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GUIDELINES

Best practice guidelines for standardised placental pathology reporting: A recommendation by perinatal pathology working group, Chapter of Perinatal and Paediatric Pathology, College of Pathologists, Malaysia

Nur Syahrina RAHIM[1,2], Nur Ayutimasery ABDULLAH[3], Hayati ABDUL RAHMAN[1], Siti Zarqah OMAR[4], Hui Hui SIAH[5], Arni TALIB[5], Geok Chin TAN[6], Lii Jye TAN[7], Yin Ping WONG[6], Khairul Anuar ZAINUN[8], Sellymiah ADZMAN[5]*

[1]Department of Medical Science II, Faculty of Medicine and Health Sciences, Universiti Sains Islam Malaysia, Nilai, Negeri Sembilan, Malaysia; [2]Department of Pathology, Hospital Selayang, Selangor, Malaysia; [3]Department of Forensic Medicine, Hospital Tengku Ampuan Rahimah, Klang, Selangor, Malaysia; [4]Department of Pathology, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Terengganu, Malaysia; [5]Department of Pathology, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia; [6]Department of Pathology, Faculty of Medicine, Universiti Kebangsaan Malaysia, Cheras, Kuala Lumpur, Malaysia; [7]Department of Forensic Medicine, Hospital Raja Permaisuri Bainun, Ipoh, Perak, Malaysia; [8]Department of Forensic Medicine, Hospital Sultan Idris Shah, Serdang, Selangor, Malaysia.

Abstract

Placental pathology provides critical diagnostic, prognostic and clinicopathological insights into maternal-foetal outcomes. While frequently discarded post-delivery, macroscopic and histopathological evaluation can clarify adverse outcomes, including stillbirth, neonatal death, prematurity, foetal growth restriction, hydrops, infection and complicated multiple gestations. This paper outlines a standardized framework for placental examination by covering rationale, indications, specimen handling, sampling and reporting designed for universal implementation across all public, private and academic healthcare facilities. To optimise institutional resources, a phased, indication-based approach is recommended initially, prioritising cases most likely to influence clinical management, parental counselling and future pregnancy care. Pre-analytical optimisation, which includes proper specimen handling, adequate fixation, representative sampling and complete clinical history, is essential to maximize diagnostic yield nationwide. We propose a structured reporting proforma aligned with the current Amsterdam Consensus Classification, including a dedicated section for multiple gestations. This standardises reporting across key pathophysiological domains: maternal vascular malperfusion (MVM), foetal vascular malperfusion (FVM), infections, inflammatory/immune lesions and villous developmental abnormalities. Finally, contextual ancillary investigations are discussed. Ultimately, establishing a unified, cross-sector national placental pathology service holds the potential to significantly improve the audit of adverse perinatal outcomes and elevate maternal-foetal care.

Keywords: placenta, placental examination; perinatal pathology; stillbirth; neonate, forensic placental examination, quality

INTRODUCTION

The placenta, as the critical interface between mother and foetus, holds invaluable diagnostic information that can shed light on maternal, foetal and neonatal health outcomes. Despite its clinical significance, the placenta is often discarded without examination. However, growing evidence underscores that both gross and histopathological evaluation of the placenta can yield essential insights into unexplained foetal or neonatal complications, maternal disorders and perinatal outcomes. Several professional bodies including the College

**Address for correspondence:* Dr Sellymiah binti Adzman, Department of Pathology, Hospital Kuala Lumpur, Jalan Pahang, 50586 Kuala Lumpur. Malaysia. Email: *sellymiah@moh.gov.my*

of American Pathologists (CAP), the Royal College of Pathologists (RCPath, UK), Royal College of Pathologists of Australasia (RCPA), and the American College of Obstetricians and Gynecologists (ACOG) strongly advocate for routine placental examination in specific clinical scenarios, including stillbirth.

Global and local data on the frequency of placental histopathological examination

An estimated 5-50 % of placentas are submitted for histopathological examination worldwide.[1-5] Based on College of American Pathologists (CAP) guidelines, a study conducted by university hospitals in the United States of America (USA) over a period of 6-months showed 48.3% of placentas were sent for histopathological examination.[3] Another survey in the USA however found that about three quarters of the institutions in the USA examined less than 25% of the placentas delivered.[6] At institutions in South Australia and Yemen, the rates reported of placental examination are 17.8% and 4.9% respectively.[4,5] In Australia, a study found only 11.2% of babies with neonatal encephalopathy was placental examination performed.[7] In contrast, placental examination was conducted in 82% of stillbirths in Australia in 2021. In Hospital Canselor Tuanku Muhriz, a teaching hospital in Malaysia, placental examination constitutes about 10% of the total surgical pathology samples that are sent for histopathological examination annually. A similar pattern is observed across local public hospitals, where placental pathology forms an integral part of daily diagnostic practice (unpublished data).

Placental pathology services should be available to all maternity units in Malaysia. Such implementation requires strategic planning in a stepwise manner with commitment from local authorities. This includes development of policies and procedures for placental collection, examination and return of placentas to families if requested, provision of adequate staffing manpower training, infrastructure development, the development of quality indicators and multidisciplinary team review of placental findings to significantly improve the audit of adverse perinatal outcomes and elevate maternal-foetal care.

RATIONALE FOR PLACENTAL EXAMINATION

Placental examination is widely recognised as an essential investigation with substantial clinical value.[8] It provides insight into the maternal and neonatal conditions at the time of delivery, helps explain adverse pregnancy outcomes and guides therapeutic decisions in the perinatal period. Placental findings may carry significant implications for future pregnancies, and their detection may reduce recurrence risk with appropriate intervention and tailored antenatal care.

The following are some rationales for placental examination:

1. Clinical and diagnostic value

Examination of the placenta can help explain acute perinatal events such as hypoxia, meconium aspiration, maternal infection or neonatal sepsis, thereby guiding urgent management of the newborn. For the mother, certain pathological findings may reflect underlying undiagnosed medical conditions including preeclampsia, diabetes mellitus, infection or thrombophilia, all of which warrant further follow-up. Placental examination may provide an explanation for antenatal ultrasound observations. For instance, abnormal Doppler waveforms may correspond to maternal vascular malperfusion or foetal vascular malperfusion in the placenta histologically.[9] Placental examination can also validate or uncover new information of ultrasound findings.[10]

2. Prognostic value

Placental examination also offers valuable prognostic implications. Recurrent lesions such as maternal floor infarction/ massive perivillous fibrin deposition, villitis of unknown aetiology (VUE) or chronic histiocytic intervillositis (CHI) indicate an increased risk in subsequent pregnancies. This can guide preventive strategies, including aspirin therapy, anticoagulation or intensified antenatal surveillance.[11] In addition, certain placental abnormalities may provide clues to possible long term neurodevelopmental risks for the newborn, thus facilitating timely follow-up and early intervention for the affected child.

3. Evaluation of severity of disease

Histological features that provide guidance to the clinicians to decide on appropriate management and determine the severity of disease. For example, the foetal inflammatory response (FIR) syndrome refers to systemic activation of the foetal innate immune system to intrauterine infection. Clinically, it is associated with adverse neonatal outcomes such as early-

onset sepsis, bronchopulmonary dysplasia, intraventricular haemorrhage and longer-term neuro-developmental sequelae.[12] While FIRS can be suggested by serology biochemical markers (elevated foetal/umbilical cord blood IL-6) histological examination of the placenta and umbilical cord is the gold standard as it can determine the severity and extent of disease.[13]

4. Investigation of adverse pregnancy outcomes
In adverse perinatal outcomes, the placenta often holds the key to understanding what went wrong. In stillbirth and neonatal death, placental examination may reveal causes such as infection, maternal vascular malperfusion and foetal vascular malperfusion. In preterm births, findings such as histological chorioamnionitis can explain premature delivery. Similarly, recurrent pregnancy loss may be linked to placental lesions including maternal floor infarction, chronic histiocytic intervillositis or chronic villitis, which then guide further work-up to uncover underlying maternal medical conditions.[14]

5. Medico-legal implications
The placenta often described as a "diary of pregnancy", as it records intrauterine events throughout pregnancy. Its histopathological assessment is therefore a critical component in the investigation of adverse pregnancy outcomes such as cerebral palsy. In the setting of death investigation involving foetal, neonatal and maternal deaths, placental examination should be an integral part of the autopsies to establish conditions that may either contribute or mask a cause of death as well as to understand circumstances of death.[15] A well-documented placental report offers an independent basis for understanding the intrauterine environment, supports accountability in clinical practice and serves as an essential document in legal proceedings or inquiries.[16]

INDICATIONS FOR PLACENTAL EXAMINATION

The criteria for submitting placental tissue for histopathological examination are well defined and may be divided into essential and desirable indications, which can further be classified under maternal, placental and foetal categories (Table 1).

The essential indications represent conditions where examination of the placenta is critical for clinical decision making and documentation. From the maternal perspective, these include cases of maternal death and maternal pyrexia. Placental indications include placental tumours. Foetal indications encompass intrauterine or neonatal death, as well as severe foetal distress, prematurity, foetal growth restriction, foetal hydrops and complicated twin pregnancies, particularly those with discordant outcomes. In such situations, histopathological assessment provides valuable insight into underlying pathophysiological processes and may guide counselling in future pregnancies.

In forensic cases, placental examination is essential for assessing gestational age, identifying evidence of intrauterine disease or hypoxia, clarifying the circumstances surrounding delivery, and supporting maternal–infant linkage through genetic testing. In maternal deaths, it may also identify obstetric complications that contributed to death and provide important documentation for medicolegal investigation.

The desirable indications are situations where placental examination is not mandatory but may yield clinically useful information. Maternal conditions in this category include hypertensive disorders of pregnancy, gestational diabetes, coagulopathies and maternal infections, such as group B streptococcal infection or prolonged (>18 hours) rupture of membranes. Placental examination is warranted in the setting of oligohydramnios, rhesus isoimmunisation and monozygotic twin pregnancies without major complications. In addition, structural abnormalities of the placenta such as abruption, abnormal adherence or abnormal shape as well as umbilical cord anomalies are relevant indications. From the foetal and neonatal perspective, prematurity, congenital malformations and the rare occurrence of neonatal malignancy are important contexts in which placental pathology can provide diagnostic clarity.

Conversely, there are certain situations where placental examination is not routinely required. These include normal pregnancies without complications, polyhydramnios in isolation and postpartum haemorrhage where no placental pathology is suspected. Maternal conditions such as substance abuse, intrahepatic cholestasis of pregnancy, pruritus and viral infections such as Hepatitis B, Hepatitis C and Human Immunodeficiency Virus (HIV), are not indicated. Similarly, the presence of placenta previa does not necessitate histopathological assessment.

Overall, careful selection of cases for placental

TABLE 1: INDICATIONS FOR PLACENTAL EXAMINATION

Essential	**Maternal Indications** – Maternal death – Maternal pyrexia (> 38 degree Celsius) **Placental Indications** Placental mass/ tumour **Foetal Indications** – Stillbirth (antepartum or intrapartum) (> 20 weeks' gestation) – Late miscarriage (16 to 20 weeks' gestation) – Severe foetal distress requiring admission to neonatal unit (with or without meconium-stained liquor) – Prematurity (less than 32+0 weeks' gestation) – foetal growth restriction with birth weight below 10th centile and corresponding abnormal foetal growth curve – foetal hydrops – Complicated twin pregnancies with discordant foetus (> 20% variation in birth weight) Additional indications for forensic cases – Suspected infanticide or abandoned newborn – Fetal and neonatal death being classified as born-before-arrival to healthcare facilities – Unexplained stillbirth or intrauterine death – Maternal death (brought in deceased)
Desirable	**Maternal Indications** – Pre-eclampsia/maternal hypertension – Gestational diabetes – Maternal group B streptococcus – Prolonged rupture of the membrane (> 18 hours) – Rhesus (and other) isoimmunization – Twins or other multiple pregnancy (uncomplicated) – Maternal coagulopathy (e.g. suspected maternal antiphospholipid syndrome, autoimmune disease) – History of placenta with pathology known to recur (e.g. abruption, CHI, massive perivillous fibrin deposition/maternal floor infarction) – Oligohydramnios (AFI less than 5cm) **Placental Indications** – Placental abruption – Morbidly adherent placenta – Abnormal placental shape (if clinically relevant) – Cord abnormalities / lesion/ abnormal cord insertion (e.g. two vessel cord, etc) **Foetal Indications** – Prematurity (32+0 – 36+6 weeks' gestation) – foetal congenital malformation – Neonatal malignancy
Not indicated	**Maternal Indication** – Normal pregnancy – Polyhydramnios – Maternal substance abuse, alcohol misuse, smoking – Cholestasis of pregnancy – Pruritus of pregnancy – Hepatitis B, HIV, etc – Other maternal disease with normal pregnancy outcome **Placental Indication** – Placenta praevia **Neonatal Indication** – Poor Apgar score

examination ensures optimal use of pathology resources while allowing clinicians to obtain critical diagnostic information in scenarios where it is most likely to influence management and counselling.

SPECIMEN HANDLING AND SUBMISSION

A consent form to request placental examination is included in Figure 1. The placenta can be sent to the laboratory either fresh or formalin-fixed, depending on the hospital protocol. The entire placenta should be submitted for more meaningful and accurate interpretation, and better patient management. In situations where the whole placenta cannot be submitted, we recommend taking representative sections from different segments (discussed later). Placentas should not be frozen, as freezing distorts features, making both macroscopic and microscopic examinations difficult. Each placental specimen should be placed in a container labelled with at least two patient identifiers.

Fresh placentas can be kept in a refrigerator at 4 degrees Celsius for up to one week. The advantages of fresh specimens include the availability of material (should the need arise) for culture studies, electron microscopy, cytogenetic, molecular and metabolic tests.

Placentas from pregnancies, should be fixed in formalin prior to handling. If the placenta is placed in a container with formalin for fixation, it is essential to ensure that the container is spacious enough to avoid distorting the appearance and shape of the placenta. The volume of formalin should be adequate to cover the entire placenta, ideally at least three times the volume of the placenta. The placenta should be primary fixed for at least 48 hours. This is particularly important in thick, congested or fused placentas.[17–21]

Placental weight loss can occur during storage, mostly due to the leakage of blood and serum, as well as evaporation. Although minimal in a normal placenta, the weight loss is usually significant for an oedematous placenta with hydrops. Fixing a placenta will increase disc weight from 4-8.5%.[22]

A special request form (Figure 2) is required for placentas submitted for histopathological examination to ensure that essential maternal, foetal and neonatal clinical information is provided. Adequate clinical details support accurate pathological interpretation, meaningful clinicopathological correlation and the delivery of high-quality diagnostic reports. Providing complete information enhances the evaluation of placental lesions in the context of pregnancy outcomes and contributes to standardized reporting and optimal multidisciplinary perinatal care.

Limited / partial placental specimen submission

When the entire placental disc is not received, sampling protocols must be adapted to maximize diagnostic yield and detect major maternal and foetal vascular; and inflammatory lesions.
The general principles are as follows:
Whole placental weight and largest disc diameter be recorded at delivery is recommended. Sampling should aim to include:

1. Umbilical cord and membrane
2. Maternal surface (basal plate/decidua from central disc)
3. Foetal surface (chorionic plate at the site of umbilical cord insertion)
4. Representative villous parenchyma
5. Any grossly abnormal areas

Diagnostic interpretation must acknowledge limited representativeness.

SPECIMEN DISSECTION AND BLOCK SELECTION

A standardized and reproducible approach to gross examination and tissue sampling is essential for placental pathology assessment, ensuring accurate clinicopathological correlation. The guide below integrates recommendations from the Amsterdam Placental Workshop Group, the American College of Obstetricians and Gynecologists (ACOG), the Royal College of Pathologists (RCPath) and the Royal College of Obstetricians and Gynaecologists (RCOG).[16-19] It is applicable to both singleton and multiple gestation placentas and emphasizes phenotypic assessment supported, where appropriate, by targeted ancillary studies. A proposed structured proforma for placental gross examination is provided in Appendix 4 as a practical aid to facilitate systematic, comprehensive and consistent documentation of macroscopic findings. This proforma is intended to be used alongside the gross examination and sampling guidance described below and provides a structured record of the umbilical cord, extraplacental membranes, foetal and maternal surfaces, placental disc measurements, cut-surface findings, focal lesions and representative block selection. Its use is encouraged to promote completeness, reproducibility and consistency

KEIZINAN PEMERIKSAAN URI (PLASENTA)

Saya (nama).....No. K/P……. dengan ini memberi keizinan untuk plasenta ini dihantar bagi tujuan Pemeriksaan Histopatologi Plasenta (HPE) seperti yang telah diterangkan kepada saya oleh Dr…….....

Pemeriksaan ini bertujuan untuk mencari kemungkinan punca berkaitan kehamilan semasa yang mungkin dapat memberi penjelasan serta membantu pengurusan kehamilan pada masa akan datang.

Saya memahami bahawa plasenta akan diproses untuk tujuan pemeriksaan dan tidak dapat dikembalikan dalam keadaan asal, dan sekiranya dipulangkan, ia mungkin telah dipotong. Saya memahami bahawa terdapat kemungkinan tiada kesimpulan muktamad dapat diperoleh daripda pemeriksaan plasenta ini.

Pilihan saya:

☐ Tidak ingin menuntut semula plasenta

☐ Ingin menuntut semula plasenta selepas pemeriksaan

……	……
Tandatangan (Pesakit)	Tandatangan (Suami/Waris)
Nama penuh: ……..……	Nama penuh: ……..……
No. Kad Pengenalan: …….	No. Kad Pengenalan: …….

Saya mengaku bahawa telah menerangkan tujuan pemeriksaan plasenta kepada pesakit.

……..	…….
Tandatangan (Pengamal Perubatan)	Tandatangan (Saksi)
Nama & Jawatan: ……	Nama & Jawatan: ……..……..

FIGURE 1. CONSENT FORM

PATIENTS DATA								
Name:						Ward/clinic:		
IC No:						Hospital:		
MRN:								
**Alternatively attach patient label*								
CLINICAL INFORMATION								
MATERNAL						**FOETAL/NEONATAL**		
Age:		Gravidity/Parity:	G		P	Gestational age:		
Onset of labour:		Spontaneous	Induced		TOP	Livebirth	MSB	FSB
Delivery:		SVD		Assisted: Yes / No		Date/Time of birth:		
		LSCS		Elective / Emergency		Birth weight:		
Intrapartum:		Meconium	Yes / No	MMSL	TMSL	Apgar score: 1’ 5’ 10’		
		Pyrexia	Yes / No	WCC:	$\times 10^9$/L	Last evidence of foetal life (Date):		
		PROM	Yes / No					
		Abruption	Yes / No					
Antenatal:		Booked	Yes / No	TORCHES:		FGR/ foetal scan:		
		HPT/PE	Yes / No					
		Diabetes	Yes / No					
		APH	Yes / No					
		Group B Strep	Yes / No					
Other relevant/previous obstetrics history: *(e.g.: oligohydramnios, infections, smoking, previous miscarriages, IVF, medications, BMI)*						Other relevant history *(e.g. foetal distress/NICU admission, hydrops, foetal abnormalities etc)*:		
Multiple pregnancy/Twin/Triplet:			Specify			Monochorionic/ Dichorionic		
Complication/interventions:								
PLACENTA								
Send as:		Fresh		In formalin		Placental weight (fresh):		
Date collected:			Time collected:					
Requesting Dr: (Sign & stamped)								

FIGURE 2. PLACENTAL EXAMINATION REQUEST FORM

in placental examination and reporting. A diagrammatic representative of the gross examination is provided in figure 4.

Specimen dissection
The placenta should be examined fresh or after adequate fixation in formalin. Any identifiers, tags, clamps or markings used to distinguish placentas or umbilical cords in multiple gestations must be verified prior to dissection.

Membranes
The membranes are identified and examined for completeness, insertion type, opacity, discoloration and lesions. A membrane roll is prepared from the site of rupture to the placental disc margin by gently grasping the free edge with fine forceps and rolling toward the disc so that the rupture site lies centrally within the roll. The membrane insertion forms the outer margin, with the amnion facing inward. This technique is recommended for optimal assessment of acute and chronic inflammatory lesions.[17–20]

Umbilical cord
The umbilical cord is examined for length, diameter, insertion site, vessel number, coiling pattern and macroscopic abnormalities such as knots, strictures, oedema or thrombosis. Standard sampling includes a transverse section from the foetal cut end and an additional section approximately 5 cm from the placental insertion site. Any focal abnormalities are sampled separately.[17,18]

Placental disc
The placental disc is trimmed of excess membranes and umbilical cord, weighed and measured. The disc is serially sectioned at approximately 1 cm intervals perpendicular to the maternal surface to assess parenchymal uniformity and identify focal lesions. After dissection, the specimen is placed in an adequate volume of formalin to ensure complete fixation.[17,21]

External Inspection
Singleton placenta
The foetal surface is examined for colour, sheen and chorionic vessel distribution. The maternal surface is assessed for completeness of cotyledons, disruption and colour changes suggestive of infarction or haemorrhage. Any retroplacental or marginal haematomas are documented with measurements.[17,18]

Multiple gestation placenta
For twin or higher-order gestations, the placenta is identified as either fused or separate. Umbilical cords must be individually identified (e.g. via tagging, clamps or sutures) and assigned to Placenta A, B, etc. The dividing membrane is evaluated for thickness and translucency to determine chorionicity. The examiner must document the vascular equator and the extent of placental fusion for each placental share. Visible vascular anastomoses (arterio-arterial, veno-venous and arterio-venous) must be identified and recorded, as these have significant clinical implications, particularly in monochorionic placenta.[17,21]

Internal Inspection
The cut surfaces of the placental disc(s) are examined systematically. The parenchyma is described as uniform or abnormal. Any focal lesions are characterised by number, size, configuration (solid, cystic, haemorrhagic thrombotic), circumscription, colour, texture and location (central vs peripheral; maternal vs foetal surface). The estimated percentage of placental involvement is recorded. Photographic documentation is recommended when lesions or structural abnormalities are present.[17–20]

In multiple gestations, each placental territory is assessed separately, with attention to discordant lesions between placentas or unequal placental sharing.[17,21]

Histological sampling
Representative sections are submitted for histological examination to allow evaluation of inflammatory, vascular and developmental lesions in accordance with established classification systems.[17,23] Sampling should include gross abnormalities with standard block allocation (see Figure 3).[18,24]

Standard sections (per placenta)
1. Two sections of umbilical cord: one from the foetal cut end and one approximately 5 cm from placental insertion.
2. One membrane roll including the rupture site.
3. One full-thickness section of placental disc parenchyma adjacent to the umbilical cord insertion, including foetal surface vessels.
4. At least two full-thickness blocks of placental parenchyma from the central two-thirds of the disc.
5. Representative sections from any lesions or abnormalities identified, if present.

For dichorionic placenta, the above scheme is repeated for each separate placental disc (e.g. Placenta A, Placenta B), with additional blocks for the dividing membrane and T-junction where applicable.[17–20] Fused placentas require careful preservation of the dividing membranes and foetal-surface vasculature before separation. In triplet or higher-order pregnancies, each cord, membrane compartment and placental territory should be designated consistently as A, B, C and so forth throughout the gross description and tissue sampling.

PROCESSING AND EMBEDDING

Local procedures for processing and embedding tissue samples should be followed. There are no specific requirements for placental tissue. Tissue is usually fixed prior to sectioning with additional fixation often required once samples are in blocks prior to processing to ensure optimal morphology. When sampling the maternal surface to examine spiral arteries in the decidua, specimens may be embedded either 'on edge' or with the decidual face downward, depending on local preference.

MICROSCOPY REPORT

A standardised placental examination proforma, as shown in Figure 3 and Figure 4, is recommended, to guide consistent microscopic reporting of placental specimens. The proforma is structured according to the Amsterdam Consensus Classification which emphasises interpretation of placental lesions according to pathophysiological domains rather than isolated findings.[17]

For twin and higher-order placentas, the proforma includes additional documentation of chorionicity, amnionicity, intertwin membrane characteristics, vascular anastomoses where applicable and differential pathology between co-twins. This structured approach supports diagnostic consistency, improves communication with clinicians and facilitates audit, research and quality assurance.

During microscopic examination, the placenta should be assessed systematically in correlation with gestational age, placental weight, gross findings and relevant clinical history. The aim is to identify clinically significant lesions that may explain the pregnancy outcome, guide neonatal management or inform care in future pregnancies.

Umbilical cord

Microscopic examination begins with the umbilical cord, as cord pathology often reflects foetal vascular compromise or inflammatory response.

The proforma prompts confirmation of vessel number (three-vessel cord or single umbilical artery) and assessment for structural abnormalities such as oedema or reduced Wharton's jelly. Inflammatory changes are evaluated by identifying umbilical vein or artery vasculitis and the FIR is staged and graded according to the Amsterdam Consensus.[17,25]

- Stage reflects anatomical progression, ranging from umbilical phlebitis (stage 1), umbilical arteritis (stage 2) to necrotising funisitis or concentric perivasculitis (stage 3).[17,25]
- Grade reflects severity (mild–moderate versus severe).[17,25]

Additional cord-specific lesions—such as peripheral funisitis, thrombi (acute or organising), vessel wall necrosis, meconium-associated vascular necrosis or embryological remnants—are documented in a structured manner, ensuring reproducible and clinically relevant reporting.

Foetal membrane (Amnion and Chorion)

The foetal membranes are assessed for both acute and chronic inflammatory pathology. The proforma guides evaluation of:

- Reactive amnion
- Pigmented macrophages (with optional iron staining to distinguish meconium from haemorrhage)
- Acute chorionitis and chorioamnionitis.

Maternal inflammatory response is staged and graded according to established criteria. [16,23] The categories are as follows:

- Stage 1 (chorionitis)
- Stage 2 (chorioamnionitis)
- Stage 3 (necrotising chorioamnionitis).

Chronic inflammatory lesions, including chronic chorioamnionitis and amnion nodosum, are recorded separately due to their different clinical implications and recurrence risks.

Membranous decidua

Assessment of the membranous decidua allows evaluation of maternal vascular and inflammatory pathology. The proforma directs the pathologist to identify:

- Decidual arteriopathy including mural hypertrophy, fibrinoid necrosis and acute atherosis.

FIGURE 3. PROFORMA FOR GROSS EXAMINATION OF PLACENTA

Singleton Placenta

Section	Item	Description
General	Specimen received	Received in formalin in a container labelled with patient data and "placenta"
	Components	Placenta with attached umbilical cord and membranes
	Weight (trimmed)	___ g (___ to ___ percentile for GA of ___ weeks)
	Disc dimensions	Diameter ___ to ___ cm; Thickness ___ cm
Forensic (if applicable)	Wrapping material	Newspaper / Plastic bag / Cloth / Other: ___
	Contamination	Cleaned / Contaminated with soil / salt / ___
	Condition	Complete / Fragmented
	State	Fresh / Early decomposed / Moderately decomposed / Advanced decomposition
	Components included	Placental disc / Umbilical cord / Amniotic membrane / Attached with foetus
Umbilical Cord	Insertion	Central / Eccentric (___ cm from edge) / Marginal / Furcate / Interpositional / Velamentous
	Length	___ cm
	Diameter	___ cm to ___ cm
	Vessels	Three
	Thrombosis	Yes / No
	True knots	Yes / No
	Cord twist	Right / Left / Mixed
	Coiling index	___ per cm
	Discolouration	None / Present (specify)
Membranes	Insertion	Marginal / Circumvallate / Circummarginate (___% of circumference)
	Appearance	Translucent / Opaque / Yellow / Green
	Point of rupture	Cannot be assessed / ___ cm from margin
	Other lesions	None / Amnion nodosum / Squamous metaplasia / Retromembranous haemorrhage
Foetal Surface	Appearance	Glistening and normal / Green / Brown
	Chorionic vasculature	No vascular thrombi identified grossly
Maternal Surface	Cotyledons	Intact and complete / Disrupted / Incomplete
	Colour	Normal / Pale / Congested
	Retroplacental haemorrhage	None / Present, measuring ___ × ___ × ___ cm
	Marginal haematoma	None / Present, measuring ___ × ___ × ___ cm
Cut Sections	Overall appearance	Unremarkable / Abnormal
	Infarcts	None / Present (___% of placental volume)
	Increased fibrin	No / Yes – maternal floor / periphery (___%)
	Intervillous thrombi	None / Present (___%; size ___ cm)
Block Allocation	A1	Umbilical cord; foetal cut end and 5 cm from placental insertion
	A2	Membrane roll
	A3	Umbilical cord insertion site, full thickness
	A4–A5	Central two-thirds placental parenchyma, full thickness
	A6 onwards	Additional blocks for abnormalities, if present

Twin/Higher order placenta

The specimen is received fresh/in formalin in a container labelled with patient data and 'placenta'. The specimen consists of a fused/separate twin placenta with attached umbilical cords and membrane.

GENERAL	
Disc:	Fused/separate
Umbilical cords twin orientation/identification:	Tagged/non-tagged or marked or separately identified Placenta A- 1 /2 cord clamps Placenta B- 1/ 2 cord clamps
Dividing membrane:	Absent/Thin and transparent/thick
Vascular equator divides:	Placenta A- ___ % fused disc Placenta B- ___ % fused disc Identified/not present.
Vascular anastomoses:	_____artery-artery (AA)anastomoses _____vein-vein (VV) anastomoses _____artery-vein (AV) anastomoses
Fused disc trimmed weight:	_____ g (__ to __ percentile for GA of __ weeks)
Fused disc dimension:	Diameter __ to __ cm; thickness __ cm
PLACENTA A	
Trimmed weight:	___ g (__ to __ percentile for GA of __ weeks)
Disc dimension:	diameter__ to __ cm; thickness __ cm
Umbilical cord:	
Insertion	Eccentric (__ cm from edge) / Central / Marginal / Furcate / Interpositional / Velamentous
Measurement	Length: __ cm; Diameter: __ cm to __ cm
Cord vessels	Three; Thrombosis: Yes/No; True knots: Yes/No
Cord twist	Right / Left / Mixed (optional)
Coiling index	___ / cm (essential)
Discoloration	
Membranes:	
Insertion	Marginal / Circumvallate / Circummarginate (_% of circumference)
Appearance:	Translucent / Opaque / Yellow / Green
Point of rupture from margin:	Cannot be assessed / __ cm from the edge (optional)
Other lesions:	None / Amnion nodosum / Squamous metaplasia / Retromembranous haemorrhage
Foetal surface of disc:	
Appearance:	Glistening and normal / Green / Brown
Chorionic vasculature:	No vascular thrombi identified grossly.
Maternal surface of disc:	
Appearance:	Intact with completeness of cotyledons / Disrupted / Incomplete,
Colour:	Normal / Pale / Congested
Lesions:	Retroplacental haemorrhage: None / Yes, focal, measuring __ x __ x __ cm Marginal haematoma: None / Yes, measuring __ x __ x __ cm Other:
Cut section of disc:	
Appearance:	Unremarkable with no infarcts or other focal lesions present (estimate % of placental volume)
Lesions	Infarcts present: None / Yes Increased fibrin: No / Yes, towards the maternal floor / towards the periphery of the disc (approximately ___% of placenta) Intervillous thrombi: None / Yes (approximately ___% of placenta, measures size of each thrombus) Other:

PLACENTA B	
Trimmed weight:	_____ g (___ to __ percentile for GA of __ weeks)
Disc dimension:	diameter ___to ___ cm; thickness ___ cm
Umbilical cord:	
Insertion	Eccentric (___ cm from edge) / Central / Marginal / Furcate / Interpositional / Velamentous
Measurement	Length: ____ cm; Diameter: ___ cm to ___ cm
Cord vessels	Three; Thrombosis: Yes/No; True knots: Yes/No
Cord twist	Right / Left / Mixed (optional)
Coiling index	___ / cm (essential)
Discoloration	
Membranes:	
Insertion	Marginal / Circumvallate / Circummarginate (___% of circumference)
Appearance:	Translucent / Opaque / Yellow / Green
Point of rupture from margin:	Cannot be assessed / __- cm from the edge (optional)
Other lesions:	None / Amnion nodosum / Squamous metaplasia / Retromembranous haemorrhage
Foetal surface of disc:	
Appearance:	Glistening and normal / Green / Brown
Chorionic vasculature:	No vascular thrombi identified grossly.
Maternal surface of disc:	
Appearance:	Intact with completeness of cotyledons / Disrupted / Incomplete
Colour:	Normal / Pale / Congested
Lesions:	Retroplacental haemorrhage: None / Yes, focal, measuring ___ x ___ x ___ cm Marginal haematoma: None / Yes, measuring ___ x ___ x __cm Other:
Cut section of disc:	
Appearance:	Unremarkable with no infarcts or other focal lesions present (estimate % of placental volume)
Lesions	Infarcts present: None / Yes Increased fibrin: No / Yes, towards the maternal floor / towards the periphery of the disc (approximately ___% of placenta) Intervillous thrombi: None / Yes (approximately ___% of placenta, measures size of each thrombus) Other:

*NOTE:

Fused disc – Only combine weight and measurement.

Separate disc – Individual weight and measurement. No vascular anastomoses.

SECTIONS SUBMITTED	
A1	Placenta A: Umbilical cord; foetal cut end and 5 cm from placental insertion
A2	Placenta A: Membrane roll
A3	Placenta A: Umbilical cord insertion site, full thickness section
A4-A5	Placenta A: Central two-third of parenchyma, full thickness section
A6	Placenta A: Lesions
A7	Placenta B: Umbilical cord; foetal cut end and 5 cm from placental insertion
A8	Placenta B: Membrane roll
A9	Placenta B: Umbilical cord insertion site, full thickness section
A10-11	Placenta B: Central two-third of parenchyma, full thickness section
A12	Placenta B: Lesions
A13	Dividing membrane & T junction

- Laminar decidual necrosis (band-like (laminar) area of coagulative necrosis located at the choriodecidual interface of the fetal membranes, using a defined threshold of ≥10% of the membrane roll)
- Diffuse decidual leukocytoclastic necrosis

Decidual arteriopathy is a key component of maternal vascular malperfusion. Laminar decidual necrosis and diffuse decidual leukocytoclastic necrosis are associated decidual lesions that may support impaired uteroplacental perfusion but are not, in isolation, diagnostic of maternal vascular malperfusion.

Chorionic plate

The chorionic plate is examined for inflammatory, haemorrhagic and pigment-associated changes. The proforma prompts evaluation of:

- Pigmented macrophages, with distinction between meconium and haemosiderin where possible.
- Acute subchorionitis, chorionitis or chorioamnionitis, with appropriate grading and staging.
- Chronic chorionitis or chorioamnionitis.

Chorionic plate vasculature

Chorionic plate vessels are examined for evidence of FIR and foetal vascular compromise. The proforma standardises assessment of:

- Foetal vasculitis (acute, eosinophilic or T-cell mediated)
- Distribution (single vessel or multifocal)
- FIR stage and grade when umbilical vessels are not involved.

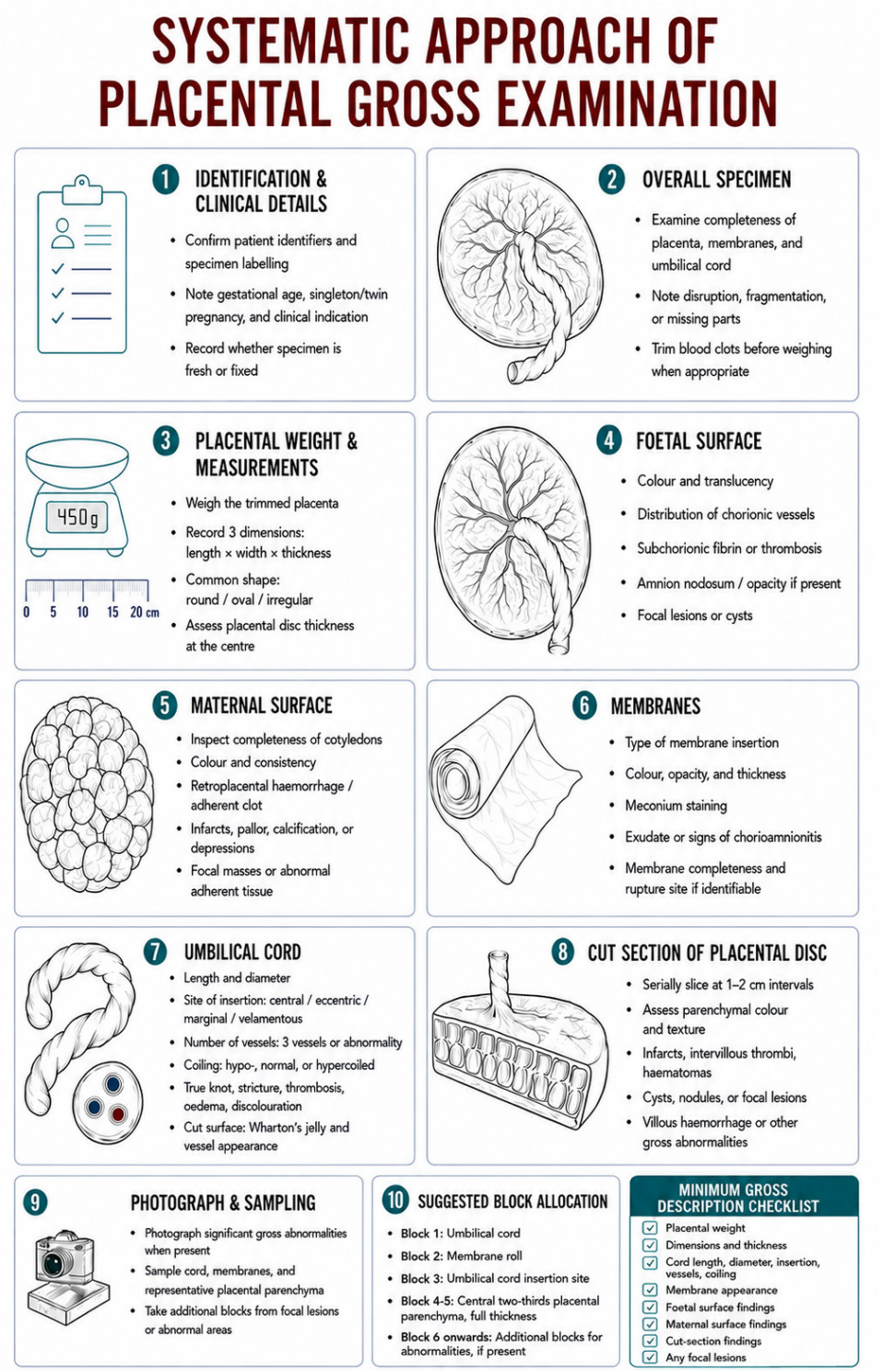
Foetal vascular lesions should also be assessed systematically. These include single or multiple thrombi, which may be acute or organising, occlusive or non-occlusive. Intramural fibrin deposition should be documented and, where possible, described as recent or remote, including associated calcification if present. Marked vascular dilatation, defined as greater than four times the diameter of adjacent vessels, should also be recorded.

These vascular findings, particularly when associated with downstream villous changes such as villous stromal-vascular karyorrhexis or avascular villi, support the diagnosis of foetal vascular malperfusion. Meconium-associated vascular necrosis should be reported separately, as it reflects vascular injury related to prolonged meconium exposure. Any additional abnormality may be documented under “Other”.

Chorionic villi

Systematic assessment of the chorionic villi is central to microscopic placental evaluation. For practical use of the proforma, villous assessment includes both stem and terminal villi. The proforma guides the pathologist to assess whether villous maturation is appropriate for gestational age, accelerated or delayed. Features such as distal villous hypoplasia, infarction, diffuse villous oedema, chorangiosis, chorangiomatosis and intravillous haemorrhage should be recorded where present.

Inflammatory villous lesions should be documented clearly. Acute villitis should be recorded when identified. Chronic villitis should be classified according to its distribution and severity as follows:



This infographic was created with the assistance of generative AI (ChatGPT).

FIGURE 4. SYSTEMIC APPROACH OF PLACENTAL GROSS EXAMINATION

- Low-grade VUE is defined as inflammation involving fewer than 10 contiguous villi per focus, with at least two foci required. It may be further classified as:
  - Focal: at least two foci involving fewer than 10 contiguous villi, identified on one slide.[17]
  - Multifocal: at least two foci involving fewer than 10 contiguous villi, identified on at least two slides.[17]
- High-grade VUE is defined as inflammation involving 10 or more contiguous villi. It may be further classified as:
  - Patchy: at least two foci of involved villi, with at least one focus containing 10 or more contiguous villi, identified on one or more slides.[17]
  - Diffuse: involvement of more than 30% of villi on any slide.[17]

If only a focus of chronic villitis is identified, the lesion may be designated as ungradable. A single focus involving fewer than 10 villi may be reported as ungradable, possible low-grade VUE, whereas a single focus involving 10 or more villi may be reported as ungradable, possible high-grade VUE.[17] Associated features, including obliterative foetal vasculopathy, increased perivillous fibrin and plasma cells, should be noted. If plasma cells are present, CMV immunohistochemistry or PCR may be considered to exclude an infective cause.[26]

Features of foetal vascular injury should also be assessed. Villous stromal-vascular karyorrhexis and avascular villi should be recorded as single or multiple foci and semi-quantified according to the number of involved villi per focus.

Increased nucleated red blood cells should be documented when present, particularly at term, as this may support foetal hypoxic stress.

Dysplastic villous changes, including irregular villous contours, hyperchromatic enlarged stromal cells, abnormal vasculature and syncytiotrophoblast vacuolation, should be recorded separately and interpreted together with the clinical and gross findings. These changes may indicate abnormal villous development and may prompt consideration of genetic, chromosomal, molar-related or other developmental abnormalities, particularly when associated with foetal anomalies, hydrops, intrauterine foetal death or severe unexplained foetal growth restriction.

Other additional significant findings should be recorded in "Other" section.

Intervillous space

The intervillous space should be assessed for inflammatory, thrombo-haemorrhagic and fibrin-related lesions. Observation of focal or multifocal acute intervillositis indicating its extent. Multifocal involvement is perhaps more clinically significant and should be correlated with other inflammatory placental lesions and clinical evidence of infection. Chronic histiocytic intervillositis should be graded according to the estimated percentage of involvement: grade 1, 5–10%; grade 2, 10–50%; and grade 3, more than 50%. This grading system is based on published semiquantitative criteria and has been shown to correlate with adverse perinatal outcome, particularly in high-grade disease.[27] CD68 immunohistochemistry provides diagnostic support by highlighting the histiocytic infiltrate, particularly in subtle cases or in the settings where placental pathology experience is limited. Associated perivillous fibrin deposition should also be documented, as it may reflect more extensive placental involvement, contribute to impaired maternal–foetal exchange, and assist in recognising overlap with significant fibrin-related lesions such as maternal floor infarction or massive perivillous fibrin deposition.[28]

In the absence of chronic histiocytic intervillositis, perivillous fibrin deposition should be assessed according to its extent and distribution. Limited focal deposition may be recorded when present, while encasement of basal villi supports maternal floor infarction. Full-thickness involvement of at least 50% of the sampled parenchyma is in keeping with massive perivillous fibrin deposition. Associated extravillous cytotrophoblast proliferation within fibrin should also be documented, as it may support recognition of a significant perivillous fibrin deposition lesion.

Intervillous thrombus or haemorrhage should be also recorded, including whether nucleated red blood cells are present. This may provide useful clinicopathological information, particularly in cases with suspected foetal bleeding, placental haemorrhage or adverse perinatal outcome. Any additional abnormality within the intervillous space should be described separately.

Perivillous fibrin deposition should be assessed according to its extent and distribution. Focal perivillous fibrin deposition may be recorded when limited, while encasement of basal villi supports maternal floor infarction. Full-thickness involvement of at least 50% of the sampled parenchyma is in keeping with

massive perivillous fibrin deposition. Associated extravillous cytotrophoblast proliferation within fibrin should also be documented, as this may support recognition of a significant fibrin-related placental lesion.

Decidua basalis

The decidua basalis should be examined for maternal vascular, haemorrhagic, inflammatory and implantation-related abnormalities. If no abnormality is identified, the report may state that the maternal spiral arteries are appropriately adapted for pregnancy. If maternal spiral arteries are not represented in the sampled decidua basalis, this should be documented to avoid overinterpretation.

Features of decidual arteriopathy should be specifically assessed, including persistence of endovascular trophoblast, persistence of vascular smooth muscle, mural hypertrophy, fibrinoid necrosis, acute atherosis and thrombosis. These lesions are important components of maternal vascular malperfusion and should be interpreted in correlation with placental weight, villous maturation, infarction, foetal growth restriction, maternal hypertension or abnormal Doppler findings.

Haemorrhagic lesions of the basal plate should also be documented. These include retroplacental haemorrhage, haemosiderin-laden macrophages, microabruption and dissecting intervillous haemorrhage. Their presence may support a diagnosis of placental abruption or related maternal vascular pathology, depending on the clinical and gross findings.

Basal plate myofibres should be recorded when identified. Stage 1 refers to basal plate myofibres with intervening decidua, whereas stage 2 refers to basal plate myofibres without intervening decidua. This finding should be reported descriptively and correlated with the clinical history, particularly when there is concern for abnormal placental adherence.[29]

Inflammatory lesions of the decidua basalis should be documented separately. These include acute deciduitis, plasma cell deciduitis and diffuse decidual leukocytoclastic necrosis. Plasma cell deciduitis may warrant further consideration of infective or chronic inflammatory causes, depending on the morphology and clinical context. Diffuse decidual leukocytoclastic necrosis and increased multinucleated trophoblast in the decidua basalis should also be recorded, as they may support decidual hypoxic injury, abnormal implantation or maternal vascular malperfusion-related pathology. However, these findings should be interpreted together with other placental lesions and clinical features and should not be regarded as diagnostic of maternal vascular malperfusion in isolation.

Twin and higher-order placentas

For twin and higher-order placentas, the proforma requires the same microscopic assessment used for singletons, plus additional documentation of:

- Chorionicity and amnionicity, including the histological characteristics of the dividing membranes.
- Pathological findings within each foetal placental territory.
- Discordant inflammatory, vascular or villous maturation patterns between co-twins.
- Placental sharing and vascular anastomoses in monochorionic placentas, where applicable.

This approach facilitates clinicopathological evaluation of multiple-gestation complications, including twin–twin transfusion syndrome, selective foetal growth restriction and single-twin demise.

Additional and forensic-relevant findings

The proforma includes space for additional relevant findings and allows documentation of forensic or advanced autopsy-related observations, such as:

- Completeness and extent/percentage of tissue involvement.
- Autolytic or putrefactive changes.
- Presence of contaminated materials such as foreign bodies or debris associated with cleansing and burial rituals.

SPECIAL INVESTIGATIONS AND ANCILLARY STUDIES

Routine placental examination is primarily based on gross and microscopic evaluation. However, selected cases may benefit from special stains, immunohistochemistry, microbiological studies or molecular investigations to support diagnosis, exclude differential considerations and provide clinically meaningful information. The use of ancillary investigations should be guided by clinical context, histological findings and multidisciplinary discussion where appropriate. Examples of ancillary studies relevant to placental examination are listed in Table 2.

Electron microscopy (EM) has a limited but well-defined, problem-oriented role in placental pathology. It is not performed routinely; however, in selected cases it provides valuable ultrastructural information that complements light microscopy, immunohistochemistry and molecular studies. This is useful in cases of hydrops foetalis or unexplained neonatal death, where EM may identify abnormal intracellular inclusions suggestive of underlying metabolic or storage disorders.

FINAL REPORT INTERPRETATION

The final diagnostic interpretation should focus on identifying the dominant pathological process i.e. maternal vascular, foetal vascular, inflammatory or developmental and correlating histological findings with clinical information. This integrative approach aligns with the Amsterdam Consensus emphasis on clinically meaningful classification and supports improved communication with obstetric and neonatal teams.

Examples:

1. Features consistent with severe maternal vascular malperfusion, including:
   - Small placenta (<10th centile for gestational age) with increased fetoplacental weight ratio (>90th centile)
   - Multiple placental infarctions involving approximately 50% of the disc
   - Distal villous hypoplasia
   - Decidual arteriopathy (fibrinoid necrosis with acute atherosis)
2. Acute chorioamnionitis with:
   - Maternal inflammatory response (MIR): Stage 2, Grade 2
   - Foetal inflammatory response (FIR): Stage 1, Grade 1

IMPLEMENTATION CONSIDERATIONS

The standardization and implementation of placental examination services should be established as a nationwide standard of care spanning all tiers of the healthcare ecosystem, including Ministry of Health (MOH) facilities, university/academic hospitals and private healthcare institutions. While placental histology yields critical diagnostic and prognostic data, universal submission of all specimens is unfeasible due to systemic variations in human resources, laboratory infrastructure and specialized perinatal pathology expertise across sectors. Consequently, a phased, indication-based framework is mandated to optimize resource allocation, standardize clinical governance and improve maternal and neonatal outcomes nationwide.

During the initial implementation phase, all participating public, private and academic healthcare institutions should prioritize histopathological placental examination for cases meeting specific clinical indications as described in Table 1. This approach allows pathology services to focus on cases in which placental findings are most likely to provide clinically meaningful information and contribute to maternal and/ or neonatal management.

A standardised request form (Figure 2) should be introduced to ensure that relevant clinical information is provided at the time of specimen submission as discussed above. Adequate clinical information is important because interpretation of placental pathology requires clinicopathological correlation.

Standard operating procedures for specimen handling, fixation, gross examination, tissue sampling and reporting should be developed and adapted according to local laboratory capacity (Figures 4 and 5). The use of a minimum sampling protocol and structured reporting proforma is encouraged to improve consistency, reduce inter-observer variation and support meaningful communication between pathologists, obstetricians, neonatologists and other clinicians. Complex cases may be referred to centres with perinatal pathology expertise, particularly cases involving recurrent pregnancy loss, unexplained stillbirth, complicated twin pregnancy, suspected metabolic disease, unusual inflammatory lesions or medico-legal concerns.

Training and competency development are essential for successful implementation. Pathologists, Medical Officers, Scientific Officers and laboratory staff should receive training in placental grossing, block selection, recognition of common placental lesions and use of standard terminology. Regular multidisciplinary discussion between pathology, obstetrics and neonatology teams should be implemented to improve understanding of the clinical relevance of placental findings.

Implementation should also include audit and quality assurance activities. Hospitals should monitor the number of placentas submitted, indications for submission, turnaround time, adequacy of clinical information, concordance

TABLE 1: INDICATIONS FOR PLACENTAL EXAMINATION

Essential	**Maternal Indications** – Maternal death – Maternal pyrexia (> 38 degree Celsius) **Placental Indications** Placental mass/ tumour **Foetal Indications** – Stillbirth (antepartum or intrapartum) (> 20 weeks' gestation) – Late miscarriage (16 to 20 weeks' gestation) – Severe foetal distress requiring admission to neonatal unit (with or without meconium-stained liquor) – Prematurity (less than 32+0 weeks' gestation) – foetal growth restriction with birth weight below 10th centile and corresponding abnormal foetal growth curve – foetal hydrops – Complicated twin pregnancies with discordant foetus (> 20% variation in birth weight) Additional indications for forensic cases – Suspected infanticide or abandoned newborn – Fetal and neonatal death being classified as born-before-arrival to healthcare facilities – Unexplained stillbirth or intrauterine death – Maternal death (brought in deceased)
Desirable	**Maternal Indications** – Pre-eclampsia/maternal hypertension – Gestational diabetes – Maternal group B streptococcus – Prolonged rupture of the membrane (> 18 hours) – Rhesus (and other) isoimmunization – Twins or other multiple pregnancy (uncomplicated) – Maternal coagulopathy (e.g. suspected maternal antiphospholipid syndrome, autoimmune disease) – History of placenta with pathology known to recur (e.g. abruption, CHI, massive perivillous fibrin deposition/maternal floor infarction) – Oligohydramnios (AFI less than 5cm) **Placental Indications** – Placental abruption – Morbidly adherent placenta – Abnormal placental shape (if clinically relevant) – Cord abnormalities / lesion/ abnormal cord insertion (e.g. two vessel cord, etc) **Foetal Indications** – Prematurity (32+0 – 36+6 weeks' gestation) – foetal congenital malformation – Neonatal malignancy
Not indicated	**Maternal Indication** – Normal pregnancy – Polyhydramnios – Maternal substance abuse, alcohol misuse, smoking – Cholestasis of pregnancy – Pruritus of pregnancy – Hepatitis B, HIV, etc – Other maternal disease with normal pregnancy outcome **Placental Indication** – Placenta praevia **Neonatal Indication** – Poor Apgar score

TABLE 2: List of ancillary studies in placental examination

Histological Finding (H&E)	Ancillary Study	Indication	Amsterdam Consensus Alignment
Chronic villitis	PAS, GMS	Exclude fungal infection	Villitis: infectious vs non-infectious
	CMV IHC ± PCR	Chronic villitis, FGR, IUFD	Required before VUE diagnosis
Acute chorioamnionitis	Gram stain ± culture	Severe or atypical inflammation	Maternal inflammatory response
Chronic histiocytic intervillositis	CD68	Confirm intervillous histiocytes	Chronic inflammatory lesion
Plasma cell deciduitis	CD138	Confirm plasma cell–rich inflammation	Chronic deciduitis
Extensive intervillous fibrin	Martius Scarlet Blue (MSB)	Suspected MVM	Maternal vascular malperfusion
Granulomatous inflammation	Ziehl–Neelsen ± PCR	Suspected TB / atypical infection	Infectious villitis/ intervillositis
Culture-negative inflammation	Broad-range PCR	Persistent uncertainty in relation to infection or inflammation	Excluding infection
Unexplained IUFD / severe FGR	Placental microarray, FISH	Exclude genetic cause	Non-placental causes assessment

with submission criteria and the proportion of cases with clinically significant findings. These data can be used to refine local protocols, identify training needs and support future expansion of the service.

With appropriate planning, training and resource allocation, stepwise implementation of placental examination services can improve the investigation of adverse pregnancy outcomes, support counselling for future pregnancies and strengthen perinatal care across all healthcare facilities in Malaysia (Figure 6).

SUMMARY

The placenta is not merely a discarded organ post-delivery, but it is a diagnostic goldmine. A national policy mandating routine placental examination across all public, private and university healthcare institutions, in cases with clinical indications, will significantly enhance maternal and neonatal healthcare delivery nationwide. This initiative aligns with global best practices in reducing maternal and infant morbidity and mortality.

GLOSSARY:

Maternal death: A death occurring during pregnancy, childbirth and puerperium (also known as pregnancy related death) is defined as the death of a woman while pregnant or within 42 days of termination of pregnancy irrespective of the cause of death (obstetric or non-obstetric).

Direct death: Direct maternal deaths are fatalities resulting from obstetric complications during pregnancy, labour or the puerperium (up to 42 days post-pregnancy). These deaths are directly caused by interventions, omissions, incorrect treatment or the chain of events resulting from these complications.

Indirect death: Indirect maternal deaths are fatalities resulting from pre-existing diseases or conditions developed during pregnancy (such as cardiac, renal or psychiatric disorders) that were not from direct obstetric causes but were aggravated by the physiological stress of pregnancy.

Fortuitous death: A death from other causes not related to or influenced by pregnancy, which happens to occur during pregnancy or the puerperium (postpartum period).

Infanticide: According to Malaysia Penal Code section 309a: When any woman by any wilful act or omission causes the death of her newly-born child, but at the time of the act or omission

FIGURE 5. PROFORMA FOR MICROSCOPIC REPORTING

Singleton Placenta:

Umbilical cord:
- 3 vessels (2 arteries and 1 vein), no abnormalities
- Single umbilical artery
- Oedema of Wharton jelly
- Decreased Wharton jelly
- Umbilical vein/artery vasculitis
    - Foetal inflammatory response stage: 1 (umbilical phlebitis) / 2 (umbilical arteritis) / 3 (necrotising funisitis or concentric perivasculitis)
    - Foetal inflammatory response grade: 1 (mild-moderate) / 2 (severe)
- Peripheral funisitis, GMS stain positive / negative
- Umbilical vein thrombus (acute / organising / with vessel wall necrosis)
- Umbilical artery thrombus (acute / organising / with vessel wall necrosis)
- Meconium-associated vascular necrosis
- Remnants present (Allantoic duct remnants/ Omphalomesenteric duct remnants)
- Other:

Foetal membranes (amnion and chorion):
- No abnormalities
- Reactive amnion
- Pigmented macrophages: Absent / rare / increased; in amnion / in chorion - optional iron (haemosiderin) stain.
- Acute chorionitis / acute chorioamnionitis
    - Maternal inflammatory response stage: 1 (acute chorionitis) / 2 (acute chorioamnionitis) / 3 (necrotising acute chorioamnionitis).
    - Maternal inflammatory response grade: 1 (mild-moderate) / 2 (severe)
- Chronic chorioamnionitis (lymphohistiocytic +/- plasma cells)
- Amnion nodosum
- Other:

Membranous decidua:
- No abnormalities
- Acute inflammation: Mild / Moderate/Severe
- Chronic inflammation: Plasma cell deciduitis / granulomatous deciduitis / perivascular lymphoid infiltrate
- Laminar decidual necrosis / diffuse decidual leukocytoclastic necrosis
- Decidual arteriopathy: Mural hypertrophy / fibrinoid necrosis /acute atherosis
- Other:

Chorionic plate:
- No abnormalities
- Pigmented macrophages: Consistent with meconium / consistent with haemosiderin (iron stain positive/negative)
- Acute subchorionitis / chorioamnionitis

    - Maternal inflammatory response stage 1 (if limited to subchorionitis) / 2 (acute chorioamnionitis) / 3 (necrotising acute chorioamnionitis).
    - Maternal inflammatory response grade: 1 (mild-moderate) / 2 (severe)
- Chronic chorionitis / chorioamnionitis
- Other:

Chorionic plate vasculature:
- No abnormalities
- Foetal vasculitis: Acute / eosinophilic / T cell; single vessel / multifocal
    - Foetal inflammatory response stage 1 (chorionic plate vasculitis, if umbilical artery is not involved)
    - Foetal inflammatory response grade: 1 (mild-moderate) / 2 (severe)
- Thrombus: Single / multiple / acute / organising / occlusive / non-occlusive / intramural fibrin deposition (recent/remote with calcification)
- Marked dilation of chorionic vessel (> 4x diameter of adjacent vessels)
- Meconium-associated vascular necrosis
- Other:

Chorionic villi:
- Villous maturity appropriate for gestational age / accelerated villous maturity / delayed villous maturity
- Distal villous hypoplasia
- Infarct: Single / multiple
- Diffuse villous oedema: Yes / No
- Chorangiosis/Chorangiomatosis: Focal / multifocal
- Intravillous haemorrhage
- Acute villitis
- Chronic villitis: Focal / multifocal / patchy / diffuse
- Low grade (<10 villi/focus) / high grade (10 or more villi/focus) / with associated obliterative foetal vasculopathy / with increased perivillous fibrin / with plasma cells (if present, CMV immunostain or PCR study positive / negative)
- Villous stromal/vascular karyorrhexis: Single focus / multiple foci; <5/focus / 5-15/focus / >15/focus
- Avascular villi: Single focus / multiple foci; <5/focus / 5-15/focus / or >15/focus
- Increased nucleated red blood cells (NRBC) (>10/40x field at term)
- Dysplastic villous changes: Irregular villous contours / hyperchromatic, enlarged stromal cells / abnormal vasculature / syncytiotrophoblast vacuolation
- Other:

Intervillous space:
- Acute intervillositis: Focal / multifocal
- Chronic histiocytic intervillositis: Grade 1 (5-10%) / 2 (10-50%) / 3 (>50%), with perivillous fibrin
- Excessive perivillous fibrin: Focal / encasing basal villi (maternal floor infarction) / full thickness involvement of at least 50% of parenchyma on slide (massive perivillous fibrin deposition) / with associated extravillous cytotrophoblast proliferation in fibrin
- Intervillous thrombus (with / or without nucleated RBC) / haemorrhage

– Other:

Decidua basalis:
– No abnormalities. Maternal spiral arteries appropriately adapted for pregnancy / or no maternal spiral arteries sampled in decidua basalis
– Decidual arteriopathy (persistent endovascular trophoblasts / persistent of smooth muscle / mural hypertrophy / fibrinoid necrosis / acute atherosis / thrombosis)
– Retroplacental haemorrhage, ± haemosiderin-laden macrophages / microabruption / dissecting intervillous haemorrhage
– Basal plate myofibers, stage 1 (decidua present) or stage 2 (decidua absent)
– Diffuse decidual leukocytoclastic necrosis
– Acute deciduitis
– Plasma cell deciduitis
– Increased multinucleated trophoblast in decidua basalis
– Other:

Twin/Higher Order Placenta

– Apply the singleton microscopic proforma separately to each labelled umbilical cord and each corresponding placental disc or vascular territory, designated consistently as Twin A, Twin B, Twin C and so forth.
– Record chorionicity and amnionicity, correlating the microscopic findings with the macroscopy, clinical or ultrasonographic information.
– Examine each dividing membrane separately, for example A–B and B–C in a triplet placenta.

Notes: This proforma is adapted and modified from Diagnostic Pathology: Placenta, 3rd Edition.[28]

she had not fully recovered from the effect of giving birth to such child and by reason thereof the balance of her mind was then disturbed, she shall, notwithstanding that the circumstances were such that but for this section the offence would have amounted to murder, be guilty of the offence of infanticide.

Concealment of birth by secret disposal of dead body: According to Malaysia Penal Code section 318: Whoever by secretly burying or otherwise disposing of the dead body of a child, whether such child dies before or after or during its birth, intentionally conceals or endeavours to conceal the birth of such child shall be punished with imprisonment for a term which may extend to two years or with fine or with both.

List of expert panels of placental working group
Prof. Emeritus Dr. Hayati Abdul Rahman
Prof. Dr. Tan Geok Chin
Assoc. Prof. Dr Nur Syahrina Rahim
Assoc. Prof. Dr Wong Yin Ping
Dr. Arni Talib
Dr. Khairul Anuar Zainun
Dr. Nur Ayutimasery Abdullah
Dr. Sellymiah Adzman
Dr. Siah Hui Hui
Dr. Siti Zarqah Omar
Dr. Tan Lii Jye

List of stakeholders
Prof. Emeritus Dato Dr. Cheong Soon Keng
President
College of Pathologists, AMM

Dr Salmi Abdullah
Consultant Anatomic Pathologist & Head of Anatomic Pathology Services
Hospital Selayang

Dr Norina Kassim
Consultant Anatomic Pathologist
Hospital Selayang

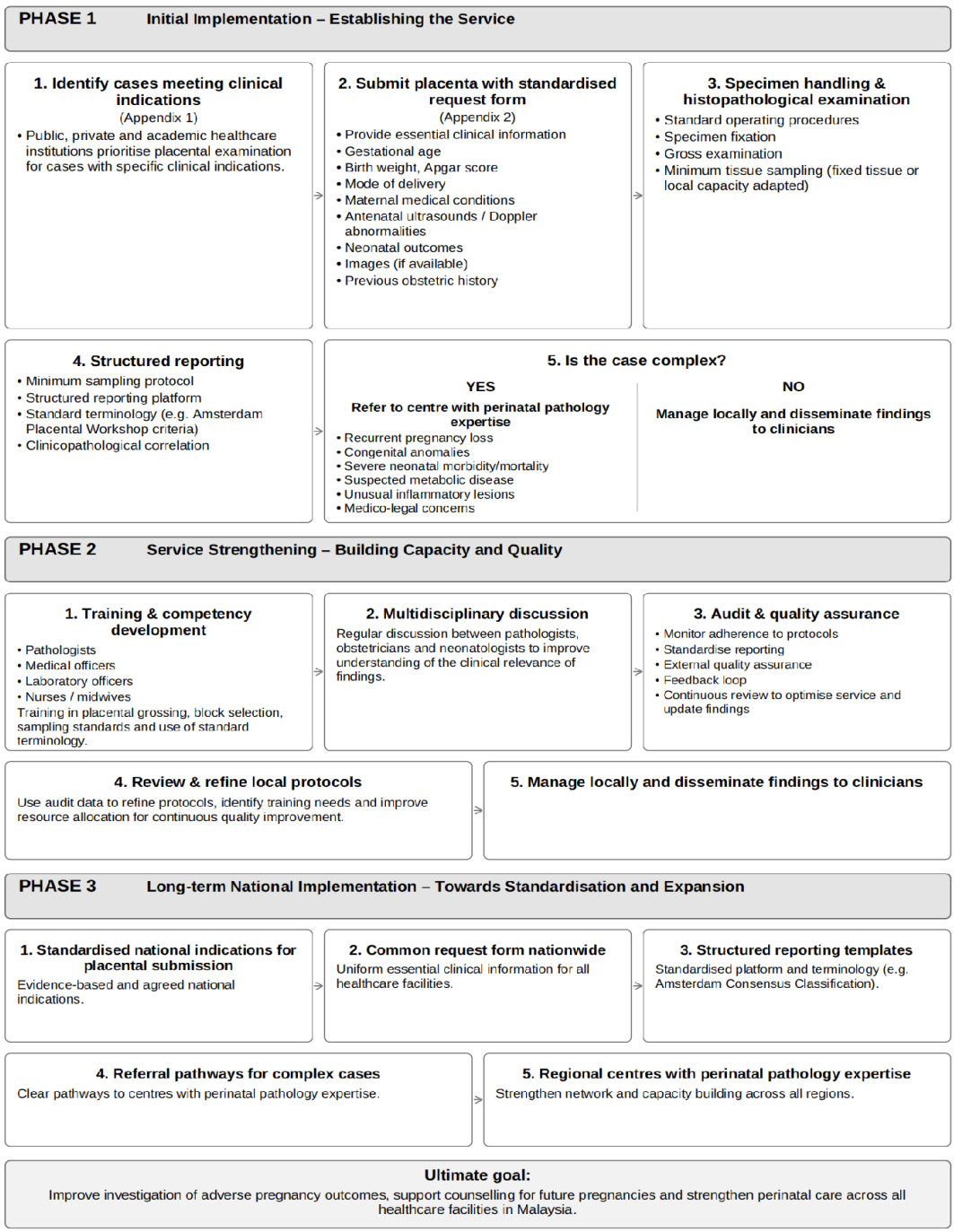


FIGURE 6. STEPWISE IMPLEMENTATION OF PLACENTAL EXAMINATION

Dr Farah Inaz Syed Abdullah
Consultant Paediatrician and Neonatologist,
Hospital Tunku Azizah Kuala Lumpur.

Dr Roziah Husin
Maternal Foetal Medicine Consultant,
Hospital Tengku Ampuan Afzan, Kuantan.

Dr Ang Ee Lee
Paediatrician and Neonatologist,
Hospital Tengku Ampuan Rahimah, Klang.

Dr Emily Christine D' Silva
Maternal Foetal Medicine Specialist,
Hospital Ampang

Dr Sarah Awang Dahlan
Ketua Penolong Pengarah Kanan
Sektor Kesihatan Ibu
Bahagian Pembangunan Kesihatan Keluarga KKM

Dr Hafizah Md Salleh
Family Physician
Klinik Kesihatan Beranang

Matron Kartina Mohammad Manan
Deputy Director, Nursing Division
Pejabat Pengarah Kejururawatan KKM

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DISCLAIMER

This document is intended to provide guidance and promote standardised practice. It should be used as a reference only and does not carry legal force or replace applicable legislation, regulatory requirements or institutional policies. The diagrams provided in the figures 4 and 6 were generated using AI ChatGPT.

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