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Research Article

Retrospective evaluation of histopathological lesion patterns and obstetric indications in placentas referred for pathological examination

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ABSTRACT

Aim: To characterize the distribution of histopathological lesion patterns in placentas referred for pathological examination on obstetric indication, to define the obstetric indications for referral, and to evaluate the associations between lesion patterns and clinical indications at a single tertiary center.

Methods: In this single-center, retrospective, descriptive and analytical archival study, records of placentas obtained from deliveries and submitted for pathological examination on clinical indication between January 2018 and February 2026 were reviewed. Obstetric indications and histopathological findings were extracted from the clinical and pathology reports and classified according to the Amsterdam Placental Workshop Group consensus into maternal vascular, fetal vascular, acute inflammatory, and chronic inflammatory patterns. Associations were assessed with the chi-square or Fisher exact test, and continuous variables were compared using the Mann-Whitney U test.

Results: A total of 399 placentas were analyzed. The mean maternal age was 30.3 years, and 73.9% of placentas were obtained by cesarean section. The most frequent indications were hypertensive disorders of pregnancy (14.3%), preterm birth (13.5%), congenital anomaly (12.8%), and fetal growth restriction (12.5%). Most placentas were reported as normal or non-specific (86.7%), and a defined lesion pattern was identified in 13.3%, among which acute inflammatory (5.3%) and maternal vascular (4.8%) patterns predominated. Acute inflammatory lesions were significantly more common among placentas referred for suspected infection (42.9%; $p = 0.005$), whereas the associations of maternal vascular malperfusion with hypertensive disorders, growth restriction, and diabetes did not reach significance.

Conclusion: In this large single-center series, most referred placentas were histologically unremarkable in routine reporting, with acute inflammatory and maternal vascular lesions predominating among abnormal cases, and acute inflammatory lesions were associated with suspected infection. Standardized sampling and synoptic reporting are needed to realize the diagnostic value of placental examination.

Keywords: placenta, placenta diseases, chorioamnionitis, pregnancy complications, pathology, retrospective studies

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Introduction

The placenta is a highly specialized, dynamic organ that lies at the center of the maternofetal interface and sustains fetal growth and development throughout gestation, while also serving important immunological and barrier functions (1,2). Because the structural and functional integrity of the placenta is tightly coupled to the intrauterine environment, a wide spectrum of obstetric complications, including fetal growth restriction (FGR), hypertensive disorders of pregnancy, preterm birth, gestational diabetes mellitus (GDM), placental abruption, and intrauterine fetal death, leaves identifiable morphological signatures in the placenta. Consequently, histopathological examination of the placenta has become a valuable tool for elucidating the pathophysiological basis of adverse pregnancy outcomes and for informing the management of subsequent pregnancies (2,3).

To standardize the reporting and interpretation of these changes, the Amsterdam Placental Workshop Group Consensus Statement classified placental injury into a limited number of reproducible patterns: maternal vascular malperfusion (MVM), fetal vascular malperfusion (FVM), acute inflammatory lesions (the amniotic fluid infection sequence, i.e., acute chorioamnionitis and the maternal and fetal inflammatory responses), and chronic inflammatory lesions (chiefly villitis of unknown etiology and related chronic patterns). This framework and its subsequent refinements continue to shape contemporary placental diagnostics, although certain entities, such as the definition and clinical significance of basal chronic villitis, remain the subject of ongoing consensus discussion (4).

A growing body of evidence links specific lesion patterns to distinct clinical presentations. Maternal vascular malperfusion lesions have been consistently associated with FGR and with hypertensive disorders of pregnancy, and have more recently been reported as a prominent feature of placentas from GDM pregnancies, particularly when complicated by growth restriction (3,5). Acute inflammatory lesions have been related to spontaneous preterm birth, increased neonatal intensive care requirements, and neonatal morbidity, and may act as independent predictors of adverse neonatal outcome and even of recurrent early preterm birth (6,7). Placental pathology has also been shown to differ between early- and late-onset presentations of placenta-associated complications; for example, early placental abruption is characterized by higher rates of maternal vascular malperfusion and maternal inflammatory response lesions and by more severe neonatal morbidity than late abruption (8). Comparable pattern-specific associations have been described in preeclampsia, including postpartum-onset disease, and in twin gestations complicated by growth restriction and preeclampsia (9,10).

Despite this accumulating knowledge, most published studies focus on a single clinical entity (e.g., GDM, FGR, or abruption in isolation) and are therefore limited in their ability to characterize the overall spectrum of placental pathology encountered in routine obstetric practice. Comprehensive, single-center analyses that evaluate all placentas referred for pathological examination across the full range of clinical indications, and that systematically relate histopathological lesion patterns to those indications, remain comparatively scarce, particularly within our national context.

Accordingly, the present study was designed to characterize the distribution of histopathological lesion patterns in placentas submitted for pathological examination at our institution, to define the obstetric indications for referral and their relative frequencies, and to evaluate the associations between lesion patterns and clinical indications. We specifically sought to determine which clinical presentations are most frequently associated with maternal vascular, fetal vascular, acute inflammatory, and chronic inflammatory lesion groups. In doing so, we

aimed to establish the institutional profile of placental pathology and to compare our findings with those reported in the international literature.

Materials and Methods

Study design and setting

This was a single-center, retrospective, descriptive and analytical archival study conducted at the Department of Medical Pathology, Kocaeli University Faculty of Medicine. Archived records of placentas obtained from deliveries performed at our institution between January 2018 and February 2026 and submitted for pathological examination on clinical indication were reviewed retrospectively. No intervention was performed; the study was based exclusively on the analysis of existing routine clinical and pathological records.

Ethical considerations

The study was approved by the Non-Interventional Clinical Research Ethics Committee of Kocaeli University Faculty of Medicine (approval no. GOKAEK-2026/05/35; project no. 2026/148; date of approval 06 March 2026) and was conducted in accordance with the principles of the Declaration of Helsinki. Because the study was a retrospective review of anonymized archival data with no patient contact, the requirement for written informed consent was waived by the ethics committee. All cases were anonymized and assigned a unique code number, and no identifying information was transferred to the study dataset.

Study population and case selection

All placentas from deliveries performed at our institution during the study period that were referred to the Department of Medical Pathology for histopathological examination on clinical indication were considered for inclusion. Cases were eligible if the delivery was performed at our institution, the placenta was submitted on a clinical indication, a completed histopathology report was available in the archive with adequate macroscopic and microscopic evaluation, and the obstetric indication for referral was retrievable from the clinical records. Specimens representing tubal ectopic pregnancies without a delivered placenta, cases with inadequate sampling or loss of tissue integrity precluding reliable assessment, and records with missing core variables were excluded.

Data sources and variables

Data were retrieved from the Hospital Information Management System, the archival records of the Department of Medical Pathology, electronic pathology reports, and delivery and clinical case files, and were entered into a structured database using a standardized case report form. The recorded variables comprised maternal age, gestational age at delivery, mode of delivery, the obstetric indication for referral, and the macroscopic and microscopic placental findings. Obstetric indications and placental histopathological findings were derived by systematic review of the free-text clinical summaries and pathology reports and were classified into predefined categories. Because a single placenta could be referred for more than one clinical reason, the obstetric indications were treated as non-mutually-exclusive variables. In a subset of cases a fetus was submitted together with the placenta for concurrent fetal autopsy; in these cases the reported fetal findings, including any structural anomalies, were recorded and reviewed alongside the placental diagnosis.

Histopathological classification

Placental lesions were categorized according to the Amsterdam Placental Workshop Group consensus framework into four groups (4): maternal vascular lesions (e.g., infarction, distal villous hypoplasia, increased syncytial knots, and decidual arteriopathy); fetal vascular lesions (e.g., avascular villi, fetal vascular thrombosis,

and stem villous vessel obliteration); acute inflammatory lesions (chorioamnionitis and funisitis); and chronic inflammatory lesions (villitis, chronic intervillositis, and chronic deciduitis). On the basis of the predominant findings, each placenta was assigned to a single mutually exclusive pattern: maternal vascular, fetal vascular, acute inflammatory, chronic inflammatory, combined (two or more groups present), or normal/non-specific. Classification was based on the diagnoses recorded in the original reports, and no re-sectioning, additional staining, or re-review of slides was undertaken.

Statistical analysis

Continuous variables were summarized as mean ± standard deviation and as median (range), and categorical variables as frequency and percentage. The normality of continuous variables was assessed using the Shapiro-Wilk test. Associations between the placental lesion groups (maternal vascular, fetal vascular, acute inflammatory, and chronic inflammatory) and the obstetric indications were evaluated with the Pearson chi-square test or, when expected cell counts were small, the Fisher exact test. Continuous variables were compared between placentas with and without an abnormal lesion pattern using the Mann-Whitney U test, as these data were not normally distributed. All tests were two-sided, and a p value less than 0.05 was considered statistically significant. Analyses were performed using GraphPad Prism (version 8.0.1; GraphPad Software, San Diego, CA, USA).

Results

During the study period, 399 placentas obtained from deliveries and meeting the inclusion criteria were analyzed, after 15 specimens representing tubal ectopic pregnancies without a delivered placenta had been excluded. The mean maternal age was 30.3 ± 6.1 years (median 30 (17-45)). Gestational age at delivery was documented in 347 cases (87.0%), with a mean of 30.7 ± 7.8 weeks (median 34 (5-41)); 271 (78.1) of these deliveries occurred before 37 weeks of gestation. Most placentas were obtained by cesarean section (295 (73.9)), whereas 104 (26.1) followed vaginal delivery. The demographic and clinical characteristics of the cohort are summarized in Table 1.

A single placenta was frequently referred for more than one clinical reason; therefore the indication categories are not mutually exclusive and their sum exceeds 100%. The most common

Table 1. Demographic and clinical characteristics of the study cohort (N = 399).

Characteristic	Value
Maternal age, years, mean ± SD	30.3 ± 6.1
Maternal age, median (range)	30 (17-45)
Gestational age, weeks, mean ± SD[a]	30.7 ± 7.8
Gestational age, median (range)[a]	34 (5-41)
<28 weeks, n	87
28-33 weeks, n	undefined
34-36 weeks, n	undefined
≥37 weeks, n	76
Preterm (<37 weeks), n (%)[a]	271 (78.1)
Mode of delivery, cesarean, n (%)	295 (73.9)
Mode of delivery, vaginal, n (%)	104 (26.1)

SD, standard deviation. [a] Gestational age was available for 347 of 399 cases (87.0%); percentages for gestational-age categories are based on this subset.

Table 2. Obstetric indications for placental referral (N = 399; categories are not mutually exclusive).

Obstetric indication	n	%
Preeclampsia/HDP	57	14.3
Preterm/PPROM	54	13.5
Congenital anomaly	51	12.8
FGR	50	12.5
Multiple gestation	49	12.3
GDM	39	9.8
Oligo/polyhydramnios	35	8.8
Abruption	26	6.5
Placenta previa/accreta	24	6.0
IUFD/fetal loss	13	3.3
Infection susp.	7	1.8
Other/unspecified	120	30.1

documented obstetric indications were hypertensive disorders of pregnancy, including preeclampsia (57; 14.3%), preterm birth or preterm premature rupture of membranes (54; 13.5%), congenital anomalies (51; 12.8%), fetal growth restriction (50; 12.5%), and multiple gestation (49; 12.3%). Gestational diabetes mellitus, amniotic fluid abnormalities, placental abruption, placenta previa or accreta spectrum, intrauterine fetal loss, and suspected infection accounted for the remaining indications. In 120 placentas (30.1%) no specific high-risk indication could be retrieved from the clinical records. The full distribution of indications is presented in Table 2 and Figure 1.

On histopathological review, the great majority of placentas showed no defined pathological pattern and were reported as normal or as showing only non-specific changes (346; 86.7%). At least one of the four Amsterdam lesion groups was identified in 53 (13.3) placentas. Among the abnormal placentas, an acute inflammatory pattern was the most frequent (21; 5.3%), followed by a maternal vascular pattern (19; 4.8%) and a chronic inflammatory pattern (9; 2.3%); a fetal vascular pattern and combined patterns were rare (two cases each). The individual lesions recorded most often were infarction (n = 17), increased syncytial knots (n = 4), chorioamnionitis (n = 22), chronic deciduitis (n = 5), fetal vascular thrombosis (n = 2), calcification (non-specific) (n = 24), perivillous fibrin (n = 5), hemorrhage/hematoma (n = 6). The distribution of lesion patterns is shown in Table 3 and Figure 2.

Table 3. Distribution of placental histopathological lesion patterns (mutually exclusive, N = 399).

Lesion pattern	n	%
Normal/non-specific	346	86.7
Acute inflammatory	21	5.3
Maternal vascular	19	4.8
Chronic inflammatory	9	2.3
Combined	2	0.5
Fetal vascular	2	0.5
Any abnormal pattern[a]	53	13.3

[a] Placentas showing at least one maternal vascular, fetal vascular, acute inflammatory, or chronic inflammatory lesion.

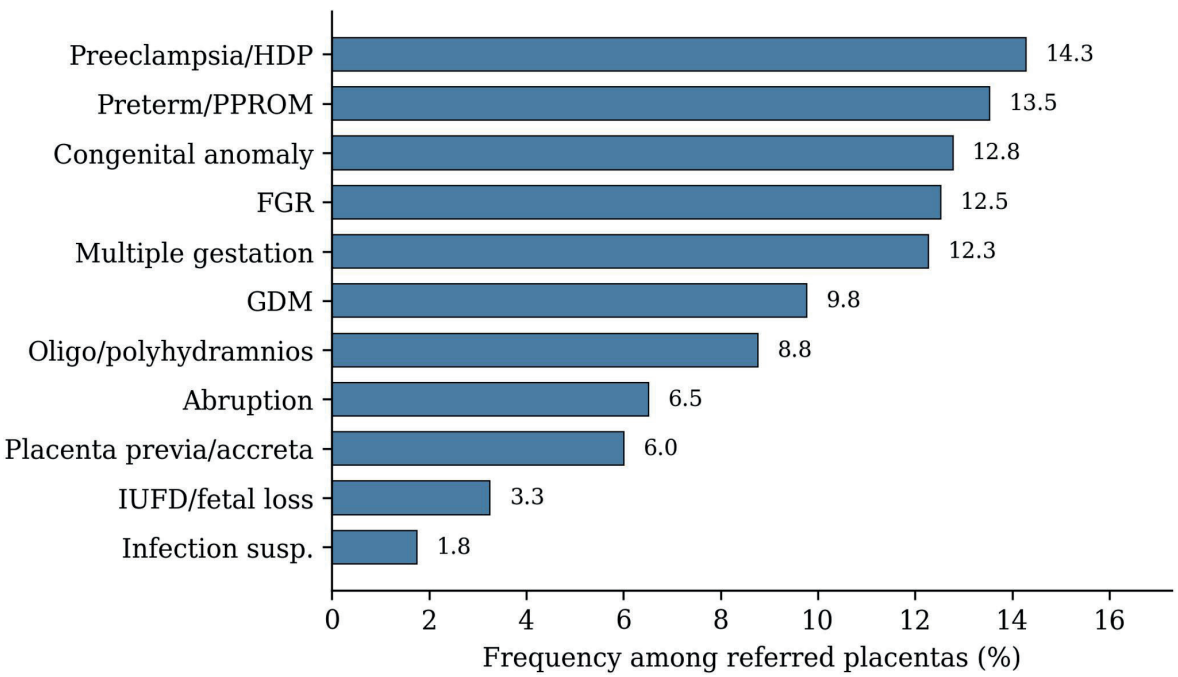

Figure 1. Frequency of documented obstetric indications for placental referral.

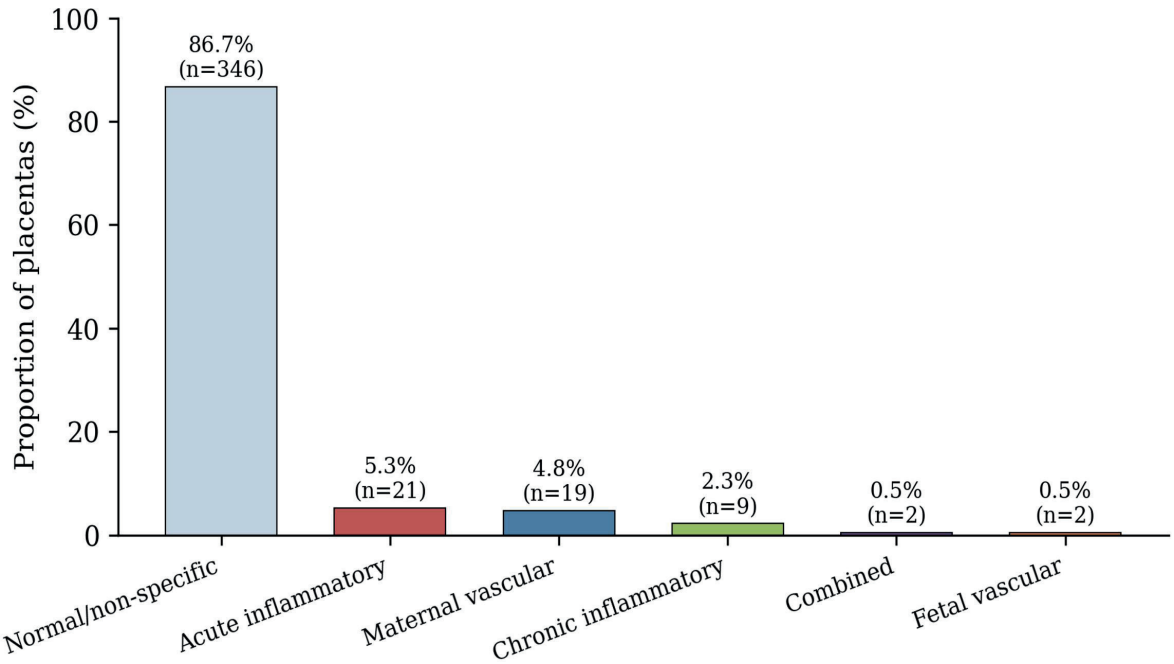

Figure 2. Distribution of placental histopathological lesion patterns.

Table 4. Prevalence of placental lesion groups according to obstetric indication.

Indication	n	MVM, n (%)	AI, n (%)	Any abnormal, n (%)	p[a]
Preeclampsia/HDP	57	4 (7.0)	5 (8.8)	9 (15.8)	0.530
FGR	50	5 (10.0)	3 (6.0)	7 (14.0)	0.826
GDM	39	3 (7.7)	4 (10.3)	6 (15.4)	0.624
Preterm/PPROM	54	2 (3.7)	4 (7.4)	9 (16.7)	0.396
Abruption	26	2 (7.7)	2 (7.7)	6 (23.1)	0.136
IUFD/fetal loss	13	1 (7.7)	2 (15.4)	3 (23.1)	0.394
Infection susp.	7	0 (0.0)	3 (42.9)	3 (42.9)	0.053
Placenta previa/accreta	24	0 (0.0)	0 (0.0)	0 (0.0)	0.057
Multiple gestation	49	1 (2.0)	1 (2.0)	2 (4.1)	0.043
Congenital anomaly	51	3 (5.9)	2 (3.9)	6 (11.8)	0.829
Oligo/polyhydramnios	35	1 (2.9)	1 (2.9)	2 (5.7)	0.202

MVM, maternal vascular malperfusion; AI, acute inflammatory. [a] Fisher exact test comparing the presence of any abnormal pattern in the indicated group versus the remainder of the cohort. Acute inflammatory lesions were significantly associated with suspected infection (3/7; 42.9%; p = 0.005).

The associations between the major lesion groups and the obstetric indications are presented in Table 4. Overall, the presence of any abnormal lesion pattern did not differ significantly across most indications. Maternal vascular lesions were somewhat more frequent among placentas referred for fetal growth restriction (10.0%) and hypertensive disorders (7.0%) than in the remainder of the cohort (Figure 3), but these differences did not reach statistical significance (all $p > 0.05$). Acute inflammatory lesions were significantly more common among placentas referred for suspected infection, being present in 3 of 7 such cases (42.9%) compared with the rest of the cohort (Fisher exact test, $p = 0.005$). Multiple gestation

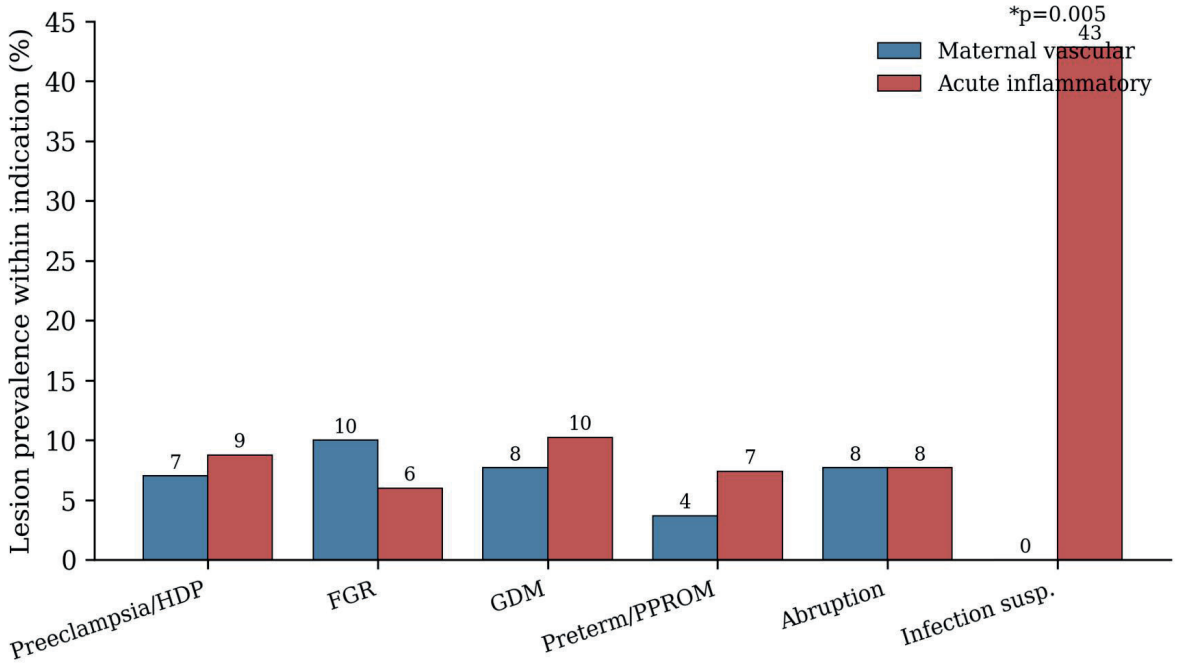

Figure 3. Prevalence of maternal vascular and acute inflammatory lesions within selected obstetric indications
*Fisher exact test, p = 0.005 for acute inflammatory lesions in suspected infection.

was inversely associated with the presence of any abnormal pattern (4.1%; p = 0.043), and no defined lesion pattern was identified in any placenta referred for placenta previa or accreta spectrum. No significant difference in maternal age or gestational age was observed between placentas with and without abnormal patterns, and the abnormal-pattern rate did not differ by mode of delivery (all p > 0.05).

Fourteen of the 399 placentas (3.5%) were submitted together with a fetus for concurrent fetal autopsy, all following a mid-trimester intrauterine loss or termination of pregnancy (gestational age approximately 11 to 22 weeks). A fetal structural anomaly was identified in 8 of these 14 fetuses and comprised central nervous system defects (anencephaly, spina bifida, and holoprosencephaly), a bilateral renal and bladder agenesis sequence, hydrops fetalis, a diaphragmatic hernia accompanied by a ventricular septal defect, dysmorphic features suggestive of aneuploidy, and a disorder of sex development; the remaining 6 fetuses showed growth restriction or nonspecific autolytic changes without a defined structural anomaly. The corresponding placentas were histologically unremarkable in 10 cases and showed only nonspecific coagulative necrosis or immature villi consistent with fetal retention in 3 cases and focal infarction in 1 case. No defined Amsterdam lesion pattern was consistently associated with the fetal anomalies.

Discussion

In this single-center retrospective analysis of 399 placentas submitted for pathological examination on obstetric indication, the majority were reported as histologically normal or as showing only non-specific changes, and a defined Amsterdam lesion pattern was identified in fewer than one in seven placentas. Among the placentas with a defined pattern, acute inflammatory and maternal vascular lesions predominated, whereas fetal vascular and combined patterns were uncommon. The only statistically significant clinicopathological relationship was the concentration of acute inflammatory lesions among placentas referred

for suspected infection. These findings establish the institutional profile of placental pathology at our center and, when read against the international literature, highlight both the value and the practical limitations of routine placental reporting.

The proportion of placentas with a defined lesion pattern in our cohort (13.3%) was substantially lower than that reported in comparable series. In a South African high-risk cohort of 176 placentas, Stevens et al.(11) identified maternal vascular malperfusion in 51.1% and ascending infection in 35.8% of cases, and Al Fahdi et al.(2) reported abnormal findings in 74% of placentas from adverse-outcome pregnancies. Using a standardized request form and a protocol-based workup, Strahler et al.(12) detected at least one pathological finding in 86.3% of clinically indicated singleton placentas. Most strikingly, Romero et al.(13) demonstrated that even placentas from term pregnancies with an entirely normal outcome harbored inflammatory or vascular lesions in 78% of cases, although severe or high-burden lesions were present in only about 1%. Taken together, these data indicate that the apparent prevalence of placental lesions depends heavily on the intensity of sampling and the rigor of reporting rather than on clinical risk alone. Our comparatively low detection rate is therefore best interpreted as a reflection of routine, largely biopsy-level reporting that did not consistently apply the systematic sampling and terminology recommended by the Amsterdam consensus (4,14), rather than as evidence of a genuinely low burden of placental disease.

The significant association between acute inflammatory lesions and suspected infection is biologically coherent and consistent with prior work. Acute chorioamnionitis and funisitis represent the histological correlates of the amniotic fluid infection sequence and of the maternal and fetal inflammatory responses (14). Orsaria et al.(6) linked acute placental inflammation to adverse neonatal outcomes, and Mizrachi et al.(7) reported that isolated acute inflammatory lesions were associated with recurrent early preterm birth. Romero et al.(13) found acute inflammatory lesions to be the most prevalent category even in uncomplicated term pregnancies, with a strong relationship to labor, and Stevens et al.(11) similarly observed ascending infection as one of the two dominant cluster diagnoses in a high-risk population. The clustering of acute inflammatory lesions within our infection-referred group therefore supports the concordance between the clinical indication and the underlying histopathology, even in a dataset with otherwise limited lesion documentation.

In contrast, the anticipated associations between maternal vascular malperfusion and hypertensive disorders, fetal growth restriction, and gestational diabetes were not statistically confirmed, although maternal vascular lesions were numerically more frequent among placentas referred for fetal growth restriction and hypertensive disorders. This diverges from the robust maternal vascular malperfusion and preeclampsia association reported by Stevens et al.(11), the maternal vascular malperfusion findings in gestational diabetes described by Byrne et al.(3), the growth-restricted preterm correlations reported by Dankó et al.(5), and the predominance of maternal vascular malperfusion among indicated singleton placentas found by Strahler et al.(12). A likely explanation is the systematic under-recording of the subtle villous features of maternal vascular malperfusion, such as increased syncytial knots, distal villous hypoplasia, and accelerated villous maturation, in routine reports. Stevens et al.(11), for example, recorded increased syncytial knots in 52.8% of their cases, whereas this feature was documented in only a small minority of ours. The cluster analysis reported by Boujenah et al.(15) likewise linked a vascular placental profile to preeclampsia, HELLP syndrome, and fetal growth restriction, reinforcing the view that these associations exist but require consistent recognition and reporting of maternal vascular malperfusion features to be detected.

No defined parenchymal lesion pattern was identified in any placenta referred for placenta previa or accreta spectrum. This is expected, because the accreta spectrum is defined by abnormal adherence or invasion at the villous and myometrial interface rather than by the parenchymal injury patterns of the Amsterdam classification. In these cases, placental examination is directed toward confirming the depth of invasion, as reflected in the large Turkish accreta-spectrum series reported by Cetinkaya Demir et al.(16), and antenatal assessment increasingly relies on imaging-based prediction of invasion and hemorrhage, as described by Kükrer et al.(17). The absence of Amsterdam patterns in this subgroup therefore reflects the distinct diagnostic purpose of examination in the accreta spectrum rather than a true lack of pathology.

Multiple gestation was inversely associated with the presence of any abnormal pattern in our cohort. This accords with the observations of Strahler et al.(12), who found lower concordance between the clinical indication and the histopathological findings in twin placentas and reported that a substantial proportion of twin placentas were histologically unremarkable, in part because twins are frequently delivered electively at 34 to 38 weeks and are commonly submitted for reasons such as documentation of chorionicity rather than for a specific lesion-associated complication. Matthews et al.(10) similarly noted that placental lesions in twin gestations were most informative when the pregnancies were complicated by growth restriction or preeclampsia. The lower abnormality rate among our multiple gestations is thus consistent with a referral pattern driven by protocol rather than by suspected placental disease.

A subset of the placentas in our series was examined together with a fetus following a mid-trimester loss or termination, most often performed for a congenital anomaly. In these cases the fetal structural anomalies, which were predominantly central nervous system and other malformations, were generally not accompanied by a specific placental lesion pattern, and the placentas were most often histologically unremarkable or showed only changes attributable to fetal retention. This observation is consistent with the concept that many congenital anomalies arise from primary genetic or early embryonic mechanisms rather than from placental malperfusion or inflammation, and it supports the practice of correlating placental findings with a dedicated fetal autopsy rather than relying on the placenta alone to account for an anomalous fetus (18).

Collectively, our findings underscore the importance of standardized sampling and structured reporting if the clinical and prognostic potential of placental examination is to be realized. As emphasized by Turowski et al.(18), placental pathologists employ terminology, such as maternal vascular malperfusion, that is distinct from clinical diagnoses, such as preeclampsia, and the utility of a report depends on clear, standardized diagnoses that clinicians can interpret and act upon. Stevens et al.(11) demonstrated that placental histopathology is frequently underutilized in the formulation of subsequent-pregnancy management plans. The stepwise application of the Amsterdam framework (14) and the adoption of a synoptic reporting template (4,18) would be expected to improve both the detection and the communication of clinically relevant lesions in a high-volume setting such as ours, and to strengthen the clinician and pathologist collaboration on which the effective use of placental pathology ultimately depends.

Limitations

This study has several limitations. First, it was a retrospective, single-center analysis based on routine archival reports, and the lesion classification was derived from the free-text diagnoses recorded at the time of original sign-out rather than from a re-review of histological slides. Lesions that were not explicitly stated in the report could not be captured, which almost certainly led to underestimation of maternal and fetal vascular malperfusion, whose

villous features are easily overlooked without dedicated sampling. The resulting abnormality rate should therefore be regarded as a lower bound that reflects reporting practice rather than the true prevalence of placental disease, as illustrated by the far higher rates in protocol-based cohorts (11-13). Second, sampling was not standardized across the study period, and many specimens were reported at a brief, biopsy-level of detail. Third, the obstetric indications were extracted from free-text clinical notes and treated as non-mutually-exclusive categories; several indications, most notably suspected infection, comprised small numbers, so the significant association between acute inflammatory lesions and infection rests on few cases and warrants cautious interpretation. Fourth, gestational age was unavailable for a minority of cases, and fetal status and placental weight were not reliably recorded, precluding weight-based assessment of maternal vascular malperfusion and correlation with neonatal outcomes. Finally, no neonatal or subsequent-pregnancy outcomes were available, and the sparse event counts precluded multivariable adjustment. In addition, although a fetus was examined together with the placenta in a subset of cases, systematic fetal phenotyping, imaging, and karyotype results were not available for all of these cases, which limited a more detailed correlation between specific fetal anomalies and placental findings.

Conclusion

In this large single-center series, most placentas referred for pathological examination on obstetric indication were reported as normal or non-specific, and among the placentas with a defined pattern, acute inflammatory and maternal vascular lesions predominated. Acute inflammatory lesions were significantly associated with suspected infection, whereas the expected associations of maternal vascular malperfusion with hypertensive disorders, fetal growth restriction, and gestational diabetes did not reach significance. The marked discrepancy between our detection rate and those of protocol-based cohorts indicates that routine reporting under-recognizes Amsterdam lesion patterns and argues for standardized sampling and synoptic reporting to realize the diagnostic and prognostic value of placental examination. Establishing this institutional profile provides a foundation for improving clinician and pathologist communication and for future prospective studies incorporating systematic histological workup and neonatal outcomes.

Ethical approval

The study was approved by the Non-Interventional Clinical Research Ethics Committee of Kocaeli University Faculty of Medicine (approval no. GOKAEK-2026/05/35; project no. 2026/148; date 6 March 2026).

Author contribution

The authors confirm contribution to the paper as follows: Study conception and design: BYB; data collection: BYB, GK; analysis and interpretation of results: BYB, GV, YD; draft manuscript preparation: BYB. All authors reviewed the results and approved the final version of the article.

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Conflict of interest

The authors declare that there is no conflict of interest.

REFERENCES

1. Govender N, Lazarus L, Abel T, Naicker T. Placental morphology and morphometry: is it a prerequisite for future pathological investigations? Adv Exp Med Biol. 2023;1392:85-105. [Crossref]
2. Al Fahdi M, Riyami NA, Ahmed B. Placental histopathological abnormalities in adverse obstetric outcomes: a retrospective cross-sectional study at Sultan Qaboos University Hospital. BMC Womens Health. 2024;24(1):613. [Crossref]
3. Byrne J, Ranaei-Zamani N, Hutchinson JC, Hillman S. A retrospective analysis of placental histopathological findings in gestational diabetes mellitus (GDM). Placenta. 2025;162:20-26. [Crossref]
4. Khong TY, Kim CJ, Rogers BB. Amsterdam Placental Workshop Group Consensus Statement definitions revisited: basal chronic villitis. Virchows Arch. 2025;487(3):501-509. [Crossref]
5. Dankó I, Kelemen E, Tankó A, Cserni G. Correlations of placental histopathology, neonatal outcome, and cardiotocogram baseline variability and acceleration patterns in the growth restricted preterm population. Pediatr Dev Pathol. 2023;26(5):447-457. [Crossref]
6. Orsaria M, Liviero S, Rossetti E, et al. Placental acute inflammation infiltrates and pregnancy outcomes: a retrospective cohort study. Sci Rep. 2021;11(1):24165. [Crossref]
7. Mizrachi Y, Barber E, Torem M, et al. Is there a role for placental histopathology in predicting the recurrence of preterm birth? Arch Gynecol Obstet. 2019;300(4):917-923. [Crossref]
8. Gonen N, Levy M, Kovo M, et al. Placental histopathology and pregnancy outcomes in "early" vs. "late" placental abruption. Reprod Sci. 2021;28(2):351-360. [Crossref]
9. Ditisheim A, Sibai B, Tatevian N. Placental findings in postpartum preeclampsia: a comparative retrospective study. Am J Perinatol. 2020;37(12):1217-1222. [Crossref]
10. Matthews KC, Fox NS, Rebarber A. The association between placental histopathology, fetal growth restriction, and preeclampsia in twin pregnancies. Am J Perinatol. 2021;38(8):784-790. [Crossref]
11. Stevens R, Odell N, Wadee R. The clinical significance of placental histopathological evaluation in the management of high-risk obstetric patients: a cross-sectional retrospective study. South African Journal of Obstetrics and Gynaecology. 2023;29(1):e2109.
12. Strahler L, Horky A, Spahn S, Bahlmann F, Gradhand E. Unveiling clinical relevance: investigating placentas submitted for histological examination and their correlation with clinical indications and histological findings. Life (Basel). 2024;14(8):927. [Crossref]
13. Romero R, Kim YM, Pacora P, et al. The frequency and type of placental histologic lesions in term pregnancies with normal outcome. J Perinat Med. 2018;46(6):613-630. [Crossref]
14. Redline RW, Ravishankar S, Bagby CM, Saab ST, Zarei S. Four major patterns of placental injury: a stepwise guide for understanding and implementing the 2016 Amsterdam consensus. Mod Pathol. 2021;34(6):1074-1092. [Crossref]
15. Boujenah J, Cohen J, Allouche M, et al. Prevalence and association of placental lesions with obstetrical features and outcome: data from French prospective study. AJOG Glob Rep. 2024;4(3):100374. [Crossref]
16. Cetinkaya Demir B, Ozerkan K, Kosan B, Aslan MK, Uncu G. Management and outcomes of 308 placenta accreta spectrum cases at a multidisciplinary tertiary center in Turkey: a 16-year experience. BMC Pregnancy Childbirth. 2026;26(1):307. [Crossref]
17. Kükrer S, Arlıer S, Dilek O, Gülümser Ç. A novel approach for the early prediction and prevention of placenta accreta spectrum and severe peripartum hemorrhage in patients with complete placenta previa: leveraging three-dimensional placental topography, cervical length, and dilatation parameters on magnetic resonance imaging. BMC Pregnancy Childbirth. 2024;24(1):784. [Crossref]
18. Turowski G, Tony Parks W, Arbuckle S, Jacobsen AF, Heazell A. The structure and utility of the placental pathology report. APMIS. 2018;126(7):638-646. [Crossref]