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FETAL VASCULAR MALPERFUSION AND PLACENTAL THROMBOSIS: MORPHOLOGICAL EVIDENCE AND NEONATAL IMPLICATIONS

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ABSTRACT

Fetal vascular malperfusion (FVM) is a placental injury pattern characterized by impaired fetal-side blood flow, often accompanied by thrombosis and downstream villous ischemic changes. This literature-based thesis summarizes the principal histomorphological criteria, potential pathogenetic mechanisms, and neonatal implications of FVM. Evidence from the Amsterdam consensus, placental pathology reviews, and a published systematic review and meta-analysis was narratively synthesized. Diagnostic lesions include thrombi in chorionic plate or stem vessels, avascular villi, villous stromal–vascular karyorrhexis, and vascular obliteration. In a meta-analysis, FVM was associated with higher odds of neonatal central nervous system injury in term or near-term infants (odds ratio [OR] 4.00; 95% confidence interval [CI] 2.72–5.90). The findings support standardized histopathological reporting and individualized neonatal neurological surveillance, while not establishing causation in every case.

Keywords: fetal vascular malperfusion; placental thrombosis; avascular villi; chorionic plate vessels; neonatal encephalopathy; perinatal stroke; placental histopathology.

INTRODUCTION

The placenta integrates maternal and fetal circulations, permitting gas and nutrient exchange without direct mixing of their blood. Interruption of the fetal vascular tree can reduce tissue perfusion and produce characteristic histological lesions. The Amsterdam Placental Workshop Group recommended the term fetal vascular malperfusion rather than the older expression fetal thrombotic vasculopathy, thereby emphasizing impaired perfusion as the broader pathophysiological process [1]. FVM must be distinguished from maternal vascular malperfusion, which is centered on abnormalities in maternal uteroplacental perfusion [1, 3].

The potential clinical significance extends beyond histopathology. FVM has been described in association with fetal growth restriction, fetal loss, neonatal encephalopathy, perinatal stroke, and later neurodevelopmental challenges [2, 4, 5]. However, lesion severity, gestational age, coincident inflammation, and clinical context influence interpretation. A placental diagnosis is a marker for risk assessment rather than a stand-alone explanation of an individual newborn's condition.

MAIN BODY

Aim and methods. This narrative review examines morphological findings of FVM and considers their relevance to neonatal outcomes. Seven peer-reviewed consensus, review, and meta-analytic sources were selected to cover standardized lesion definitions, mechanisms, differential diagnosis, and neonatal

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associations [1–7]. No original placental specimens or patient-level datasets were analyzed; the numerical evidence shown below is taken from a published meta-analysis rather than a new clinical cohort.

Morphological basis. Umbilical-cord compression, excessive coiling, abnormal cord insertion, fetal circulatory dysfunction, and conditions promoting stasis or hypercoagulability can impair fetal perfusion [2, 4]. A segmental complete pattern may exhibit thrombosis in fetal vessels and localized downstream avascular villi; a global partial pattern may show more diffuse effects of intermittently reduced fetal flow [2]. Pathologists should describe thrombus location, size, distribution, and associated stromal–vascular lesions, while considering whether tissue autolysis or postmortem changes mimic ante-mortem ischemia [1, 4].

Table 1. Major morphological indicators of fetal vascular malperfusion

Lesion	Histological interpretation	Reporting significance
Fetal vascular thrombus	Intraluminal fibrin/erythrocyte clot in fetal vessel	Specify location; differentiate from congestion
Avascular villi	Groups of villi lacking patent fetal capillaries	Assess number, extent, and distribution
Stromal–vascular karyorrhexis	Fragmentation of fetal vascular/stromal nuclei	Suggests evolving fetal-side ischemic injury
Stem-vessel obliteration	Narrowed or fibrotically occluded fetal vessel	Indicates chronic impairment of fetal flow
Intramural fibrin deposition	Fibrin within the fetal vessel wall	Supports vessel injury and timing assessment

The combination of thrombotic lesions and their downstream villous effects helps differentiate localized obstruction from more generalized perfusion disturbances. Isolated microscopic changes may be nonspecific; therefore, clinically meaningful interpretation requires lesion burden, sampling adequacy, associated cord pathology, and neonatal history [1–3]. The Amsterdam framework also distinguishes high-grade FVM from limited focal findings by explicit quantitative and topographic criteria [1].

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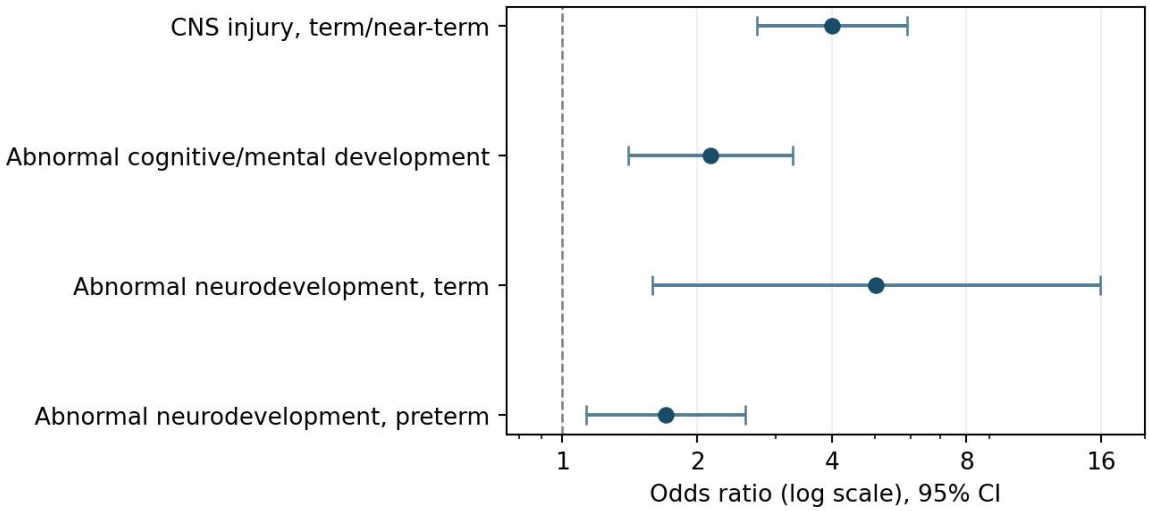


Figure 1. Associations reported in Spinillo et al. [5]. These are published meta-analytic estimates, not newly collected patient data.

Neonatal implications. A meta-analysis by Spinillo and colleagues reported a fourfold association between FVM and central nervous system injury (neonatal encephalopathy or perinatal stroke) in term or near-term infants (OR 4.00; 95% CI 2.72–5.90). Increased odds of abnormal neurodevelopment were observed in both term and preterm populations, although effect sizes differed [5]. These observational associations cannot prove that placental thrombosis directly caused the neurological event; comorbid placental lesions, obstetric events, gestational age, and selection effects must be considered.

In premature infants, the same meta-analysis did not demonstrate a statistically clear association of FVM with intracranial hemorrhage or periventricular leukomalacia (OR 1.40; 95% CI 0.90–2.18) [5]. This finding is important because it discourages undifferentiated claims that all fetal vascular lesions predict every form of neonatal brain injury. Clinical follow-up should be driven by the complete neonatal examination and overall risk profile.

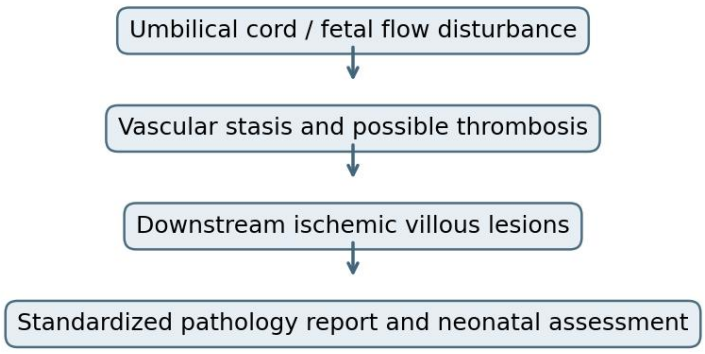


Figure 2. Conceptual pathway connecting placental pathology with clinical review (illustrative; not a causal estimate).

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Clinical interpretation and practice. The placental examination should encompass the umbilical cord, chorionic plate vessels, and sufficiently sampled parenchyma. A standardized report should record lesion distribution and grade, maternal and fetal inflammatory findings, and other competing pathological patterns [1, 3, 6]. When moderate or high-grade FVM accompanies neonatal seizures, encephalopathy, suspected stroke, or unexplained neurological symptoms, multidisciplinary discussion among pathologists, neonatologists, and pediatric neurologists is appropriate [5–7]. Routine neuroimaging or thrombophilia testing should not be inferred from the placenta alone; their indications are clinical.

CONCLUSION

Fetal vascular malperfusion represents a recognizable spectrum of fetal-side placental circulatory injury, including thrombosis, avascular villi, and stromal–vascular karyorrhexis. Morphology may help reconstruct a plausible pathway of compromised fetal perfusion, although timing and causation remain uncertain. Published data associate FVM with neonatal encephalopathy, perinatal stroke, and subsequent neurodevelopmental impairment, particularly in term infants [5]. Standardized Amsterdam-based reporting, careful differentiation of coexisting placental lesions, and targeted neonatal follow-up may enhance clinicopathological communication. Future prospective studies with placental grading and long-term infant outcomes are needed.

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