Opinion Paper

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Placenta-oriented counseling: challenges and opportunities in obstetric practice

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Abstract: Placental histopathology is an invaluable yet underutilized resource in obstetric care. While it offers unique insights into the etiology of adverse pregnancy outcomes, its clinical integration is often limited by inconsistencies in placenta submission, reporting delays, lack of standardized interpretation, and limited interdisciplinary dialogue. Certain placental abnormalities provide immediate clinical insights, guiding acute management decisions, while others indicate an increased risk of recurrence in future pregnancies. Additionally, many lesions help contextualize antenatal findings or clarify postnatal complications, offering valuable diagnostic and prognostic information. The article reviews the spectrum of placental lesions, ranging from common findings to rare high-risk entities, and their implications for future pregnancy outcomes. It examines both the persistent challenges and emerging opportunities associated with placenta-oriented counseling in modern obstetric practice. It also explores strategies for translating placental findings into actionable care, emphasizing the importance of implementing structured, multidisciplinary review processes. Furthermore, it highlights how placental histologic findings must be interpreted within the broader clinical context, considering maternal history, laboratory investigations, microbiological cultures, genetic analyses, and neonatal outcomes, to ensure their diagnostic relevance and support informed patient-centered decision-making focused on the maternal–placental–fetal triad.

Keywords: placenta; histology; Amsterdam consensus; stillbirth; preeclampsia; fetal growth restriction

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Introduction

The placenta is a transient fetomaternal organ that performs vital functions in pregnancy, including oxygen/carbon dioxide exchange, nutrient delivery, waste removal, and hormone production, thereby sustaining fetal growth and development. It also serves as an immunological interface between mother and fetus. Given its central role, the placenta's condition often reflects the health of the pregnancy [1–3].

Systematic placental examination plays a pivotal role in modern obstetric care by providing diagnostic and prognostic insights into adverse pregnancy outcomes and neonatal complications. Placental pathology findings frequently offer explanations for such outcomes and, in many cases, deliver valuable information that informs both immediate clinical management and future pregnancy planning. Placental pathology also helps distinguish between acute and chronic intrauterine events, identify recurrent risk factors, guide therapeutic decisions, and establish the timeline of intrauterine insults, which is especially relevant in medicolegal contexts [4, 5].

The clinical utility of placental pathology lies in its targeted application, guided by established criteria. While specific indications may vary across institutions, they generally include fetal growth restriction (FGR), maternal infections, preeclampsia, preterm birth, and other obstetric complications with potential implications for maternal and neonatal outcomes [2, 6, 7]. Its value is particularly evident in the evaluation of stillbirth, where it serves as a key diagnostic tool [8], with Gibbins et al. [9] reporting that placental pathology yielded clinically useful findings in approximately 65 % of cases. Furthermore, a large prospective study by Korteweg et al. [10] found abnormal placental findings in 89.2 % of fetal death cases, identifying it as the single most informative diagnostic modality for determining the cause of stillbirth.

Histopathological evaluation of the placenta provides critical insights into maternal health beyond pregnancy, as complications such as preeclampsia, gestational diabetes, and placental abruption are strongly associated with increased risks of cardiovascular disease, chronic kidney

disease, and metabolic disorders later in life [11]. Identifying placental abnormalities allows for early risk stratification and targeted interventions, potentially mitigating long-term health consequences for affected women [11, 12].

In recent years, there has been a growing emphasis on enhancing the clinical utility of placental pathology reports, with multiple publications advocating for a more standardized approach to identifying placental findings [1, 2, 13, 14]. However, despite its proven value, placental pathology remains underutilized in routine obstetric practice due to a range of challenges encountered at various levels.

Emerging frameworks in fetal–neonatal neurology emphasize the maternal–placental–fetal (MPF) triad as a central unit of analysis in understanding pregnancy outcomes. This triad reflects the dynamic interplay between maternal health, placental function, and fetal neurological development, offering a more integrated lens for interpreting placental pathology. Incorporating the MPF triad into clinical reasoning helps contextualize placental findings within broader disease pathways that may shape neonatal and long-term brain health [15].

This article explores how placental examination findings can be translated into meaningful counseling for women and their partners in obstetric care. It reviews the spectrum of placental lesions, ranging from common findings to rare high-risk entities, and their implications for future pregnancy outcomes within the context of the MPF triad. Persistent challenges in integrating placental pathology into routine obstetric care, including inconsistent submission practices, limited access to specialized pathology expertise, reporting delays, lack of standardized interpretation, limited interobserver reliability, and communication barriers, will be examined. In parallel, the manuscript will highlight opportunities to enhance postpartum counseling through standardized histologic evaluation, foster interdisciplinary collaboration between clinicians and pathologists, and recognition of women and their partners as active participants in care, laying the groundwork for future evidence-based perinatal research.

Placental pathology: major categories associated with adverse outcomes

The Amsterdam placental workshop group consensus statement (2016) established standardized definitions and sampling guidelines for a broad spectrum of placental lesions, aiming to harmonize reporting practices and enhance clinical interpretability across institutions [16]. It provides a foundational framework for consistent terminology and diagnostic criteria. Building on this foundation, Redline et al. [17] introduced a stepwise diagnostic approach that integrates clinical history, gross examination, and histologic assessment to improve accuracy and reproducibility in placental pathology evaluation.

This approach has helped categorize placental lesions by underlying pathophysiological mechanisms, thereby enhancing their clinical relevance in obstetric care and improving their utility for placenta-oriented counseling (Table 1).

Acute inflammatory lesions typically indicate infection ascending from the genital tract, manifesting as chorioamnionitis. Acute histologic chorioamnionitis is defined by neutrophilic infiltration within the chorioamniotic membranes, indicative of a maternal inflammatory response. The presence of funisitis and chorionic vasculitis denotes fetal involvement, as neutrophils migrate into the umbilical cord and chorionic vessels. The predominant microorganisms associated with intraamniotic infection include *Ureaplasma* species, *Gardnerella vaginalis*, *Mycoplasma hominis*, and *Streptococcus agalactiae.* Polymicrobial infections occur in approximately 70 % of cases, with anaerobes such as *Fusobacterium* and *Bacteroides* species frequently identified. Acute chorioamnionitis (ACA) is common in preterm birth, prolonged rupture of membranes, prolonged labour and after digital examinations before and during labour [18]. In a recent narrative review and reevaluation of placental lesions based on the Amsterdam criteria, Yaguchi et al. [19] reported that acute inflammatory lesions, across most studies, did not show consistent associations with long-term neurodevelopmental outcomes in infants. These findings suggest that while acute inflammation may contribute to early neonatal morbidity, its impact on later developmental trajectories may be limited compared to chronic placental lesions such as maternal vascular malperfusion or fetal vascular malperfusion. The prevalence of chorioamnionitis is a function of gestational age at birth, and present in 3–5% of term placentas and in 94 % of placentas delivered at 21–24 weeks of gestation [20]. A prior diagnosis of clinical chorioamnionitis significantly increases the risk of recurrence in subsequent pregnancies, with odds estimated to be 2- to 3-fold higher compared to those without a prior history [21]. Recurrence risk is especially high when chorioamnionitis leads to delivery between 20 and 24 weeks, likely reflecting the increased rate of intraamniotic infection at early gestations [22]. Following the histopathological diagnosis of chorioamnionitis, extension of neonatal antibiotic therapy may be warranted. Evaluation and treatment of potential underlying maternal conditions, such as periodontal disease or chronic endometritis, should

Table 1: Placental lesions with counseling and management recommendations.

Placental lesion	Associated complications	Estimated prevalence	Recurrence risk	Counseling and management recommendations
Maternal vascular malperfusion	Hypertensive disorders, FGR, stillbirth, preterm birth	~35.7 % in term pregnancies; high-burden lesions in ~7.4 %	~10–25 %	Review maternal health (cardiovascular risk, glucose tolerance, renal function). Consider weight loss if indicated. In next pregnancy: low-dose aspirin, uterine artery Doppler, enhanced surveillance for preeclampsia/growth.
Fetal vascular malperfusion	Stillbirth, FGR, neurodevelopmental impairment	~19.7 % in term pregnancies (mild forms); high-grade lesions are much less common	Low; generally sporadic lesion (no significant recurrence in absence of underlying thrombophilia)	Discuss cause of outcome (e.g. cord issue, clot). If lesions extensive, evaluate maternal/fetal thrombophilia. Future pregnancy generally reassuring: standard fetal monitoring; consider enhanced surveillance if indicated.
Villitis of unknown etiology	FGR, stillbirth, neurodevelopmental impairment, preeclampsia	Low-grade: ~13.1 %; high-grade: ~1.2 %	~34–100 % (in high-grade VUE). Higher risk if prior late losses or maternal immune factors.	Explain that mild VUE is common, typically with good prognosis. If high-grade or recurrent, consider maternal immune evaluation. For future pregnancy: plan serial growth scans; consider low-dose aspirin and/or immunosuppressive therapy if indicated.
Chronic histiocytic intervillositis	FGR, recurrent miscarriage, stillbirth	~0.06 %	Very high (≈70–100 %), frequently recurs without intervention.	Discuss high recurrence risk. Refer to specialist. In future pregnancy: consider experimental treatments (e.g. steroids, hydroxychloroquine, aspirin); intensive monitoring advised; early delivery may be considered.
Massive perivillous fibrin deposition	FGR, oligohydramnios, preterm birth, stillbirth, fetal metabolic disorders and malformations, neurological impairment	~0.03 %	Very high (often >75 %; near 100 % in some series)	Counsel on high recurrence and risk of adverse outcomes. No proven therapy, but immunotherapy or anticoagulation (e.g. aspirin, heparin) may be considered. Early specialist input and close monitoring with serial growth scans in future pregnancy.
Acute inflammatory lesions (associated with preterm birth)	Maternal sepsis, spontaneous preterm delivery, neonatal sepsis	~3–5% at term; up to 94 % in extremely preterm births (21–24 weeks)	~10–25 %	Prolong neonatal antibiotics if indicated. Treat maternal sources (e.g. periodontal disease, chronic endometritis). In future pregnancy: early cervical length screening and surveillance for signs of infection.

FGR, fetal growth restriction; VUE, villitis of unknown etiology.

be considered. Although formal guidelines for the management of subsequent pregnancies after acute chorioamnionitis are lacking, certain measures may prove beneficial. These include close antenatal surveillance for early signs of infection, targeted microbiologic testing for *Ureaplasma* and *Mycoplasma* species to guide antibiotic selection, and early second-trimester cervical ultrasonography to assess for cervical insufficiency or other risk factors [3, 18].

Villitis of Unknown Etiology (VUE) is a chronic inflammatory condition of the placenta, characterized by maternal T-cell infiltration into the fetal villous stroma, leading to villous injury and placental dysfunction [1]. It is a common placental lesion, affecting 5–15 % of all third-trimester placentas [23]. Romero et al. [24] found that in normal term placentas, low-grade VUE was observed in 13.1 % of cases, while high-grade VUE was present in only 1.2 %. While the clinical significance of low-grade VUE remains debated, high-grade cases are strongly associated with adverse pregnancy outcomes, including FGR, stillbirth, and neurodevelopmental impairment [1, 23]. Additionally, obliterative fetal vasculopathy in VUE cases has been identified as a significant risk factor for cerebral palsy [23]. In contrast to this, a recent reevaluation by Yaguchi et al. [19], synthesizing findings across multiple studies, found no consistent

association between VUE and infantile neurodevelopmental outcomes. However, one cohort of newborns with hypoxic-ischemic encephalopathy grouped VUE with other chronic placental abnormalities and linked it to a negative base deficit within the first hour of life. These findings suggest that while VUE may not independently predict long-term outcomes, its presence in combination with other lesions could contribute to early neonatal compromise, highlighting ongoing uncertainty and the need for further investigation. High-grade VUE has a substantial recurrence risk, estimated between 34 and 100 %, warranting enhanced fetal surveillance in subsequent pregnancies [25, 26]. Additionally, Freedman et al. [26] reported that high-grade chronic inflammation and fetal vascular malperfusion were more prevalent in the second pregnancy among individuals with recurrent chronic villitis, suggesting a progressive worsening of placental pathology. Given its immunologic nature, emerging research suggests that maternal immunomodulatory therapy, such as low-dose aspirin or corticosteroids, may improve pregnancy outcomes in affected individuals [25]. The role of complement deposition (C4d) and regulatory T-cell markers in VUE is under investigation as potential biomarkers for risk stratification and diagnostic refinement [1].

Maternal vascular malperfusion (MVM) is a placental pathology characterized by impaired uteroplacental blood flow, leading to placental hypoplasia, infarction, and retroplacental hemorrhage. Histologically, MVM presents with accelerated villous maturation, increased syncytial knots, and fibrinoid necrosis, reflecting chronic placental insufficiency. Clinically, MVM is strongly associated with hypertensive disorders in pregnancy, particularly chronic hypertension and preeclampsia, which significantly impact placental circulation [27]. In a study by Kovo et al. [28] chronic hypertension emerged as a significant predictor of MVM, increasing the risk more than sixfold, while preeclampsia more than tripled the likelihood of this placental pathology. These vascular abnormalities contribute to FGR, stillbirth, and preterm birth, as compromised placental perfusion limits oxygen and nutrient delivery to the fetus, increasing perinatal morbidity and mortality [29–31]. Findings by Yaguchi et al. [19] further underscore the significance of MVM, identifying it as a potential biomarker for adverse infant neurodevelopmental outcomes, based on consistent associations between MVM and poorer neurodevelopmental scores, even in low-risk populations. MVM has been reported to recur in 10–25 % of subsequent pregnancies, highlighting the importance of early detection and preventive measures. Given the association between MVM and adverse pregnancy outcomes, close monitoring, prophylactic use of acetylsalicylic acid, and uterine artery Doppler assessments are recommended to mitigate risks in future pregnancies [27]. Additionally, early third-trimester ultrasound, and indicated late-preterm and early-term deliveries may be beneficial in optimizing outcomes in subsequent pregnancies [2]. MVM has been identified as a potential marker for long-term cardiovascular risk in women. A study by Janssen et al. [12] demonstrated that women with a history of spontaneous preterm birth (SPTB) and placental MVM had a higher prevalence of hypertension later in life compared to those with SPTB but without MVM (48.4 vs. 35.3 %). Furthermore, these women exhibited higher mean diastolic blood pressure, mean arterial pressure, and HbA_{1c} levels approximately 13 years post-delivery, suggesting an elevated cardiometabolic risk. The detection of placental MVM might help to identify a group of women who are at elevated risk of cardiovascular disease and might benefit from clinical screening programs.

Fetal vascular malperfusion (FVM) is a significant placental pathology characterized by compromised fetal blood flow, often due to umbilical cord abnormalities, fetal cardiac dysfunction, or maternal hypercoagulable states. Structural anomalies such as hypercoiling, excessive cord length, velamentous insertion, and true knots can contribute to circulatory obstruction, increasing the risk of venous stasis, endothelial injury, and thrombosis [32, 33]. Histologically, FVM presents with thrombosis, avascular villi, villous stromal-vascular karyorrhexis, and stem villous vascular obliteration, reflecting ischemic injury to the placenta. Clinically, severe FVM is associated with adverse pregnancy outcomes, including FGR, perinatal stroke, and stillbirth [33]. A systematic review and meta-analysis by Spinillo et al. [34] suggest a strong association between FVM placental lesions and an elevated risk of brain injury in term neonates, as well as neurodevelopmental impairment in both term and preterm infants. MVM, characterized by circulatory abnormalities, can result in fetoplacental unit underperfusion, contributing to the development of FVM. Conditions such as preeclampsia, hypertension, and hypercoagulable states, including antiphospholipid syndrome, lupus anticoagulant, factor V Leiden mutation, protein S deficiency, and protein C deficiency, have been implicated in FVM cases. Additionally, maternal diabetes has been observed as a potential contributing factor in some instances of FVM [32]. FVM linked to an umbilical cord abnormalities generally has a low recurrence rate in subsequent pregnancies. However, the likelihood of recurrence may be higher in cases where there is a maternal or neonatal history of thromboembolic disease [33]. Postpartum counseling should include a detailed explanation of the underlying cause of the adverse pregnancy outcome, such as umbilical cord pathology or fetal vascular thrombosis. In cases of extensive placental

lesions, screening for maternal thrombophilia can be recommended prior to conception to assess potential risk factors. Given the low recurrence rate of FVM, standard fetal monitoring remains appropriate for most subsequent pregnancies; however, enhanced fetal surveillance may be warranted in select cases based on individual clinical risk factors.

Massive perivillous fibrin deposition (MPFD) is a rare placental disorder marked by extensive intervillous fibrin accumulation and maternal floor thickening, leading to villous entrapment and impaired maternal-fetal exchange [35, 36]. The incidence of MPFD is very low, approximately 0.03 %, with recurrence rates of 12–78 % [37]. The etiology of MPFD remains unclear, though maternal alloimmune and autoimmune mechanisms have been implicated [35]. This condition is associated with FGR, prematurity, oligohydramnios, fetal metabolic disorders and malformations, and adverse neonatal outcomes, including an increased risk of neurological impairment [13, 35, 36]. Spinillo et al. [38] studied MPFD in FGR pregnancies, finding it in 11.8 % of cases. MPFD was linked to larger placental volumes, a lower birthweight/placental weight ratio, and higher rates of severe neonatal complications, including intraventricular hemorrhage, necrotizing enterocolitis, and neonatal sepsis. Postpartum counseling should emphasize the high recurrence risk of MPFD and its association with adverse perinatal outcomes, highlighting the need for close monitoring with serial growth ultrasounds in subsequent pregnancies. Although no definitive therapy exists, managing MPFD in subsequent pregnancies may benefit from strategies that support placental function and reduce recurrence risk [13]. Low-dose heparin and aspirin have been utilized to enhance placental circulation and mitigate fibrinoid accumulation, potentially leading to better pregnancy outcomes. Additionally, intravenous immunoglobulin therapy has shown promise in cases associated with antiphospholipid syndrome, providing immunomodulatory effects that may help preserve placental integrity [35]. Further research is needed to refine treatment protocols and optimize perinatal care for affected pregnancies.

Chronic histiocytic intervillositis (CHI) is an extremely rare placental disorder characterized by maternal macrophage infiltration, extensive perivillous fibrin deposition, and trophoblast necrosis, affecting approximately 6 in every 10,000 pregnancies [25, 39]. CHI has been strongly linked to FGR, miscarriage, and stillbirth, with recurrence risks ranging from 70 to 100 %, making it one of the most recurrent placental lesions [25]. The immunopathology of CHI suggests a maternal immune reaction against fetal trophoblast cells, resembling allograft rejection. The role of maternal alloantibodies and complement activation in fetal vascular injury and impaired placental function has been increasingly recognized. Despite its poor perinatal outcomes, therapeutic approaches such as immunosuppressive treatments (corticosteroids, hydroxychloroquine) and antithrombotic therapy (low-molecular-weight heparin, aspirin) have shown promise in reducing recurrence and improving pregnancy outcomes [25, 39]. Due to its high recurrence rate and link to adverse perinatal outcomes, regular monitoring through serial growth ultrasounds is crucial [13].

Placental histological examination plays a crucial role in identifying various other pathological conditions, including benign and malignant masses. While most placental tumors are non-threatening, metastatic malignancies such as melanoma and choriocarcinoma can be detected through histopathologic analysis. Additionally, placental evaluation aids in diagnosing maternal and fetal infections, including cytomegalovirus (CMV), syphilis, *Listeria monocytogenes*, malaria, *Zika virus,* and, less commonly, *herpes simplex virus* or *toxoplasmosis.* Given the potential for serious neonatal and maternal complications, prompt identification and intervention are essential [13].

Challenges: delivering timely, interpretable, and clinically relevant placental histology and clear perinatal counseling

From a healthcare systems perspective, first challenge is ensuring that placental pathology is consistently obtained when indicated and that results are available in a timely fashion. In many settings, placental examination may be underutilized due to cost or availability of expert pathologists. Shortening the turnaround time for pathology reports can also have an impact, particularly for neonatal management decisions. For instance, a rapid identification of ACA or a suspicion of fetal thrombotic vasculopathy might influence neonatal care (antibiotics, brain imaging, etc.) in the first days of life [1]. Placental diagnoses should ideally be documented in the electronic medical record within 48–72 h of receipt, allowing integration into discharge planning and facilitating communication with families [1]. However, timely reporting is often compromised at lower-level delivery sites, where placental specimens must be transferred to higher-level facilities with access to perinatal pathology services. Responsibility for ensuring placental preservation and appropriate handling lies both with healthcare professionals and informed families, underscoring the importance of clear institutional protocols, provider awareness,

and collaborative communication. Women and their partners should be encouraged to advocate for placental preservation at delivery, with transport teams informed of their shared decision to have the placenta reviewed. This ensures specimen integrity and timely transfer to pathology services, reinforcing the role of families as active participants in care and the need for coordinated support across disciplines [40].

Clinical application of placental pathology reports remains challenging due to their complex, anatomically detailed nature and lack of standardized interpretive guidance. While reports often include detailed descriptions of the umbilical cord, membranes, and parenchyma, the absence of clinically oriented correlation limits their utility for obstetricians without specialized training. This challenge is compounded by inconsistent submission criteria, variable reporting standards, and limited exposure to placental pathology in general pathology training. Consequently, clinicians may struggle to interpret findings and translate them into effective patient care strategies [1].

While placental pathology provides valuable insights into pregnancy complications, not all adverse outcomes can be attributed to placental abnormalities, nor do all detected lesions necessarily result in clinical consequences. The extent to which placental findings influence maternal or neonatal health depends on the nature, severity, and context of the observed lesions. This perspective is supported by the study by Romero et al. [24], which analyzed 944 placentas from term pregnancies with normal outcomes. Remarkably, 78 % of these placentas exhibited histologic lesions, primarily mild inflammatory or vascular abnormalities, despite the absence of any clinical complications. These findings underscore that many placental changes may reflect physiological processes associated with term gestation or parturition rather than pathology, reinforcing the importance of interpreting histologic findings within the full clinical context. Awareness of the potential discordance between histologic and clinical ACA is crucial, as histologic evidence of inflammation may be present in the absence of clinical manifestations; therefore, such findings should not be regarded as definitive clinical markers but rather interpreted within the broader context of maternal and fetal conditions. Findings from Horvath et al. [41] support this distinction, demonstrating that histologic chorioamnionitis was detected in 13.6 % of placentas, yet only 6.2 % of these cases met clinical criteria for chorioamnionitis. Notably, silent chorioamnionitis was significantly more frequent in preterm pregnancies (30.0 %) compared to term deliveries (5.1 %), reinforcing the need for a nuanced approach when interpreting histologic inflammation. Additionally, microbiological cultures from the intrauterine cavity showed that bacterial colonization occurred in 19.9 % of cases, yet some histologically confirmed cases lacked identifiable microbial pathogens. These findings underscore the complexity of intra-amniotic infections and the necessity of integrating histological, microbiological, and clinical assessments in pregnancy management. Similarly, before initiating management for VUE, clinicians should assess whether the lesion is truly implicated in adverse outcomes, given that low-grade VUE is frequently observed and can also appear in otherwise normal pregnancies [23], just as morphologic changes of FVM may reflect normal degenerative changes following fetal demise rather than true pathology [13, 34].

One of the persistent challenges is also the variability in interpretation of placental lesions, reflected in limited interobserver reliability, which can lead to inconsistent clinical decisions and missed opportunities for targeted neonatal or maternal interventions. Redline et al. [42] conducted a focused evaluation of interobserver reliability for placental lesions defined by the Amsterdam consensus, using scanned digital slides from a subset of cases selected to represent a spectrum of pathologic findings. While diagnostic agreement was good to excellent for inflammatory lesions such as acute chorioamnionitis and villitis of unknown etiology, reliability was notably lower for fetal vascular malperfusion. Accelerated villous maturation demonstrated the poorest agreement among observers. These findings underscore the need for more explicit histologic criteria and clearer diagnostic boundaries to improve reproducibility and facilitate interdisciplinary communication.

Finally, an important yet often overlooked challenge lies in communicating placental pathology findings to women and their partners in a clear and sensitive manner, ensuring that the information is neither misinterpreted nor causes undue anxiety, especially when the clinical relevance of certain lesions remains uncertain. A recent study demonstrated that including lay-language comments in placental pathology reports significantly improved providers' understanding and confidence in discussing findings with patients, highlighting the value of patient-centered communication strategies [43].

Opportunities: enhancing postpartum counseling for patients through standardized histologic evaluation, interdisciplinary dialogue, and foundations for future research

Translating placental pathology findings into clinical care is increasingly prioritized in perinatology, as it helps clarify

the cause of adverse outcomes and guide future pregnancy management. Firstly, placental examination provides immediate postnatal diagnostic value, particularly when findings, such as ACA in preterm labor, offer a clear etiology. This improves documentation, informs targeted treatment (e.g. antibiotics for infection), and facilitates patient understanding and emotional processing. Certain lesions, like hematogenous villitis, may also prompt urgent neonatal evaluation. Secondly, placental pathology informs management of subsequent pregnancies by identifying underlying maternal conditions or recurrent risk factors. Findings such as diffuse infarction or fetal vascular thrombosis may prompt maternal evaluation for thrombophilia or autoimmune conditions, while specific lesions can direct genetic counseling, early referral to high-risk care, or screening strategies. Prior placental pathology also supports personalized surveillance and prophylaxis planning. Finally, the implications of placental pathology extend beyond the immediate perinatal period to long-term maternal and child health. High-grade maternal vascular malperfusion lesions not only explain acute pregnancy complications but are also linked to elevated maternal cardiovascular risk later in life [11, 44]. Likewise, evidence suggests that placental underperfusion may contribute to adult-onset hypertension in offspring, supporting the developmental origins of health and disease hypothesis [45].

Clearly defined, locally adapted indications for placental histopathological examination are essential to ensure consistent and timely decision-making in clinical practice. A multidisciplinary consensus outlines maternal, fetal, and placental criteria guiding examination to optimize perinatal care [7]. Recently, Roberts et al. [6] proposed comprehensive, expert-driven criteria for when placentas should be submitted for histologic evaluation, aiming to standardize practice across obstetric and neonatal care.

Building on this framework, it is equally important to ensure that placental findings are not only obtained but also meaningfully applied in postnatal management. After neonatal stabilization, early interdisciplinary discussion is recommended to support timely clinical decision-making. As emphasized by Redline et al. [1], collaboration among neonatologists, neurologists, obstetricians, and perinatal pathologists can guide diagnostic workup, inform prognosis, and support individualized care, even in cases with subtle or evolving clinical signs.

This approach is further supported by emerging interdisciplinary perspectives in fetal-neonatal neurology, which suggest that even neonates with minimal clinical signs of encephalopathy or seizures, who may not receive therapeutic hypothermia or antiepileptic drugs, could benefit from placental-cord assessments to uncover antepartum disease pathways [40]. A wide range of placental lesions, including villous dysmaturity, vascular malperfusion, chronic inflammatory conditions, and structural anomalies of the uterus, placenta, or cord, should be incorporated into considerations for genetic testing. These findings may reflect underlying gene-environment interactions and warrant further evaluation in the context of fetal-neonatal brain injury [46].

An opportunity to overcome the common challenge of unclear or insufficiently clinically oriented placental pathology reports lies in improving interdisciplinary communication between pathology and obstetric teams. This can be achieved by implementing standardized terminology, such as that recommended by the Amsterdam consensus, and by ensuring pathology reports include a clear interpretation section written in accessible, clinically meaningful language [16, 17]. Gross anatomical observations made at delivery, such as placental surface abnormalities (e.g., calcifications and green or pale discoloration), and cord anomalies like knots and velamentous insertion, as well as amniotic fluid volume and the amount of vaginal blood expressed, should be documented and conveyed by the obstetric team, as they may inform pathology interpretation and enhance clinical relevance [1]. To extend this collaborative approach into neonatal care, neonatal and neurology providers are encouraged to review prepared placental slides with the perinatal pathologist during work rounds using a binocular microscope. This hands-on engagement promotes shared understanding and supports informed decision-making. Equally important is fostering open dialogue: obstetric providers should feel encouraged to consult with placental pathologists when clarification is needed. Regular interdisciplinary meetings or case discussions can further support the effective translation of histologic findings into actionable clinical insights and enhance the quality of patient counseling.

Incorporating placental histology into routine obstetric care offers substantial educational benefits for clinicians. Regular engagement with histopathological findings enhances diagnostic precision, deepens understanding of pregnancy-related pathophysiology, and promotes more thoughtful clinical decision-making. Although variability in training and reporting remains a challenge, these gaps can be addressed through interdisciplinary collaboration and targeted educational efforts.

Placental histology offers valuable research opportunities by linking pathology findings with clinical outcomes to uncover novel associations, refine diagnostics, and support predictive modeling. Integrating research into care also enables quality improvement, fosters collaboration, and contributes to the development of targeted therapies [45, 47,

48]. Future progress in placental pathology will likely require moving beyond routine histology. Several ancillary techniques not yet fully validated for clinical diagnosis, such as immunohistochemistry for markers like CD34, CD15, C4d, and caspase-3, can provide additional diagnostic clarity and may be considered in select cases [1].

Conclusions

Placental pathology represents a vital yet underutilized resource in perinatal medicine. To harness its full clinical potential, efforts must be directed toward bridging the communication gap between pathologists and clinicians, ensuring that histologic findings are not only accurately interpreted but also meaningfully applied in clinical decision-making. This bridge must ultimately extend to women and their partners, supporting transparent, informed postpartum counseling.

To fully translate placental findings into actionable care, institutions are encouraged to establish dedicated multidisciplinary review committees, inclusive of obstetricians, maternal-fetal medicine specialists, midwives, nurses, pediatricians, geneticists, and pathologists, to provide a comprehensive evaluation of cases with adverse pregnancy outcomes and foster trust and compliance through collaborative care.

In this collaborative framework, it is essential to recognize women and their partners as active stakeholders in obstetric decision-making. Scher et al. [15] propose a transdisciplinary model of care that integrates maternal, fetal, neonatal, neurological, and pathology perspectives, encouraging shared clinical decisions from preconception through the first 1,000 days of life. By involving families in discussions with obstetricians, midwives, nurses, and other specialists, care bundles can be tailored to individual needs, fostering trust, compliance, and improved outcomes. This approach promotes continuity of care and empowers families to advocate for practices such as placental preservation and pathology review, even in resource-limited settings.

Histological findings should be interpreted in context, alongside clinical history, laboratory data, genetic testing, microbiologic results, and neonatal assessments. Appropriate and uniform clinical follow-up for important placental diagnoses should be tracked, documented, and discussed at regularly scheduled interdepartmental conferences, which should also generate detailed, patient-centered reports to be shared during postpartum consultations and used to inform future pregnancy management [1]. Such structured collaboration is essential to optimizing maternal–fetal care, reducing recurrence risk, and advancing evidence-based, patient-centered obstetric practice.

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