SUBCHORIONIC HEMATOMA IN EARLY GESTATION, THE RISK OF FETAL GROWTH RESTRICTION AND ADVERSE PERINATAL OUTCOMES

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Abstract. *Early gestational bleeding is a common obstetric complication and is increasingly recognized as a clinical marker of impaired implantation and placental dysfunction. One of the most frequent ultrasound findings in this context is sub chorionic hematoma (SCH), which has been associated with a spectrum of adverse perinatal outcomes. Accumulating evidence suggests that SCH reflects abnormal placentation processes that may persist beyond the first trimester and contribute to fetal growth restriction, preterm delivery, placental abruption, and other complications. This review synthesizes current evidence on the epidemiology, pathophysiology, prognostic significance, and clinical implications of SCH and early pregnancy bleeding, with particular emphasis on fetal growth restriction and perinatal outcomes.*

Keywords: *sub chorionic hematoma, early pregnancy bleeding, placental dysfunction, fetal growth restriction, perinatal outcomes.*

Introduction

Bleeding during early pregnancy remains one of the most frequent reasons for obstetric consultation and occurs in approximately 15–25% of all gestations [6,9]. Although many pregnancies with early bleeding progress to term, increasing evidence indicates that such bleeding is not always a benign event and may reflect abnormal implantation or early placental instability [7,10]. Sub chorionic hematoma is the most common sonographic finding associated with first-trimester bleeding [2,3,8]. While often detected incidentally, SCH has attracted growing attention as a potential marker of placental dysfunction [1,7]. The clinical significance of SCH remains controversial, with studies reporting heterogeneous associations with miscarriage, preterm birth, fetal growth restriction (FGR), and placental abruption [1,9-11]. Sub chorionic hematoma is defined as an accumulation of blood between the chorionic membrane and the uterine wall resulting from partial detachment of trophoblastic tissue [2,5]. Reported prevalence varies widely, ranging from <1% to >20%, depending on diagnostic criteria, gestational age at detection, population characteristics, and ultrasound resolution [1-4].

According to World Health Organization data, the prevalence of growth-restricted newborns varies considerably by region, ranging from over 30% in parts of Central Asia to less than 7% in developed European countries. In the United States, fetal growth restriction is diagnosed in 10–15% of births, and severe intrapartum hypoxia is observed in approximately one-third of affected infants. In the Russian Federation, reported rates range from 2.4% to 17% [15]. Infants with fetal growth restriction account for nearly one-third of all low birth weight newborns. Among term infants, growth restriction occurs in approximately 67 per 1,000 live births, increasing to nearly 180 per 1,000 among preterm neonates. In many cases, reduced fetal size is accompanied by delayed development of individual organs, particularly parenchymal structures.

In asymptomatic women, SCH is detected in approximately 1.7–3.1% of pregnancies, whereas among women presenting with threatened miscarriage, prevalence may reach 20% [1,3,8]. Sonographically, SCH typically appears as a crescent-shaped hypoechoic or anechoic area adjacent to the gestational sac in the first trimester [4,8].

The mechanisms behind SCH formation are still not fully understood. Current hypotheses propose mechanical or vascular disruption at the chorionic–decidual interface, resulting in low-pressure bleeding from marginal placental vessels [2,27]. Growing evidence supports viewing SCH as an early sign of impaired placentation [7,10]. Normal placental development requires adequate trophoblastic invasion and remodelling of spiral arteries [7]. Defective placentation has been identified in the majority of early pregnancy failures and is strongly associated with later complications such as hypertensive disorders, placental abruption, spontaneous preterm birth, and fetal growth restriction [7,11,17]. Premature exposure of placental tissues to maternal circulation may induce excessive oxidative stress before sufficient antioxidant defences are established, contributing to placental injury and pregnancy loss [20].

Numerous studies report associations between SCH and adverse pregnancy outcomes, including spontaneous miscarriage, stillbirth, placental abruption, and preterm delivery [1,3,8-11]. Meta-analyses confirm a consistent association between SCH and pregnancy loss, while links with preterm birth and FGR are less consistent [1,9,10]. Hematoma size has been proposed as an important prognostic factor. Several investigations demonstrate higher risks of miscarriage and placental complications with increasing hematoma volume, particularly when SCH is diagnosed early in gestation [8,18]. However, other studies report weaker associations after adjustment for confounding variables [6,10].

The potential impact of SCH on pregnancy prognosis appears to be influenced by multiple factors, including hematoma size, gestational age at diagnosis, and placental function. Adverse outcomes such as placental abruption, preterm delivery, and early pregnancy loss have been reported in association with SCH across multiple studies [7,10–16]. Some investigators have emphasized the importance of hematoma volume, suggesting that specific measurement methods demonstrate stronger correlations with first-trimester outcomes than others [17]. Both the presence and size of SCH have been linked to an increased likelihood of miscarriage, intrauterine growth restriction, recurrent vaginal bleeding, placental abruption, preterm delivery, and fetal growth impairment [1,6–8,14,18–20]. A large meta-analysis including over 1,700 women with SCH and more than 70,000 controls identified a significantly higher risk of spontaneous abortion, stillbirth, placental abruption, preterm birth, and preterm premature rupture of membranes in pregnancies complicated by SCH, although no association was observed with small-for-gestational-age infants or preeclampsia [10].

The location of the hematoma does not appear to significantly influence overall pregnancy outcome, although posteriorly located SCH has been more strongly associated with fetal distress [1,29]. Use of anticoagulant or antiplatelet therapies, including low-dose ASA, heparin, and thrombolytic agents, has been associated with an increased risk of SCH formation in early pregnancy, while progesterone therapy has not consistently demonstrated protective effects against early pregnancy loss [38–40]. Importantly, vaginal bleeding itself—independent of SCH—has been identified as an independent predictor of adverse maternal and neonatal outcomes, including shortened gestational duration, low Apgar scores, and increased neonatal intensive care unit admission [6,9,19].

Fetal growth restriction represents one of the most clinically significant consequences of placental insufficiency [12,13]. Impaired uteroplacental blood flow leads to chronic fetal hypoxia and metabolic disturbances, resulting in reduced fetal growth potential [12]. Epidemiological data demonstrate substantial regional variation in FGR prevalence, ranging from >30% in parts of Central Asia to <7% in developed European countries [15]. In pregnancies complicated by early bleeding and SCH, the risk of delivering growth-restricted or low birth weight infants is consistently elevated [9-11,18]. Low-dose acetylsalicylic acid (ASA) improves uteroplacental perfusion through inhibition of platelet aggregation and has demonstrated efficacy in reducing the risk of preeclampsia and preterm birth in high-risk populations [21,23-25]. Its role in pregnancies complicated by SCH, however, remains controversial.

Although low-dose ASA is routinely recommended for women at high risk of preeclampsia [24], emerging evidence suggests that its use during the first trimester may increase the likelihood of SCH formation. Based on these observations, some authors recommend postponing the initiation of prophylactic ASA therapy until the second trimester in women with a history of preeclampsia. Current guidelines from the U.S. Preventive Services Task Force advise low-dose ASA (81 mg) for women with specific risk factors, including prior preeclampsia, multifetal gestation, chronic hypertension, diabetes, renal disease, and autoimmune disorders [31]. While cost-effectiveness analyses support widespread ASA prophylaxis, these models do not fully account for the potential increase in SCH-related morbidity [32].

Enhanced endometrial and subendometrial vascularity has been observed in women with successful pregnancies compared to those who miscarry, providing a physiological rationale for ASA use in selected populations [33]. Nevertheless, routine administration of ASA in assisted reproduction cycles is not recommended due to insufficient evidence of benefit and a potential risk of harm [22,34–36]. Maternal age, gestational age at diagnosis, and hematoma size have all been identified as independent factors influencing SCH prevalence [36]. Some studies suggest that first-trimester ASA use may increase the likelihood of SCH formation [22,24], prompting debate regarding optimal timing of prophylaxis. Current guidelines recommend ASA for women at high risk of preeclampsia, although SCH-specific recommendations are lacking [24]. Given the heterogeneity of published data, risk assessment in pregnancies complicated by SCH should be individualised [1,10]. Serial ultrasound monitoring allows evaluation of hematoma evolution and early detection of placental complications [8,18]. Although no specific preventive therapy for SCH exists, enhanced surveillance may facilitate the timely management of emerging obstetric risks.

Conclusion

Subchorionic hematoma and early pregnancy bleeding are associated with a modest but reproducible increase in the risk of adverse perinatal outcomes, including pregnancy loss, preterm delivery, placental abruption, and fetal growth restriction. The strongest and most consistent association is observed with placental abruption, supporting the concept of SCH as a marker of abnormal placentation. Integration of sonographic findings, biochemical markers, and clinical risk factors is essential for individualised counselling and management. Further prospective studies are needed to refine prognostic criteria and optimise preventive strategies in early gestation. Overall, while the association between SCH and adverse perinatal outcomes appears modest for some endpoints, such as pregnancy loss and preterm delivery, a stronger relationship has been consistently observed with placental abruption. Given the heterogeneity of available data, risk assessment should be individualized, taking into account hematoma characteristics and additional

maternal risk factors. Future clinical research comparing different approaches to evaluating hematoma size and location may further improve prognostic accuracy and perinatal care.

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