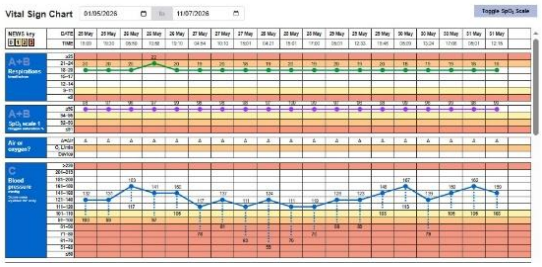

Figure 6. Serial maternal blood pressure measurements during hospitalization from May 25 to May 31, 2026, demonstrating fluctuating hypertension with several elevated systolic blood pressure readings.

Laboratory evaluation revealed mild hypokalemia (3.3 mmol/L) without evidence of significant dehydration. Further evaluation for maternal complications related to preeclampsia was performed, including complete blood count, electrolyte profile, and urine protein assessment. Complete blood count revealed anemia, with hemoglobin of 9.1 g/dL, hematocrit of 28.1%, and erythrocyte count of 3.0×10^6/μL. Leukocyte count was 10.3×10^3/μL and platelet count was 329×10^3/μL, indicating no thrombocytopenia. The urinalysis result is shown in Figure 6.

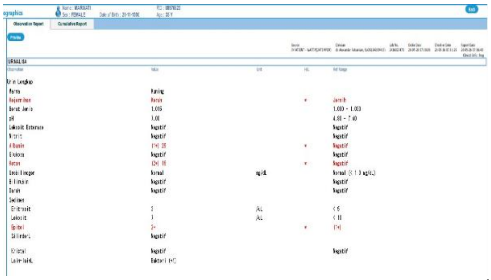

Figure 7. Urinalysis report showing turbid yellow urine, albumin/protein +1, ketone +2, epithelial cells 2+, and bacteria +1, with negative glucose, nitrite, leukocyte esterase, bilirubin, and blood.

Urinalysis showed turbid yellow urine with albumin/protein +1, ketone +2, erythrocytes 3/μL, leukocytes 7/μL, epithelial cells 2+, and bacteria +1, while glucose, nitrite, leukocyte esterase, bilirubin, and blood were negative. Electrolyte examination showed sodium 131 mmol/L, potassium 3.1 mmol/L, and chloride 98 mmol/L. The serum electrolyte profile is shown in Figure 7.

Figure 8. Serum electrolyte profile showing mild hyponatremia with sodium 131 mmol/L and mild hypokalemia with potassium 3.1 mmol/L, while chloride remained within the normal range at 98 mmol/L.

These findings supported the presence of mild electrolyte imbalance and proteinuria without laboratory evidence of thrombocytopenia. Liver and renal function test results were not documented in the available data.

Based on the clinical presentation, obstetric history, and supporting investigations, the patient was diagnosed with G5P3A1 at 21 weeks of gestation complicated by hyperemesis gravidarum without dehydration, superimposed preeclampsia, previous severe preeclampsia, previous cesarean section, vanishing twin syndrome, and mild hypokalemia.

The patient was managed with multidisciplinary high-risk pregnancy surveillance. Treatment included intravenous fluid replacement with Ringer's lactate and normal saline, antiemetic therapy consisting of ondansetron and metoclopramide, gastric protection with ranitidine and sucralfate, electrolyte correction with intravenous potassium chloride followed by oral potassium supplementation, and antihypertensive therapy with nifedipine. Calcium carbonate, folic acid, and vitamin B6 supplementation were also administered. Due to respiratory symptoms and previous fever, ceftriaxone and N-acetylcysteine were administered during hospitalization.

Potassium replacement therapy was initiated on May 25, 2026, using intravenous potassium chloride supplementation and continued until May 27, 2026, with subsequent oral potassium supplementation using potassium chloride sustained release. Ceftriaxone therapy was administered from May 25 to May 29, 2026. Maternal and fetal conditions were monitored closely due to the increased risk of disease progression associated with superimposed preeclampsia in the second trimester and the patient's significant obstetric history.

2. Discussion

The present case describes a clinically challenging combination of VTS and suspected superimposed preeclampsia in a pregnant woman with chronic hypertension, previous severe preeclampsia, and a prior cesarean delivery. This scenario represents a convergence of maternal vascular and placental risk factors. Although VTS is often considered a transient event, evidence associates it with selected adverse perinatal outcomes; whether it independently contributes to hypertensive disease remains uncertain (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; Batsry & Yinon, 2022; Seong et al., 2020; Sun et al., 2017; Zhou et al., 2020).

Preeclampsia is a complex multisystem disorder originating primarily from abnormal placentation and impaired maternal adaptation to pregnancy. Inadequate trophoblastic invasion and incomplete spiral-artery remodeling can reduce uteroplacental perfusion, promote placental ischemia and oxidative stress, and increase the release of antiangiogenic mediators. The resulting angiogenic imbalance, including increased soluble fms-like tyrosine kinase-1 and reduced placental growth factor activity, contributes to systemic endothelial dysfunction and the clinical manifestations of preeclampsia (Batsry & Yinon 2022).

In this case, suspected superimposed preeclampsia may reflect the interaction between pre-existing maternal vascular dysfunction and pregnancy-related placental abnormalities. Chronic hypertension is associated with endothelial dysfunction, impaired vascular remodeling, placental insufficiency, fetal growth restriction, and an increased risk of superimposed preeclampsia. Guidelines emphasize accurate blood pressure assessment, appropriate antihypertensive treatment, laboratory evaluation, and close maternal-fetal surveillance (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019).

A previous history of severe preeclampsia further increases recurrence risk in subsequent pregnancies. This susceptibility may reflect persistent cardiovascular risk, genetic and immunologic factors, and recurrent placental dysfunction. Women with previous early or severe disease therefore require individualized risk assessment and intensified antenatal monitoring,

particularly when chronic hypertension is also present (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020).

VTS refers to the spontaneous loss and resorption of one fetus from an initially recognized multiple gestation. Although early VTS has historically been considered relatively benign, cohort studies and meta-analyses report increased risks of preterm birth, lower birth weight, and growth-related adverse outcomes in some surviving singletons. These findings suggest that VTS may be a marker of abnormal early embryonic or placental development rather than an isolated fetal event (Batsry & Yinon, 2022; Seong et al., 2020; Sun et al., 2017; Zhou et al., 2020).

A direct association between VTS and preeclampsia remains unproven. Nevertheless, a shared placental hypothesis is biologically plausible: early fetal demise and abnormal placentation may alter inflammatory and angiogenic signaling, while pre-existing maternal vascular vulnerability may amplify endothelial dysfunction. This interpretation should be regarded as hypothesis-generating rather than causal (Phipps et al., 2016; Batsry & Yinon, 2022).

One major clinical challenge is differentiating worsening chronic hypertension from superimposed preeclampsia. Assessment should integrate serial blood pressure measurements, new or increasing proteinuria, thrombocytopenia, renal or hepatic dysfunction, neurological symptoms, pulmonary edema, and evidence of uteroplacental dysfunction. Prompt recognition is important because progression may result in HELLP syndrome, placental abruption, maternal organ dysfunction, fetal growth restriction, and preterm delivery (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019).

Management was further complicated by hyperemesis gravidarum and electrolyte imbalance. Persistent vomiting may cause dehydration, nutritional deficiency, ketonuria, hyponatremia, and hypokalemia. Careful fluid replacement, antiemetic treatment, electrolyte correction, and nutritional support are therefore central components of management (Royal College of Obstetricians and Gynaecologists [RCOG], 2024; Committee on Practice Bulletins–Obstetrics, 2018). A previous cesarean delivery also influences obstetric decision-making. The timing and mode of delivery in a patient with chronic hypertension or superimposed preeclampsia must balance maternal indications, fetal condition and maturity, disease severity, and factors related to the uterine scar. When deterioration occurs remote from term, multidisciplinary assessment is required to determine whether expectant management remains appropriate or delivery is safer (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019). This case has several clinical implications. First, VTS should not automatically be treated as an incidental finding because it may be associated with adverse perinatal outcomes and may justify closer surveillance of the surviving fetus [6-9]. Second, chronic hypertension combined with previous severe preeclampsia places the patient in a high-risk category requiring proactive monitoring and individualized management (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019).Third, overlapping maternal and placental risks support multidisciplinary collaboration involving maternal-fetal medicine specialists, obstetricians, internists, anesthesiologists, and neonatologists.

Although a direct causal relationship between VTS and superimposed preeclampsia cannot be established from a single case, abnormal placental development and dysregulated maternal vascular adaptation may represent shared pathways. Further research is required to determine whether VTS independently predicts hypertensive pregnancy disorders, especially in women with pre-existing cardiovascular risk factors (Phipps et al., 2016; Batsry & Yinon, 2022; Seong et al., 2020; Sun et al., 2017; Zhou et al., 2020).

In conclusion, this case represents a high-risk pregnancy complicated by interacting maternal and placental abnormalities. The coexistence of VTS, chronic hypertension, previous severe preeclampsia, and suspected superimposed preeclampsia requires vigilant surveillance, early recognition of deterioration, and individualized multidisciplinary management (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; Batsry & Yinon, 2022).

4. Diagnosis and Management

Diagnosis

Based on the patient's history, physical examination, obstetric history, and supporting investigations, the patient was diagnosed as G5P3A1 at 21 weeks of gestation with a high-risk pregnancy, complicated by hyperemesis gravidarum without significant dehydration, second-

trimester superimposed preeclampsia, history of severe preeclampsia in previous pregnancies, previous cesarean section, vanishing twin syndrome, mild hypokalemia, mild hyponatremia, and anemia in pregnancy.

The diagnosis of hyperemesis gravidarum was supported by severe nausea and vomiting more than 10 times per day, accompanied by urinary ketones (+2). Although the patient had persistent vomiting, there was no clear clinical evidence of significant dehydration. Electrolyte imbalance included mild hypokalemia (3.1 mmol/L) and mild hyponatremia (131 mmol/L) (Royal College of Obstetricians and Gynaecologists [RCOG], 2024; Committee on Practice Bulletins–Obstetrics, 2018).

Superimposed preeclampsia was considered on the basis of the patient's hypertensive course, high-risk obstetric background, previous severe preeclampsia, and protein/albumin +1 on urinalysis after 20 weeks of gestation. However, a dipstick result of +1 alone does not establish significant proteinuria; diagnostic confirmation should integrate documented chronic hypertension, serial blood pressure measurements, quantitative urinary protein assessment, and evidence of maternal organ or uteroplacental dysfunction. The platelet count remained normal at $329 \times 10^3/\mu L$, with no thrombocytopenia (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019).

Hematologic evaluation revealed anemia, with hemoglobin of 9.1 g/dL, hematocrit of 28.1%, and erythrocyte count of $3.0 \times 10^6/\mu L$ [12]. Leukocyte count was $10.3 \times 10^3/\mu L$, and platelet count was $329 \times 10^3/\mu L$. Urinalysis showed turbid yellow urine, specific gravity of 1.015, pH 7.0, albumin/protein +1, ketone +2, erythrocytes 3/μL, leukocytes 7/μL, epithelial cells 2+, and bacteria +1.

Management

The patient was managed as a high-risk pregnancy with a multidisciplinary approach. Initial management focused on maternal stabilization, correction of electrolyte imbalance, control of nausea and vomiting, and close monitoring for progression of preeclampsia.

Intravenous Ringer's lactate and normal saline were administered to maintain hydration and fluid balance. Fluids were given cautiously because hypertensive disorders of pregnancy increase the risk of fluid overload and pulmonary edema, with regular monitoring of vital signs, urine output, and clinical status (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019).To control nausea and vomiting, the patient received ondansetron and metoclopramide. Gastrointestinal protection was provided with ranitidine and sucralfate, while folic acid, vitamin B6, and calcium carbonate were administered as supportive supplementation (Royal College of Obstetricians and Gynaecologists [RCOG], 2024; Committee on Practice Bulletins–Obstetrics, 2018).

Mild hypokalemia was corrected with intravenous potassium chloride during hospitalization, followed by sustained-release oral potassium chloride after discharge. Correction was required because repeated vomiting can cause potassium depletion and clinically important electrolyte disturbance (Royal College of Obstetricians and Gynaecologists [RCOG], 2024; Committee on Practice Bulletins–Obstetrics, 2018).

Because the patient had a history of fever and productive cough with whitish sputum, she was treated with ceftriaxone during hospitalization and N-acetylcysteine as supportive therapy for respiratory symptoms. After clinical improvement, antibiotic therapy was continued with cefadroxil 500 mg as discharge medication.

During hospitalization, the patient was monitored for blood pressure, level of consciousness, pulse, temperature, fluid balance, and symptoms of worsening preeclampsia, including severe headache, visual disturbance, epigastric pain, dyspnea, seizure, and reduced fetal movement. Laboratory monitoring included complete blood count, electrolytes, urinalysis, and renal and liver function testing when available. Fetal surveillance was performed according to gestational age and obstetric indications (American College of Obstetricians and Gynecologists [ACOG], 2019, 2020; National Institute for Health and Care Excellence [NICE], 2019).

5. Conclusion

This case illustrates a complex high-risk pregnancy with second trimester superimposed preeclampsia following vanishing twin syndrome in a patient with a previous history of severe preeclampsia and cesarean section. The coexistence of hyperemesis gravidarum, mild hypokalemia, mild hyponatremia, anemia, and proteinuria further increased the complexity of clinical management. The patient's previous history of severe preeclampsia, together with the finding

of albumin/protein +1 on urinalysis after 20 weeks of gestation, emphasized the need for close maternal and fetal surveillance. Although there was no laboratory evidence of thrombocytopenia or severe maternal organ dysfunction in the available data, the patient remained at high risk for disease progression due to multiple overlapping obstetric and maternal risk factors.

This case highlights the importance of early risk identification, careful interpretation of laboratory findings, correction of electrolyte imbalance, and multidisciplinary management in pregnancies complicated by superimposed preeclampsia and vanishing twin syndrome. Further follow-up is essential to monitor maternal condition, fetal growth, blood pressure control, and possible progression of preeclampsia throughout pregnancy. Although a direct causal relationship between vanishing twin syndrome and superimposed preeclampsia cannot be established from a single case, this report supports the need for heightened clinical awareness when vanishing twin syndrome occurs in patients with pre-existing maternal vascular risk factors or a history of severe preeclampsia.

Acknowledgments

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