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Succenturiate Placental Lobe Abruption: A Placental Pathology Complicating a Dangerous Delivery

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ABSTRACT

The development of a placenta is a complex process that occurs without a clinically significant issue in most pregnancies. At times, however, the process develops in a way that isolates an island of placental tissue away from the main body, connected only by unprotected vasculature within the amniotic membranes. The vessels of this succenturiate lobe of the placenta are vulnerable both to compression or laceration, threatening the antepartum period with poor weight gain or the peripartum period with fetal distress, hemorrhage, or retained products of conception. A majority of the time, this pathology is undiagnosed until recognized innocuously following delivery of the placenta. A placental abruption is a premature separation of the placenta from the uterus that can result in painful bleeding and fetal distress. This increased distress of the mother or baby from continued blood loss usually necessitates delivery either vaginally, if stability is maintained, or by cesarean if it is not. The amount of distress correlates to where and how much of the placenta is affected. While succenturiate lobes of the placenta and placental abruptions are not routinely associated with each other, the abruption of only the succenturiate lobe of the placenta in this instance minimized the severity to the fetus by allowing the main body of the placenta to remain intact. As the bleeding coagulated at the lobe, maternal well-being was maintained allowing enough time to complete a vaginal delivery. This case report describes a rarely discussed positive benefit of an SLP in the presence of placental abruption and the decision-making that allowed the patient to have a safe and successful vaginal delivery.

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INTRODUCTION

The placenta is a round or oval disc, usually 22 centimeters (cm) in diameter and 2-2.5 cm thick, composed of both maternal and embryonic tissue when it develops correctly.[1] In the first five gestational weeks, it establishes circulation and mediates nutrition, gas, and waste exchange between the pregnant mother and her fetus.[1-3] Development of the placenta is highly coordinated; any variation can significantly impact maternal and fetal morbidity and mortality.[1,2] If a lobe develops embedded in the amnion apart from the main placental body, and both lobes are roughly equal-sized, it is described as a bilobed placenta.[3,4] If lobes are not roughly equal-sized, it is described as a succenturiate lobe (Figure 1).[3-8]

A succenturiate lobe of the placenta (SLP) occurs in 0.16% to 5% of pregnancies.[1,4,7,9] Antepartum diagnosis only occurs 13% of the time, primarily by ultrasound with color doppler.[4-7,9] More often, it is first identified at the time of delivery when the intervening cord is either compressed (causing decelerations in fetal heart rate pattern) or severed (leading to fetal exsanguination or placental retention).[4-6,8] The vast majority of literature on SLP is either epidemiologic or revolves around its antenatal ultrasonographic diagnosis. In the case presented here, in which the SLP was diagnosed after delivery, a

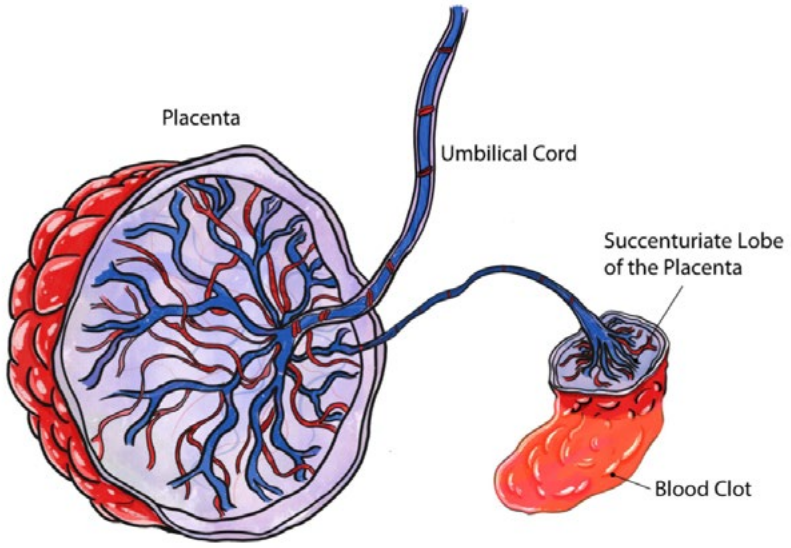

FIGURE 1: Representation of a succenturiate lobe of the placenta with a large clot attached.

patient with a previously undetected SLP abrupted only her succenturiate lobe. By understanding the diagnostic modalities and management strategies associated with SLP and placental abruption, conclusions can be drawn to reduce maternal and fetal morbidity and mortality. The following case examines the decision-making that allowed this patient to have a safe and successful vaginal delivery through the benefit of an SLP.

CASE PRESENTATION

A 21-year-old Gravida 3, Para 2002 at 34 5/7th weeks gestation presented to labor and delivery triage by ambulance for vaginal bleeding. When asked to quantify the patient's bleeding, the admitting nurse replied, "When she stood up, it ran out of her like a faucet." The initial vital signs for the patient were within normal limits and stable. The fetal heart rate monitor showed a category I tracing with a baseline of 140 beats/min. Contractions on the external tocometer were every three to four minutes and were associated with pain. A bedside ultrasound showed a cephalic presentation with a posterior placenta. There was a moderate amount of bright red blood noted in the vagina, and the patient's cervix was 4 centimeters dilated and 75% effaced, and the presenting part was at 0 station. A placental abruption was the initial working diagnosis.

The patient was admitted, and two large-bore intravenous (IV) accesses were placed through which lactated ringers were infused. A complete blood count was drawn, and two units of packed red blood cells (pRBCs) were typed and cross-matched. Her hemoglobin on presentation returned at 10.4 mg/dL. Coagulation studies were normal. Antibiotics were started for the infant's preterm status. Despite suspecting an abruption, continuous monitoring and vaginal delivery were initially planned because the patient had quickly delivered her first two children and both mother and fetus were stable. The patient was prepped for cesarean delivery prophylactically, as emergent delivery would be necessitated by a change in the status of either the mother or fetus. Two units were transfused in hopes of mitigating the continued blood loss burden on her admission anemia.

As the patient's cervix changed, bleeding decreased but always persisted at a level greater than expected for normal bloody show. Amniotomy was successful at 8 cm and 30 minutes later, she delivered a 4-pound, 5-ounce female with APGAR scores of 8 and 9 at one and five minutes of life, respectively. Because of her prematurity, the infant was taken to the neonatal intensive care unit for monitoring where she stayed for 10 days. The third stage of labor was short, as the placenta was easily delivered within seven minutes with gentle cord traction. Upon inspection of the placenta, a 5-6 cm succenturiate placental lobe was noted. An approximately 300 cc clot was connected to the lobe, evidencing the source of the abruption. Pathologic examination of the main body of the placenta, unlike the SLP, did not have any evidence of abruption. Follow-up hemoglobin was 8.9 mg/dL.

DISCUSSION

Normal and Abnormal Placental Development

The first step in understanding how placental abnormalities occur is knowledge of normal

placental development. This highly-coordinated process is initiated during blastocyst implantation when trophoblasts invade the endometrium and differentiate into an inner cytotrophoblastic (CTB) layer and an outer multinucleated syncytiotrophoblastic (SCTB) mass.[2,3] The SCTB layer invades the endometrial connective tissue, allowing the blastocyst to sink into the endometrium.[2] Within 13 to 14 days, the CTBs evaginate the SCTB layer, forming chorionic villi.[2,3] These villi extend into blood sinuses and remodel uterine spiral artery walls, creating maternal-fetal circulation within the first five weeks of gestation.[2,3] By eight weeks, interstitial invasion promotes circumferential placental expansion and recruits maternal arterioles. Villi become multinucleated and form the placental bed, or decidua basalis.[2,3] A decidual reaction occurs to restrain the invasive blastocyst.[2] Villi then atrophy on the decidua capsularis, leaving a smooth surface called the chorionic laeve and creating a definite placental disc.[2,3] Low oxygen tension in the first trimester from endovascular CTB debris in the uterine arteries is thought to be an important regulator of CTB function. By 10 to 12 weeks, maternal spiral arteries bring in more oxygen-rich blood.[2] By three months, placental cotyledons are formed by partitioning of the villi and infolding of the decidua basalis.[3] An SLP results from the persistence of proliferating villi on the decidua capsularis due to localized failure of normal non-villous atrophy, forming one or more accessory lobes attached to the main placenta by vasculature.[3,6,8] This is suspected to occur from either an abnormality during implantation of the blastocyst, poor nutrition, or abnormalities in oxygen tension during the attachment of the blastocyst.[8,10] However, one-third of SLPs are associated with damage to an intervening segment of the placenta (from an infarct or inadequate nutrition) early in the embryologic process.[8,9]

From a macroscopic sense, an SLP develops when one or more cotyledons develop apart from the main placental body.[4,6,8] Risk factors of placental implantation on previous scars or leiomyomas,1 advanced maternal age, and in vitro fertilization lead to pathogenic causative theories of underlying vascular endothelial damage.[1,4,6,8,9] Usually, the placental tissue is connected by blood vessels running through the amniotic membranes unprotected by Wharton's jelly. This lack of vessel protection and stability is similar to that seen between the umbilical cord and the placenta in a velamentous cord insertion.[11] These "unprotected" vessels are susceptible to compression, leading to poor fetal growth, low infant birth weight antenatally, and fetal distress during labor.[8,9,11] More acutely dangerous is the risk of laceration, not only from amniotomy causing rapid fetal bleeding but also during the third stage of labor, causing an SLP to be retained.[5,7,8] These exposed vessels, if proximate to the internal cervical os, are called vasa previa. This can occur in one in 2,500 pregnancies and carries nearly a 10% mortality rate, necessitating a cesarean delivery.[4,6,9,12] An antenatal ultrasound can detect this anomaly and rule out an amniotic band, as it appears as sheets on a gray scale,[1,4,5,6,7] but a majority of SLPs unable to be identified antenatally are diagnosed at delivery. Therefore, it must always be considered in cases of bleeding during pregnancy.

Placental Abruption

High in the differential of late second and third trimester bleeding is the placental abruption, which complicates 1% of all births and 10% of preterm births.[1,13] It remains a leading cause of perinatal morbidity and mortality[10,14] and occurs when the maternal portion of the decidua-basalis prematurely separates from the endometrium, causing vascular compromise and hemorrhage.[9,10,13] As blood accumulates, it forces a further separation.10 Clinical presentation is dependent upon location (central or marginal) and severity (partial or complete), but bleeding is not always a reliable indicator of severity, as the abruption may be concealed.[10,13] There are more than 50 different risk factors for abruption,[10,14] but causes can be primarily grouped into vascular and mechanical etiologies (Table 1).[13] When there is vaginal bleeding with a firm, tender uterus, one must work under a presumptive diagnosis of abruption until a definitive diagnosis can be rendered after the third stage of labor.[10] Management requires a combination of continuous monitoring and supportive care by giving oxygen, IV fluids, and sometimes blood, but ultimately results in delivery.[13,14]

Management Decisions

This patient's management revolved around

Vascular Issues	Mechanical (stretching/sheering) Issues
Poor Placentation	*Sudden Uterine Decompression*
Leiomyoma	Multiple Gestation
Placental Previa	Polyhydramnios
Previous Cesarean Delivery	*Trauma*
Coagulopathy	Direct Blow to the Abdomen
Factor V Leiden Deficiency	External Cephalic Version
Hyperhomocysteinemia	Fall
MTHFR, homozygous	Motor Vehicle Accident
Prothrombin Mutation	Short Umbilical Cord
Vascular Damage	
Smoking	
Hypertension (current or history)	
Diabetes	
Abruption History	
Pre-Eclampsia	
Maternal Age > 35yo	
Cocaine - vasospasm	

TABLE 1: Causes of Abruption[10,14]. Causes of abruption divided into pathologic causes. (MTHFR = Methyltetrahydrofolate Reductase)

Class	0	1	2	3
Description	Asymptomatic	Mild	Moderate	Severe
Bleeding	None	None or Small	None or Moderate	None or Heavy
Uterus	Non-Tender	Slight Tenderness	Significant Tenderness	Tetanic Uterus
Maternal	Normal BP and HR	Normal BP and HR	Changes to BP and HR	Shock
Fetal	No Fetal Distress	No Fetal Distress	Fetal Distress	Fetal Death
Other	Found After Delivery		Clotting Profile Changed	Coagulopathy

TABLE 2: Abruption Classification[10, 14], Characterizes abruption by a rating system. Highlighted regions demonstrate this case's findings that span classes 1 and 2, demonstrating the clinical findings of an abrupted succenturiate lobe of the placenta.

the assumption that abruption was the sole pathologic process, as it was unknown that she had an SLP. Management decisions balanced relative maternal and fetal stability, cervical change, and gestational age.[10,13,14] If both mother and infant are stable, continued pregnancy for antibiotics and lung maturity is warranted, but labor could force a movement toward delivery at any moment. Vaginal delivery can be considered if delivery is not prolonged and maternal and fetal stability is maintained, but hemorrhage constantly threatens to disrupt that stability.[10,13] Assessment of blood volume status also proves problematic, as it is often underestimated.[14] The final ramifications of acute blood loss are not reflected in the initial hemoglobin and hematocrit, as maternal circulation must first come to homeostasis following the initial and ongoing losses of volume.[13] The expected decrease in blood volume may necessitate a transfusion at a higher hemoglobin level than usual to blunt this decrease and maintain a reassuring maternal blood pressure and fetal heart rate. While retained lobes and vessel laceration are well-documented outcomes of an SLP, the literature only offers a connection between an SLP and third-trimester bleeding; it does not describe the effect.[1,7,9] In our patient's case, the fact that only the SLP abrupted kept the consequences for the mother and infant from being any more severe. Table 2[10,14] demonstrates how an abruption of only the SLP defied clear classification.

The main portion of the placenta was sequestered from the sheering forces of the abrupting lobe and allowed for the continued exchange of nutrients and wastes. The bleeding from the SLP clotted, and labor continued as expected, leading to a vaginal delivery.

CONCLUSION

An SLP is a vastly under-reported condition affecting pregnancies; however, it is an afterthought to delivery much of the time. In some circumstances, its unique characteristics can have a dramatic impact on the delivery outcome. An abruption of only the SLP likely mitigated both maternal and fetal consequences of bleeding in our patient. In conjunction with conservative rules for abruption management, it allowed for a safe vaginal delivery of a preterm infant. If the mother and fetus are stable, third-trimester bleeding should be managed conservatively to allow for the potential of vaginal delivery, as a concomitant SLP may exist.

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