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CHRONIC VENOUS DISEASE IN PREGNANCY: PATHOPHYSIOLOGICAL MECHANISMS, MATERNAL COMPLICATIONS AND IMPLICATIONS FOR FETAL OUTCOMES

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Relevance. Pregnancy is a significant risk factor for the development and exacerbation of chronic venous disease (CVD). The physiological adaptations of gestation including hormonal venodilation, increased blood volume, and mechanical compression by the gravid uterus create a unique hemodynamic environment that precipitates venous hypertension. While often dismissed as a benign discomfort, CVD in pregnancy carries significant pathogenic potential for both maternal and fetal health.

Aim: this review aims to elucidate the pathophysiological mechanisms of chronic venous disease during pregnancy, analyze its maternal complications (thrombotic, dermatological, and pelvic), and evaluate the emerging evidence regarding its impact on placental function and fetal outcomes.

Materials and methods. A comprehensive literature review was conducted using PubMed, Scopus, and Cochrane Library databases (2000–2024). Studies investigating venous hemodynamics in pregnancy, the incidence of venous thromboembolism (VTE), pelvic congestion syndrome, and the placental implications of maternal venous hypertension were analyzed. Priority was given to clinical trials, systematic reviews, and pathophysiological studies.

Results and their discussion. The pathogenesis of CVD in pregnancy is driven by a triad of factors: (1) Hormonal venodilation (progesterone/relaxin-mediated), leading to valvular incompetence and venous reflux; (2) Mechanical obstruction by the gravid uterus, causing inferior vena cava (IVC) compression and elevated ambulatory venous pressure; and (3) A hypercoagulable state, increasing thrombotic risk. These mechanisms culminate in several key complications:

Venous Thromboembolism (VTE): Pregnancy increases VTE risk 4- to 5-fold, with left-sided iliofemoral DVT predominating due to May-Thurner anatomy exacerbated by uterine compression.

Pulmonary embolism remains a leading cause of maternal mortality.

Pelvic Congestion Syndrome (PCS): Ovarian vein incompetence leads to pelvic varicosities, resulting in chronic pelvic pain, dyspareunia, and vulvar varicosities that impair mobility and quality of life.

Placental Implications: Emerging evidence suggests that severe maternal pelvic venous hypertension may increase uterine vein pressure, potentially reducing the utero-placental perfusion gradient. This has been associated with an increased risk of intrauterine growth restriction (IUGR) and oligohydramnios, although causal relationships require further investigation.

Postpartum Legacy: Venous damage sustained during pregnancy often leads to irreversible valvular destruction, resulting in chronic venous insufficiency (CVI) and recurrent varicosities in subsequent pregnancies and later life.

Conclusions. Chronic venous disease in pregnancy is a significant, multi-system pathological process extending far beyond cosmetic concerns. It encompasses thrombotic emergencies, debilitating pelvic pain, and potential placental dysfunction. Early identification of at-risk women, compression therapy, and postpartum surveillance are critical to mitigating complications. Future research should focus on the hemodynamic impact of venous congestion on placental perfusion and the long-term cardiovascular health of affected women.