CASE REPORT | OBSTETRICS & GYNECOLOGY

Placental Compromise and Severe Intrauterine Growth Restriction in the Surviving Co-Twin After Early Vanishing Twin Syndrome: A Case Report

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Abstract

Background: Vanishing Twin Syndrome (VTS)—the early spontaneous loss of one fetus in a multifetal gestation—occurs in up to 36% of twin pregnancies assisted by reproductive technology and approximately 10–15% of spontaneous twin pregnancies. While historically considered a benign event for the surviving co-twin, emerging evidence indicates that VTS can significantly impair residual placental function and fetal growth trajectories.

Case presentation: This report describes a primigravida who presented with early first-trimester ultrasound confirmation of a dichorionic-diamniotic (DCDA) twin pregnancy. Subsequent follow-up imaging demonstrated complete spontaneous reabsorption of one twin sac, indicating VTS. While antenatal monitoring continued as a single gestation, progressive fetal growth restriction (FGR) and altered umbilical artery (UA) Doppler indices were identified in the surviving fetus during the late second and third trimesters. Serial growth tracking and obstetric management resulted in the delivery of a very low birth weight (VLBW) infant at late term without major acute neonatal morbidities.

Conclusion: This case highlights that VTS should not be treated as a routine single gestation once twin disappearance is confirmed. Heightened biophysical and Doppler surveillance throughout the second and third trimesters is essential to detect latent FGR and optimize timing of delivery.

Keywords: Vanishing Twin Syndrome; Fetal Growth Restriction; Intrauterine Growth Restriction; Multiple Gestation; Placental Insufficiency; Case Report

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Introduction

Vanishing Twin Syndrome (VTS) is defined as the early spontaneous loss and partial or complete reabsorption of one fetus or gestational sac in a multifetal pregnancy [1]. First described in the pre-ultrasound era via gross placental examination, the widespread adoption of routine early first-trimester ultrasonography and assisted reproductive technologies (ART) has markedly increased its clinical recognition [1]. Current epidemiological estimates indicate that VTS occurs in approximately 10% to 15% of spontaneous twin gestations and in up to 36% of twin pregnancies conceived via ART [2].

Historically, early first-trimester embryonic loss was considered a benign clinical event for the surviving co-twin, operating under the assumption that early reabsorption allows the remaining fetus to proceed as an uncomplicated singleton pregnancy [2]. However, growing epidemiological and clinical evidence demonstrates that VTS is associated with heightened risks of adverse perinatal outcomes [2, 3]. Studies comparing singleton pregnancies resulting from VTS with initial singleton gestations have identified increased incidences of extremely preterm birth, low birth weight

(LBW), small for gestational age (SGA) status, and structural or neurodevelopmental complications in the surviving fetus [2, 3].

The underlying pathophysiology linking early twin demise to late fetal growth restriction (FGR) in the survivor remains a subject of ongoing investigation [3]. Several interconnected mechanisms have been hypothesized to explain this phenomenon. First, in dichorionic gestations, the demised gestational sac and its associated trophoblastic tissue may leave a persistent area of non-functional, infarcted, or fibrotic placental tissue, thereby reducing the effective functional surface area available for nutrient transfer and gas exchange to the surviving fetus during late gestation [3]. Second, the release of necrotic tissue products or inflammatory cytokines from the vanishing twin into the surrounding maternal-fetal circulation may induce localized microvascular thrombosis or impair normal spiral artery remodeling [3]. Finally, in unrecognized monochorionic gestations, acute hemodynamic shifts following embryonic demise can precipitate transient fetal hypotension or altered Doppler perfusion indices in the surviving co-twin [3]. Despite these recognized risk factors, standardized international consensus guidelines regarding the frequency and duration of antenatal surveillance following a diagnosed vanishing twin remain limited [3]. Once the loss of one sac is confirmed on early ultrasound, clinicians frequently reclassify the pregnancy as a low-risk singleton, potentially missing subtle or late-onset FGR.

This case report documents a primigravida who experienced first-trimester VTS followed by progressive, late-trimester FGR and altered umbilical artery (UA) Doppler dynamics in the surviving twin, resulting in a very low birth weight (VLBW) infant delivered at late term. By detailing the clinical, imaging, and Doppler trajectory of this pregnancy, this report underscores the critical necessity of maintaining serial biophysical and Doppler surveillance throughout the second and third trimesters in pregnancies complicated by early multifetal reduction.

Case Presentation

Patient History and Initial Early Evaluation

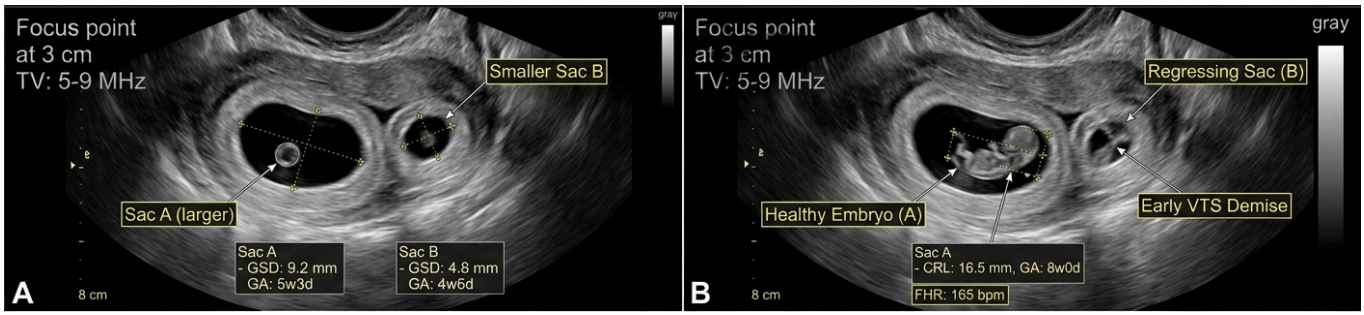
A 36-year-old primigravida presented to the antenatal outpatient clinic following a positive home pregnancy test shortly after her missed period. Her medical, surgical, and family histories were unremarkable, with no pre-existing maternal systemic hypertension, pre-gestational diabetes mellitus, autoimmune conditions (such as antiphospholipid syndrome or systemic lupus erythematosus), or underlying renal pathology. The pregnancy was conceived spontaneously without the use of ovulation induction agents or ART. Maternal baseline body mass index (BMI) was slightly elevated at 25.7 kg/m^2 (overweight category), and she denied any history of tobacco, alcohol, or illicit substance exposure.

An early transvaginal ultrasound (TVUS) performed at 5 weeks and 3 days of gestation (calculated from her last menstrual period) confirmed an early dichorionic-diamniotic (DCDA) twin gestation with two distinct intrauterine gestational sacs. Gestational Sac A demonstrated a crown-rump length (CRL) corresponding to 5 weeks and 3 days with a regular fetal heart rate (FHR) of 115 bpm and a normal yolk sac. Gestational Sac B displayed a smaller, lagging gestational sac with a visible fetal pole corresponding to 4 weeks and 6 days; a fetal heartbeat was detected, but it was noticeably weaker and bradycardic (FHR 82 bpm), raising clinical concern for impending early embryonic demise. Two separate implantation sites separated by a clear, thick chorionic membrane were visualized, establishing dichorionicity.

Confirmation of Vanishing Twin Syndrome

The patient returned for a scheduled follow-up ultrasound at 8 weeks of gestation. Ultrasound examination revealed a single, normally developing fetus (Twin A) with a CRL corresponding to 8 weeks and 1 day and a robust FHR of 164 bpm. Twin B and its gestational sac had completely regressed and were no longer identifiable on ultrasound, confirming early first-trimester VTS (Figure 1). Given her advanced maternal age (36 years), the genetic and diagnostic implications of early VTS on non-invasive prenatal testing (NIPT) and cell-free DNA (cfDNA) screening were discussed with the patient, as apoptotic cfDNA from the demised twin can persist in the maternal circulation for several weeks and potentially confound aneuploidy screening results. The patient remained completely asymptomatic throughout this period, reporting no vaginal bleeding, pelvic cramping, spotting, or passage of tissue per vaginum. Because the vanished twin was completely reabsorbed early in the first trimester without maternal clinical compromise, routine antenatal care proceeded under the assumption of a low-risk singleton pregnancy.

Figure 1. Serial Transvaginal Sonograms Demonstrating Early Co-Twin Demise.



(A) Transvaginal ultrasonography at 5 weeks, 3 days of gestation establishing a dichorionic-diamniotic (DCDA) twin pregnancy with two distinct gestational sacs. (B) Follow-up transvaginal scan at 8 weeks, 0 days reveals complete arrest and spontaneous resorption of Twin B (Vanishing Twin Syndrome), alongside a single viable fetus in Gestational Sac A with appropriate biometric progress.

Mid-Trimester Antenatal Surveillance

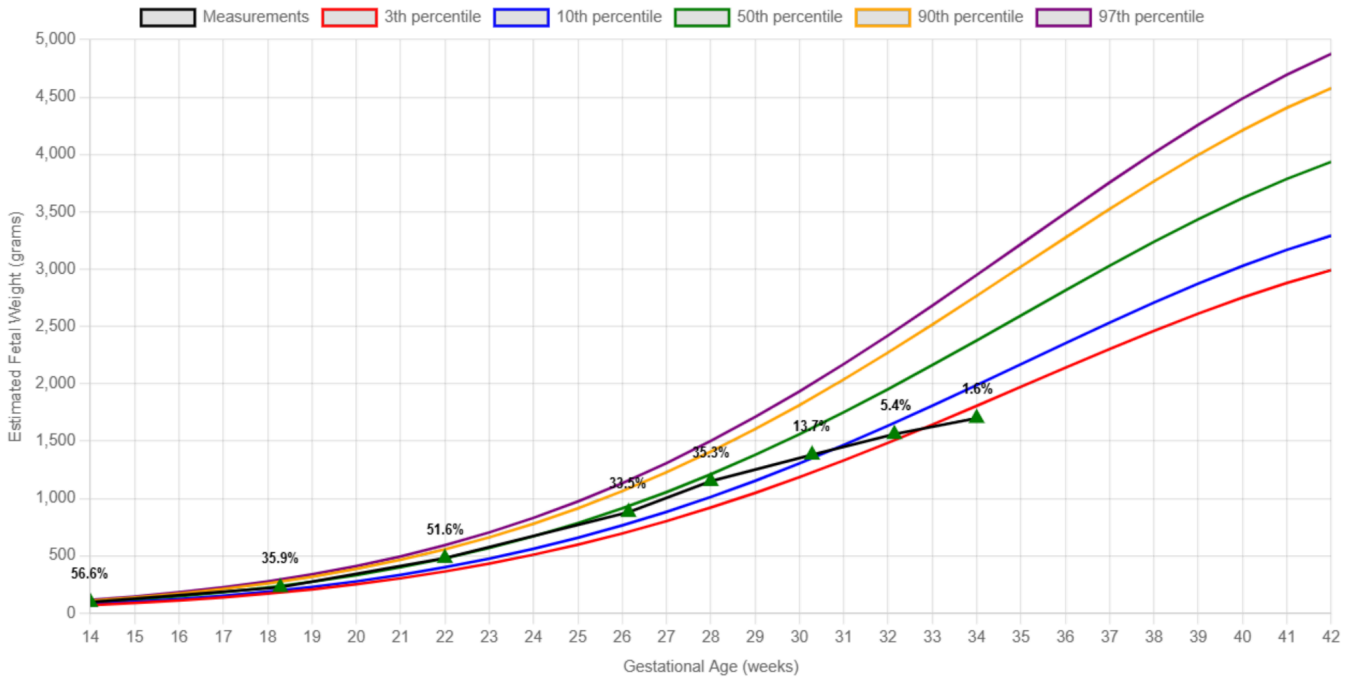
A detailed fetal anatomical survey scan conducted at 20 weeks of gestation showed no structural anomalies or soft markers in the surviving fetus. Fetal biometry—including head circumference (HC), abdominal circumference (AC), biparietal diameter (BPD), and femur length (FL)—was fully congruent with gestational age (GA), plotting within the 45th percentile on standardized Hadlock growth curves [4]. Placental evaluation demonstrated a single anterior placenta without sonographic signs of abruption, placenta previa, or structural anomalies, while the regressed tissue of the demised twin was no longer sonographically identifiable.

However, routine follow-up ultrasonography at 28 weeks of gestation revealed early growth divergence. While HC and FL remained appropriate for GA at the 35th percentile, the fetal AC had dropped to the 14th percentile, indicating an early asymmetric pattern of growth restriction. Color Doppler evaluation of the UA demonstrated a Pulsatility Index (PI) within normal limits for GA (1.02), and amniotic fluid volume remained adequate with a single deepest pocket (SDP) of 4.2 cm.

Third-Trimester Growth Restriction and Doppler Trajectory

By 34 weeks of gestation, progressive and severe late-onset FGR was established (Figure 2). The estimated fetal weight (EFW) fell to 1,580 g, placing the fetus at the 4th percentile for GA. Serial color and spectral Doppler examination of the UA demonstrated markedly elevated downstream vascular resistance, with the UA-PI rising to 1.42 (exceeding the 95th percentile for 34 weeks), though end-diastolic flow remained positive without reversal or absence. Spectral Doppler of the middle cerebral artery (MCA) revealed compensatory cerebrovascular vasodilatation (the "brain-sparing effect"), yielding a reduced cerebroplacental ratio (CPR < 1.0) (Table 1).

Figure 2. Longitudinal Estimated Fetal Weight Trajectory Demonstrating Late-Onset Fetal Growth Restriction.



Serial ultrasound measurements from 14 to 34 weeks of gestation plotted against standardized population reference percentiles (3rd, 10th, 50th, 90th, and 97th). The fetus exhibited normal growth velocity tracking along the 50th percentile through the second trimester (51.6% at 22 weeks), followed by progressive growth deceleration. Acute crossing of percentile curves was observed after 28 weeks, falling below the 10th percentile at 32 weeks (5.4%) and reaching severe fetal growth restriction by 34 weeks, with an estimated fetal weight of 1,580 g at the 4th percentile.

Maternal blood pressure readings (consistently <120/80 mmHg), routine urine dipstick tests (negative for proteinuria), and comprehensive maternal laboratory workup—including liver enzymes, serum creatinine, uric acid, and platelet counts—remained strictly within normal reference ranges throughout the third trimester. This ruled out maternal preeclampsia, Hemolysis, Elevated Liver enzymes, Low Platelets (HELLP) syndrome, or gestational hypertension as secondary maternal causes of placental insufficiency. Serial biophysical profiles (BPP) were performed twice weekly alongside non-stress testing (NST), consistently yielding scores of 8/10 or 10/10 with normal cardiotocography (CTG) reactivity and baseline variability.

Table 1. Longitudinal Antenatal Ultrasound & Doppler Trajectory

Gestational Age	Fetal Parameters / EFW	Percentile	UA Doppler (UA-PI)	MCA / CPR	Amniotic Fluid / BPP	Clinical Interpretation
5w 3d	Twin A: CRL 5w3d, FHR 115; Twin B: CRL 4w6d, FHR 82	N/A	Normal flow	N/A	Normal dual sacs	Early DCDA twin gestation; weak FHR in Twin B
8w 0d	Single viable fetus (Twin A)	N/A	Normal flow	N/A	Single gestational sac	Confirmed VTS; complete resolution of Twin B
20w 0d	HC, AC, BPD, FL appropriate	45th	Normal	Normal	Normal SDP (4.2 cm)	Normal mid-trimester structural survey
28w 0d	Asymmetric growth lag (AC)	14th	1.02 (Normal)	Normal	Normal SDP (4.2 cm)	Early asymmetric growth divergence
34w 0d	EFW 1,580 g	4th	1.42 (>95th percentile)	CPR <1.0 (Brain-sparing)	BPP 8/10 or 10/10	Severe late-onset FGR & placental insufficiency

41w 2d	Birth weight 1,700 g	<1st	N/A	N/A	Clear fluid at SROM	Emergency LUSCS for failure of descent/failure to progress; VLBW/SGA neonate

Legend: Longitudinal tracking of fetal biometry, estimated fetal weight percentiles, and spectral Doppler indices across gestation. Growth percentiles are calculated based on standard Hadlock fetal growth curves. Bolded values at 34 weeks highlight the onset of severe fetal growth restriction (EFW at the 4th percentile) accompanied by placental insufficiency (UA-PI = 1.42, > 95th percentile) and compensatory cerebral hemodynamics (CPR < 1.0). Delivery was achieved via emergency Lower Uterine Segment Cesarean Section at 41 weeks due to failure of labor progress.
Abbreviations: AC: Abdominal Circumference; BPD: Biparietal Diameter; BPP: Biophysical Profile; CPR: Cerebroplacental Ratio; CRL: Crown-Rump Length; DCDA: Dichorionic-Diamniotic Twin Gestation; EFW: Estimated Fetal Weight; FGR: Fetal Growth Restriction; FL: Femur Length; HC: Head Circumference; LUSCS: Lower Uterine Segment Cesarean Section; MCA: Middle Cerebral Artery; SDP: Single Deepest Pocket; UA-PI: Umbilical Artery Pulsatility Index; VTS: Vanishing Twin Syndrome.

Delivery, Neonatal Management, and Placental Histopathology

Because maternal clinical parameters, BPP scoring, and Doppler surveillance remained reassuring without absent or reversed end-diastolic flow (AREDF), conservative outpatient management with close surveillance was maintained until late term. At 41 weeks and 2 days of gestation, the patient experienced spontaneous rupture of membranes (SROM) with clear amniotic fluid, followed shortly by the onset of regular, painful uterine contractions.

Upon presentation to the labor unit, digital cervical examination revealed an unfavorable cervix with an unengaged, high-station fetus (-3 station) remaining high in the uterine cavity. Despite active uterine labor, there was no progression of cervical dilation or fetal descent into the pelvic inlet over an extended observation period. Due to failure of fetal descent and failure to progress following membrane rupture at 41 weeks and 2 days, the decision was made to perform an emergency lower uterine segment Cesarean section (LUSCS).

Surgery proceeded under spinal anesthesia without intraoperative complications, delivering a live female infant. The neonate weighed 1,700 g (<1st percentile for 41 weeks of gestation) and demonstrated a symmetrically reduced crown-heel length (42 cm), classifying her as VLBW and severe SGA. Appearance, Pulse, Grimace, Activity, Respiration (Apgar) scores were 7 and 9 at 1 and 5 minutes, respectively. Physical examination at birth confirmed a globally small and short neonate with significantly reduced subcutaneous fat reserves, but with intact skin integrity and no abdominal abnormalities, dysmorphic features, or acute respiratory distress.

The neonate was immediately admitted to the Neonatal Intensive Care Unit (NICU) for specialized VLBW care, thermal support in a closed incubator, and close metabolic surveillance. Initial laboratory investigations revealed normal serum glucose levels (58 mg/dL) without hypoglycemia, normal serum electrolytes, and a CBC showing a hemoglobin of 16.2 g/dL and hematocrit of 49%, excluding polycythemia or hyperviscosity syndrome. A routine screening cranial ultrasound performed on postnatal day 3 demonstrated normal intracranial architecture without intraventricular hemorrhage (IVH) or hypoxic-ischemic encephalopathy (HIE). Minimal enteral trophic feeds with fortified express breast milk were initiated on day 2 and advanced smoothly without feed intolerance or signs of necrotizing enterocolitis (NEC).

Gross examination of the placenta revealed a markedly small, thin single disk weighing 280 g (<3rd percentile for GA). Formal histopathologic analysis confirmed a distinct peripheral region exhibiting focal villous infarction, avascular stromal villi, perivillous fibrin deposition, and localized intervillous thrombosis, representing the retained, fibrotic trophoblastic remnant of the vanished co-twin. Following steady daily weight gain, complete temperature stability in an open crib, and achievement of full oral bottle/breast feedings, the neonate was discharged home in stable condition on postnatal day 12 weighing 1,850 g (Table 2).

Table 2. Chronological Case Synopsis, Sonographic Tracking, and Clinical Evolution

Gestational Stage	Key Clinical Events & Monitoring	Sonographic & Biometric Findings	Hemodynamic & Pathological Status	Clinical Management & Outcome
First Trimester (5w3d–8w0d)	Patient remained asymptomatic; confirmed DCDA twin gestation.	5w3d: Sac A & Sac B visible with embryo poles; 8w0d: spontaneous demise/resorption Twin B	Acute localized perivillous fibrin deposition and focal microvascular thrombosis at Twin B site	Conservative management; serial ultrasound surveillance initiated.
Second Trimester (14w0d–26w1d)	Routine antenatal surveillance; patient asymptomatic with normotensive BP.	Normal appropriate growth velocity tracking along 50th percentile (EFW 95 g at 14w to 880 g at 26w).	Normal maternal-fetal Doppler hemodynamics; normal AFI.	Routine outpatient follow-up maintained.
Early Third Trimester (28w0d–30w2d)	Routine growth assessment.	Initial growth deceleration; EFW drops from 42nd percentile (1,150 g) at 28w to 18th percentile (1,380 g) at 30w.	UA-PI remained within normal limits at 28 weeks (1.02); amniotic fluid volume remained adequate.	Monitoring increased to bi-weekly.
Late Third Trimester (32w1d–34w0d)	Outpatient evaluation reveals severe growth lag; criteria met for late-onset FGR.	34w0d: EFW 1,580 g, 4th percentile; severe late-onset FGR established.	Elevated UA-PI (1.42, >95th percentile) with preserved positive end-diastolic flow; reduced CPR <1.0 consistent with brain-sparing.	Close outpatient surveillance with twice-weekly BPP and NST was continued.
Delivery & Postnatal (41w2d)	SROM at 41w2d followed by labor; emergency Cesarean performed for failure of fetal descent and failure to progress.	Birth weight 1,700 g, <1st percentile; VLBW and severe SGA.	Placental gross pathology: peripheral mummified fibrous remnant of vanished co-twin with focal ischemic villous infarction.	Successful delivery; NICU supportive care/growth recovery.

Legend: Comprehensive longitudinal summary of maternal-fetal surveillance, growth trajectory, hemodynamic status, and clinical management from first-trimester Vanishing Twin Syndrome (VTS) presentation to term delivery and placental pathology.
Abbreviations: AC: Abdominal Circumference; AFI: Amniotic Fluid Index; BP: Blood Pressure; BPD: Biparietal Diameter; DCDA: Dichorionic-Diamniotic; EFW: Estimated Fetal Weight; FGR: Fetal Growth Restriction; FL: Femur Length; GA: Gestational Age; HC: Head Circumference; NICU: Neonatal Intensive Care Unit; UA: Umbilical Artery; VLBW: Very Low Birth Weight; VTS: Vanishing Twin Syndrome.

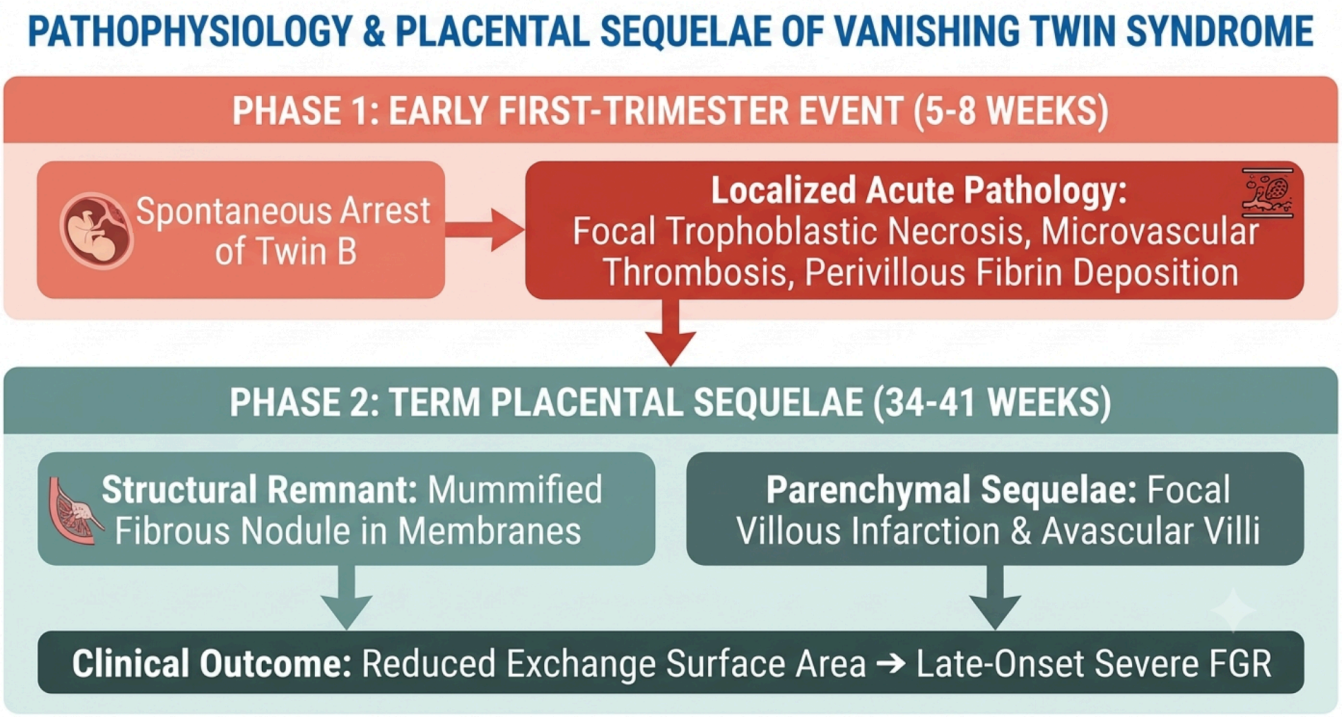
Discussion

Vanishing Twin Syndrome, while historically regarded as a benign early-pregnancy event in dichorionic gestations, is increasingly recognized as a clinical marker for latent placental dysfunction and fetal growth impairment in the surviving co-twin. This case demonstrates a profound trajectory of severe late-onset FGR, resulting in a late-term VLBW (1,700 g) neonate following very early first-trimester embryonic loss at 5 weeks of gestation.

Pathophysiological Mechanisms of Placental Compromise

The primary physiological question raised by this case is how an embryonic loss occurring as early as 5 weeks of gestation—with complete sonographic reabsorption by 8 weeks—can precipitate severe, late-trimester placental insufficiency. Several interconnected mechanisms help explain this association. First, in dichorionic gestations, early trophoblastic tissue occupies significant uterine mucosal surface area during initial implantation. Following embryonic demise, the regressed sac and adjacent chorion undergo focal ischemia, hyalinization, and fibrosis rather than complete, functional tissue restoration [5, 6]. As demonstrated by the gross and microscopic placental pathology in this case—a total placental weight of only 280 g (<3rd percentile) with focal peripheral villous infarction, avascular stromal villi, perivillous fibrin deposition, and intervillous thrombosis—the effective functional surface area available for terminal villous branching and nutrient transfer during late gestation was permanently compromised (Figure 3) [7, 8].

Figure 3. Conceptual Pathophysiological Flowchart of Vanishing Twin Syndrome and Placental Sequelae.



A structural schematic mapping the multi-stage biological progression linking early co-twin demise to late-stage placental dysfunction. The initial phase illustrates early first-trimester arrest of Twin B triggering localized acute pathology, including focal trophoblastic necrosis, microvascular thrombosis, and perivillous fibrin deposition. The second phase depicts long-term placental sequelae, where mummified fibrous remnants persist within extraembryonic membranes while parenchymal damage presents as focal villous infarction and avascular villi. The cumulative loss of functional maternal-fetal exchange surface area leads to secondary chronic placental insufficiency and severe late-onset fetal growth restriction.

Second, early trophoblastic invasion of maternal spiral arteries by the demised gestational sac may be incomplete or disrupted [9]. The resulting localized inflammatory and microvascular thrombotic cascades at the implantation bed can impair physiological remodeling of adjacent uterine vessel networks [9, 10]. This raises downstream vascular resistance, directly accounting for the elevated umbilical artery Pulsatility Index (UA-PI > 95th percentile) and compensatory brain-sparing response (reduced CPR < 1.0) observed from 34 weeks onward [11, 12]. Finally, competitive early allocation of uterine endometrial territory by two distinct implantation sites can restrict the physical expansion zone of the surviving twin's placenta, leaving behind a small-for-gestational-age placenta incapable of supporting the exponential metabolic demands of late third-trimester growth [12].

Maternal Risk Factors and Clinical Implications for Antenatal Surveillance

Current obstetric protocols frequently reclassify pregnancies complicated by early VTS as low-risk singletons once the demised sac is no longer visible on ultrasound. However, this case illustrates that complete sonographic reabsorption of the vanished twin by 8 weeks does not equal complete pathophysiological resolution of the underlying placental unit. A single mid-trimester anomaly scan at 20 weeks may display entirely normal biometry, as seen in this patient whose fetal growth initially plotted at the 45th percentile. Because growth divergence often manifests subtly in the late second or early third trimester, maintaining routine serial growth scans at 4-week intervals throughout the third trimester should be considered for all post-VTS gestations [13, 14].

Furthermore, integrating UA and MCA Doppler surveillance is essential for detecting early hemodynamics of placental insufficiency, allowing clinicians to distinguish true pathologically growth-restricted fetuses from constitutionally small ones [13, 14]. Additionally, given the patient's age (36 years), clinicians must remain mindful of the genetic screening implications of VTS. Persistent cfDNA released from the apoptotic trophoblasts of the demised twin can circulate in maternal blood for several weeks, potentially confounding NIPT results [15].

Labor, Delivery, and Perinatal Outcomes

Severe FGR secondary to chronic placental insufficiency is frequently accompanied by reduced intrapartum fetal reserve and dysfunctional labor dynamics [15, 16]. In this patient, the combination of late-term SROM, failure of fetal descent with an unengaged fetus remaining high in the uterine cavity (-3 station), and lack of cervical progress necessitated an emergency Cesarean section. This presentation highlights how chronic placental compromise can alter labor biomechanics and fetal engagement, reinforcing the need for continuous intrapartum monitoring and low thresholds for operative delivery in post-VTS pregnancies complicated by severe late-onset growth restriction [15, 16]. Specialized NICU admission for thermal regulation, glycemic surveillance, and nutritional fortification remains essential to achieve successful catch-up growth and safe discharge in these neonates.

Conclusions

In summary, this case demonstrates that early first-trimester VTS—even when occurring as early as 5 weeks of gestation with complete sonographic resolution by 8 weeks—can serve as an early sentinel marker for latent placental insufficiency and severe late-onset FGR. Although the sonographic disappearance of a demised twin often suggests a benign clinical course, the underlying placental bed may retain areas of focal ischemia, villous infarction, and fibrotic tissue that permanently limit the functional surface area required for exponential third-trimester fetal growth.

Rather than viewing post-VTS pregnancies as routine singletons following early sac reabsorption, clinicians are encouraged to maintain a heightened index of suspicion throughout the second half of gestation. Adopting a proactive surveillance framework—including serial sonographic growth evaluations every 3 to 4 weeks during the third trimester alongside umbilical and MCA Doppler monitoring—allows for timely detection of hemodynamic divergence. Ultimately, recognizing the long-term placental implications of early multifetal reduction ensures optimal multidisciplinary planning, timely delivery decisions, and favorable neonatal outcomes.

Statements and Declarations

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Competing Interests

The author declares no conflicts or competing interests.

Ethics Approval

Ethical approval for a single retrospective case report is exempt according to local institutional policy, provided patient anonymity is fully maintained.

Consent to Participate

Obtained directly from the patient.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical image/data.

Availability of Data and Materials

All relevant data supporting the conclusions of this article are included within the manuscript.

Author Contributions

MME designed the study, managed the clinical case data, performed the literature review, and wrote the manuscript.

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