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Best Practice No 178

Examination of the human placenta

B Hargitai, T Marton, P M Cox

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The human placenta is an underexamined organ. The clinical indications for placental examination have no gold standards. There is also inconsistency in the histological reports and the quality is variable. There is great interobserver variability concerning the different entities. Although there are still grey areas in clinicopathological associations, a few mainstream observations have now been clarified. The histopathological examination and diagnosis of the placenta may provide crucial information. It is possible to highlight treatable maternal conditions and identify placental or fetal conditions that can be recurrent or inherited. To achieve optimal benefit from placental reports, it is essential to standardise the method of placenta examination. This article summarises the clinical indications for placenta referral and the most common acknowledged clinicopathological correlations.

See end of article for authors' affiliations

Correspondence to: Dr T Marton, Department of Histopathology, Birmingham Women's Hospital, Metchley Park Road, Edgbaston, Birmingham B15 2TG, UK; Tamas.Marton@bwhct.nhs.uk

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According to the guidelines of the Royal College of Pathology, samples of diagnostic value removed from the human body should be histologically examined, with only a few exceptions.[1] One of the exceptions is the healthy human placenta, but even with valid indications the human placenta is one of the most underexamined specimens.[2] There is also evidence that the quality of reports on the investigation of the placenta is very variable.[3] According to a recent study, there is a considerable discrepancy rate in the diagnosis of placental disease, and it is common for general surgical pathologists not to recognise placental lesions that may have clinical relevance.[4] In this best practice article, we summarise those circumstances in which it is recommended that the placenta should be examined, the minimum criteria of sampling, and the acknowledged clinicopathological correlations.

> "It is common for general surgical pathologists not to recognise placental lesions that may have clinical relevance"

Lesions of the placenta often reflect or explain the condition in which the baby was born and some have clinicopathological implications. However, in most cases, there is no clinicopathological relevance to a placental examination, such as in the case of normal pregnancy and delivery.

CLINICAL APPROACH

What do we expect from the pathological examination?

The placenta forms a functional unit between the mother and the fetus. Therefore, any pathological event that concerns the mother or the fetus will influence the normal function of the placenta, occasionally resulting in morphological change. Severe abnormalities of the placenta may lead to adverse fetal outcome. However, placental lesions are not necessarily the cause of unfavourable outcome, and some structural changes may be the consequences of poor fetal condition. The placenta is an easily available specimen and the costs of a routine pathological examination are moderate.

The benefits that can be expected from the examination include revealing the aetiology of stillbirth, preterm delivery, intrauterine growth restriction (IUGR), and neurodevelopmental impairment. It may be possible to decide whether the pathological condition that endangered the well being of the fetus was an acute or a chronic process.[5 6]

In the case of twin pregnancies, the type of twinning can be identified and pathological aspects of twin pregnancy (for example, twin-to-twin transfusion syndrome) can be studied.

Conditions with the risk of recurrence can be recognised, resulting in adequate treatment and preventive measures during subsequent pregnancies.

Placental examination may have medicolegal aspects—for example, concerning the aetiology of longterm neurodevelopmental sequelae or the approximate timing of an intrauterine death.[7 8]

Which placentas should be examined?

There are different approaches to the examination of the placenta. It would produce a pointless increase in workload if all placentas, including those from normal pregnancies and normal deliveries resulting in a healthy infant, were examined in a routine pathology laboratory setting.

Because it is the decision of the midwife and/or obstetrician which placentas to send to the

Abbreviations: AAA, arterio–arterial anastomosis; AVA, arterio–venous anastomosis; IUGR, intrauterine growth restriction; VVA, veno–venous anastomosis

Fetal conditions
IUGR (birthweight below 2.5 kg or 3rd centile)
Prematurity (less than 37 weeks of gestation)
Abruption
Fetal hydrops
Fetal abnormality, chromosome aberration
Stillbirth
Severe fetal distress requiring admission to NNU
Rhesus (and other) isoimmunisation
Morbidity adherant placenta
Twins/other multiple pregnancy (uncomplicated)
Abnormal placental shape (if clinically relevant, including placentile tumours) or other postnatally diagnosed disorders of the placenta (haematoma, too big or too small placenta, infarction, discolouration of membranes)
2 vessel cord, etc
Premature rupture of membranes (more than 36 hours)
Diseases of the neonate with possible IU origin
Infection (pneumonia, sepsis within 72 hours)
Neurological signs
Maternal diseases that might have consequences in the neonate
Maternal pyrexia and maternal group B streptococcus
Pre-eclampsia, hypertension
Severe diabetes, including gestational diabetes, maternal thrombopathies-thrombophilias, other metabolic disease, autoimmune disease, tumour, storage disease, etc

Figure 1 Indications for pathological referral of the placenta. IU, intrauterine; IUGR, intrauterine growth restriction; NNU, neonatal unit.

pathology department, a clinically oriented approach (fig 1) may be used to define the indications for histopathological examination.

Referral is not indicated for:

- cholestasis of pregnancy
- hepatitis B, human immunodeficiency virus, etc
- other maternal disease with normal pregnancy outcome
- normal pregnancy
- placenta praevia
- postpartum haemorrhage.

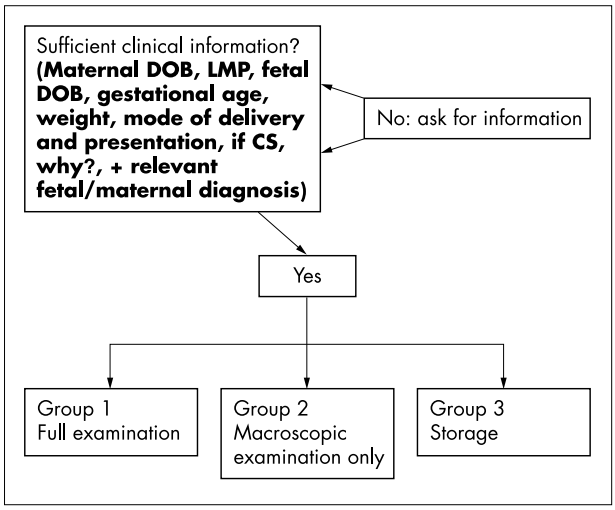


Figure 2 An algorithm to sort the placentas in view of further action. CS, caesarian section; DOB, date of birth; LMP, last menstrual period.

A RATIONAL SORTING OF THE REFERRED PLACENTAS

Figure 2 is an algorithm for selecting which of the referred placentas should be subjected to further study. Figure 3 contains a recommendation for sampling the placentas based on the clinical context.

NORMAL VARIANTS

As mentioned above, many features can be judged only in the clinicopathological context. This is partly because of the loose correlation between some histological changes and clinical symptoms, and partly because of the large reserve capacity of the placenta.

To record the macroscopic appearance of the placenta we recommend the use of a worksheet as shown in fig 4. This proforma can be useful to describe normal placentas; however, each abnormality should be documented individually.

Umbilical cord

The normal length of the umbilical cord at term varies between 40 and 70 cm and cords of less than 32 cm are considered to be short and those more than 100 cm are considered long. The importance of length and coiling should be treated cautiously, because the proportion of the umbilical cord received in pathology laboratories varies and is thus not reliable. Umbilical cords normally show a degree of coiling. The normal coil index is said to be one coil/5 cm.[9 10] The normal cord contains three vessels, and this has to be assessed at least 5 cm from the placental insertion.[11] False knots may be the site of thrombosis, or rarely bleeding, but most often they have no clinical relevance.

Embryonic remnants of the vitelline duct and urachus are normal findings. Cysts may arise from these vestigial remnants. It may be necessary to differentiate the embryonic remnants of the cord from teratomas and haemangiomas.[12]

Group 1: full examination	Recommended minimum samples
Group 1A Rhesus isoimmunisation with admission to the NNU Any IU anaemia requiring IU transfusion Morbidity adherent placenta	**Group 1A** Umbilical cord × 2 Membrane roll × 1 Representative lesions (if any) Paracentral (i.e. not marginal), grossly normal placenta including fetal and maternal surface × 3
Group 1B Maternal pyrexia Prematurity (< 34 weeks and not PET/IUGR) Severe fetal distress, admission to the NNU Neonatal infection	**Group 1B** Umbilical cord × 3 (× 2 if < 10cm) Membrane roll × 1 Representative lesions (if any) Paracentral (i.e. not marginal), grossly normal placenta including fetal and maternal surface × 3
Group 1C IUGR Prematurity (< 34 weeks) due to PET/IUGR Severe PET Abruption	**Group 1C** Umbilical cord × 2 Membrane roll × 1 Representative lesions (if any) Paracentral (i.e. not marginal), grossly normal placenta including fetal and maternal surface × 3 Spiral artery blocks
Group 1D Hydrops Fetal anomaly Stillbirth	**Group 1D** As 1A, with special investigations if necessary

Group 2: macroscopic examination only and afterwards storage (unfixed, 2 weeks, 4°C, urgent examination on clinical request)
Abnormal shape
Single umbilical artery
Uncomplicated twin pregnancy

Group 3: storage (unfixed, 2 weeks, 4°C, urgent examination on clinical request)
PROM
Prematurity, 34–36 weeks
Gestational diabetes
Rhesus negative mother
Maternal group B streptococcus
Uncomplicated pre-eclampsia

Figure 3 Indications to examine the placenta, with examples of the minimum blocks. IU, intrauterine; IUGR, intrauterine growth restriction; NNU, neonatal unit; PET, pre-eclamptic toxaemia; PROM, premature rupture of membranes.

Extraplacental membranes and the fetal surface[12–14]

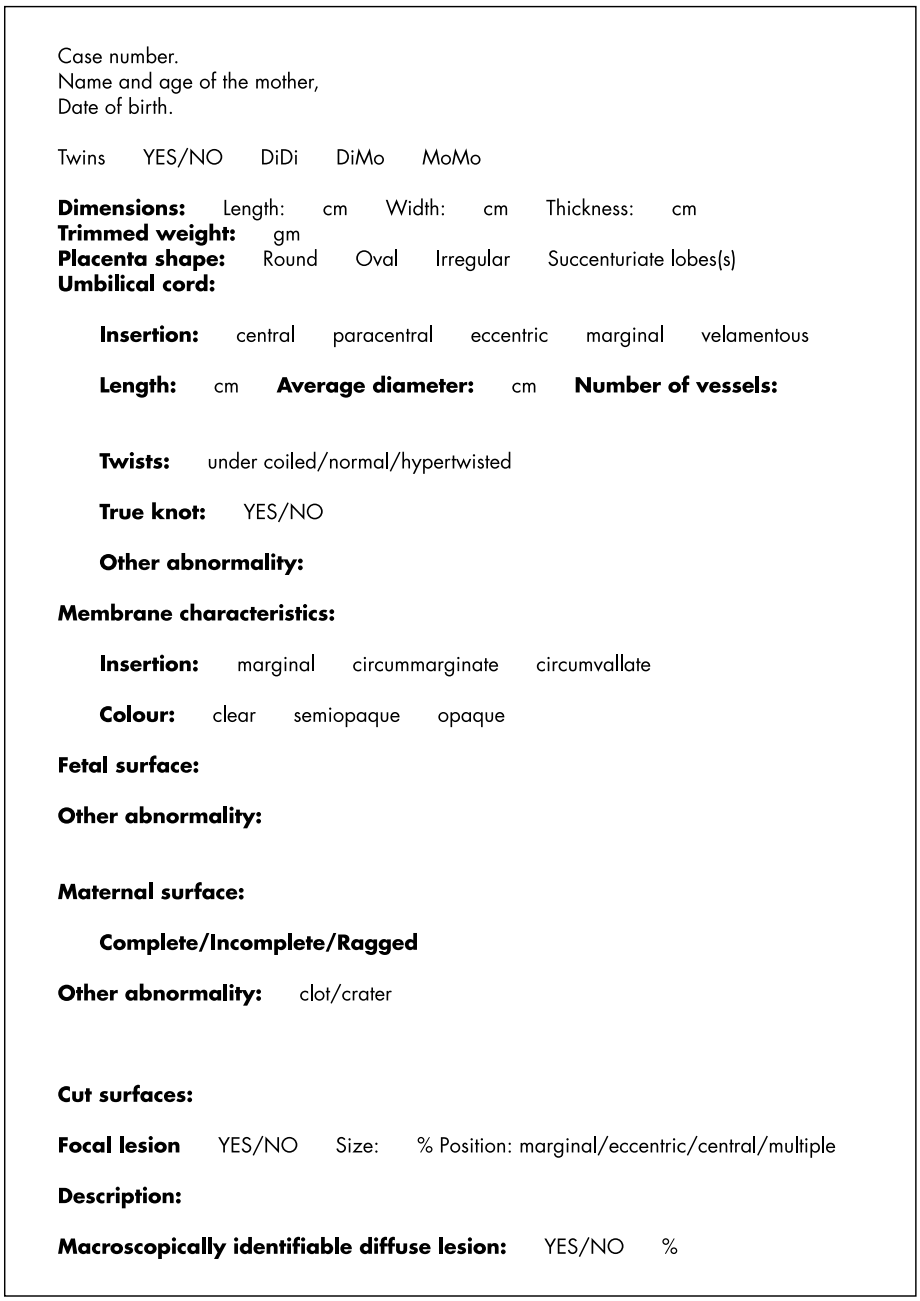
The importance of circummarginate and circumvallate placentas is uncertain, although an association with IUGR and acute and chronic maternal haemorrhage has been proposed in circumvallate placentas. Amnion nodosum (granular grey/white nodules, consisting of keratin and vernix) are a sign of oligo/anhydramnios, but squamous metaplasia of the amnion is a normal feature.

A small amount of subchorionic fibrin deposition (Langhans fibrinoid) is not pathological, because it accumulates from eddying of the intervillous flow.

Placenta

A low placental weight is found in ''small for gestational age'' placentas. Normal values of fetal to placenta weight ratio change during the course of gestation, and vary between 1 at 14 weeks and 7.23 at term. Hydrops or congestion can result in a high placental weight, but the placenta weight can vary to some degree (a table of normal values can be found in Benirschke and Kaufmann[13]). Deviation from the round or oval shape such as an irregularly shaped, bilobed, or multilobed placenta can be attributed to disturbed implantation or uterine abnormalities, but it can be assessed only in the clinicopathological context. Increased calcification has been mentioned in association with maternal smoking and high socio-economic status, but the feature itself has no clinical relevance.

Minor perivillous fibrin deposition is almost always present in term placentas. This is of no clinical relevance if marginal, or if it does not exceed 10% of the villous tissue. A range of values is found in the literature with regard to the amount of the villous tissue loss required to define whether infarction or


Case number.
Name and age of the mother,
Date of birth.

Twins YES/NO DiDi DiMo MoMo

Dimensions: Length: cm Width: cm Thickness: cm
Trimmed weight: gm
Placenta shape: Round Oval Irregular Succenturiate lobes(s)
Umbilical cord:

Insertion: central paracentral eccentric marginal velamentous

Length: cm **Average diameter:** cm **Number of vessels:**

Twists: under coiled/normal/hypertwisted

True knot: YES/NO

Other abnormality:

Membrane characteristics:

Insertion: marginal circummarginate circumvallate

Colour: clear semiopaque opaque

Fetal surface:

Other abnormality:

Maternal surface:

Complete/Incomplete/Ragged

Other abnormality: clot/crater

Cut surfaces:

Focal lesion YES/NO Size: % Position: marginal/eccentric/central/multiple

Description:

Macroscopically identifiable diffuse lesion: YES/NO %

Figure 4 Worksheet for macroscopic examination. DiDi, dichorionic diamniotic; DiMo, diamniotic monochorionic; MoMo, monochorionic monoamniotic.

perivillous fibrin deposition is ''extensive'' or relevant—that is, large enough to account for adverse fetal outcome. The reported percentage of minimal villous tissue loss ranges from 10% to 30% in the case of significant placental infarcts and 20% to 30% in perivillous fibrin deposition. In general, there is no clinical relevance if the lesion is single, marginal, and/or involves less than approximately 5% of the villous tissue. Obviously, the functional reserve capacity of the placenta depends not only on the quantity, but also on the quality of the uninvolved tissue and the original size of the placenta. In the case of a small placenta, a smaller amount of parenchymal loss can lead to fetal demise or morbidity.

X cell islands (extravillous cytotrophoblast islands, X-cell proliferation) are considered to be a normal feature.

The origin of septal cysts is unknown. They are reported to occur more frequently in oedematous placentas, but are of no clinical relevance.[13]

Examination of twin placentas

Twin placentas should be labelled after the delivery to identify which cord belongs to which fetus. The examination of placentas from multiple gestations should establish the chorionicity of the sample and whether there are signs of twin-to-twin transfusion syndrome. Separated twin placentas have to be examined in the same way as those of singletons. Fused placentas can be monochorionic or dichorionic. The dividing membrane should be studied to identify the chorionicity. The dividing membrane in monochorionic pregnancy is thin and translucent (with no

Table 1 The clinical relevance of placental abnormalities

Disorder	Clinicopathological correlation
Umbilical cord	
Short cord (less than 40 cm)	High fetal and neonatal mortality rates and increased frequency of neurological abnormality
Long cord (longer than 70 cm)[19]	Maternal factors: systemic diseases, delivery complications, increased maternal age Fetal factors: non-reassuring fetal status, respiratory distress, vertex presentation, cord entanglement, male sex, increased birth weight Gross placental features: increased placental weight, overcoiled cord, true knots, congestion, cord prolapse causing fetal distress
Marginal cord insertion	IUGR, still birth, neonatal death, premature birth, low birth weight
Velamentous insertion	Fetal haemorrhage, fetal death, low birth weight, premature birth, maternal smoking, advanced maternal age
Overcoiling or undercoiling of the cord[9 10]	Fetal demise fetal intolerance to labour, IUGR, chorioamnionitis
True knot	If tight, associated with perinatal mortality of 10% and umbilical vessel thrombosis
Single umbilical artery[20 21]	Single umbilical artery is associated with fetal malformation chromosome aberration in 25–50%, with IUGR and increased perinatal mortality in normally formed infants
Thrombosis of umbilical cord vessels[12 13 22 23]	Thromboembolic spread to placental or fetal vessels. The consequences of cord vessel thrombosis for the fetus may be wide. Severe sequelae such as fetal death, cerebral palsy and IUGR have been described, but delivery of a healthy, live neonate may also occur
Umbilical cord vessel vasculitis and funisitis[12]	Umbilical cord vessel vasculitis and funisitis are associated with cord vessel thrombosis, preterm delivery, amniotic infection, vasospasm of cord vessels
Necrotising funisitis[12 13 24 25]	It is often associated with premature rupture of the membranes, preterm labour, IUGR, intrauterine death. Usually seen with acute chorioamnionitis. Candida, streptococci, herpes, and syphilis are reported to play a role in the pathogenesis of necrotising funisitis. Mostly associated with chorioamnionitis NOS
Membranes	
Acute chorioamnionitis (including ''subchorial intervillositis'')[26–34]	Strong association with premature rupture of membranes and preterm delivery. Fetal intrauterine infection may occur. Maternal fever and tachycardia are described, but may be asymptotic. Recently, chorioamnionitis has been implicated as a risk factor for periventricular leucomalacia and cerebral palsy
Chronic chorioamnionitis[12 13 35]	Association with premature rupture of membranes, preterm delivery, and prolonged rupture of membranes has been observed. It has been described in herpes virus infection
Amnion epithelial vacuolisation[12 13 36]	Cell degeneration and necrosis of amniotic epithelial cells can be seen in normal and abnormal pregnancies and the evaluation of these alterations might be fairly uncertain because of artefact effects. Small, lipid containing vacuoles in the cytoplasm are the feature, strongly associated with gastroschisis
Pigmented macrophages, meconium staining[12 13 32 37]	The presence of meconium staining is not necessarily associated with adverse fetal outcome. Meconium staining indicates the danger of meconium aspiration and with other histological signs of fetal distress may underline the diagnosis. Vasospasm of cord vessels and fetal chorionic vessels is reported as a consequence of meconium exposure
Deciduitis, acute deciduitis, chronic decidual necrosis[13]	Acute deciduitis in the decidua capsularis is often associated with ascending infiltrations of the placental membranes, and may be unimportant in isolation. Severe, necrotising, acute deciduitis can be found in placentas with retroplacental haematoma. Chronic deciduitis with scattered infiltration may represent a physiological condition of maternal lymphocyte response
Placenta	
Low placental weight, below 10th centile for gestational age[38–40]	IUGR, pre-eclampsia, increased intervillous fibrin deposition, villitis of unknown origin, and trisomy
High placental weight [38 39]	Maternal diabetes mellitus, maternal or fetal anaemia, fetal hydrops; may also be seen in congenital syphilis, Beckwith-Wiedemann syndrome, congenital nephrotic syndrome
Thin placenta (placenta annulare or placenta membranacea)[38 41 42]	Average thickness less than 2 cm, placenta with large membranous area. Risk of maternal bleeding, placenta praevia, placenta accreta. Often premature delivery occurs. Possibly more frequent in IUGR
Placental haemorrhage	
Retroplacental haematoma[12 13]	Large retroplacental haematomas can cause extensive infarction involving a sufficient proportion of the villous tissue to cause fetal hypoxia or lead to perinatal death. Early separation is called abruptio placentae, and can be lethal if extensive. The AFP concentration may be raised if an old haematoma
Subchorionic haematoma (massive subchorial thrombosis, Breus's mole)[12 13]	This is a normal finding when patchy, focal, or diffuse. However, subchorionic thromboses of large size have been reported in association with abortion, premature delivery, and live-born infants also
Placenta praevia	Associated with high risk of third trimester bleeding
Placenta accreta, increta, and percreta	Potentially life threatening clinical conditions, causing uterine rupture and massive postpartum haemorrhage, or leading to caesarean section if prenatally diagnosed. It is often an indication of postpartum hysterectomy because of excessive bleeding. To make the pathological diagnosis of a placenta accreta, examination of the entire uterus is necessary
Placental chorionic villi and intervillous space abnormalities	
Syncytial knots[12 13]	Increased numbers of syncytial knots occur in: pre-eclampsia, hypertension, diabetes mellitus, maternal anaemia, pregnancy at high altitude, thick section (artefact). A correlation between increased syncytial knotting and fetal hypoxia has not been reported. An excessive increase of syncytial knotting may result from reduced fetal perfusion and placental hypoxia or can be the sign of accelerated maturation if the duration of pregnancy was less than 40 weeks
Infarct (acute or old)[13]	No clinical relevance if it is single, marginal, and/or involves less than about 5% of the villous tissue
Extensive placental infarction[12 13 31 43]	Involving more than 10% of villous tissue: fetal hypoxia, IUGR, stillbirth, pregnancy induced hypertension, abruptio placentae, neurological abnormalities
Nucleated RBC[13]	Raised numbers may occur in many causes of chronic hypoxia, IUGR, stillbirth, acute fetal blood loss, maternal diabetes, and erythroblastosis fetalis
Villous basal membrane thickening[12]	Pre-eclampsia, essential hypertension, diabetes mellitus
VSM deficiency[12 13]	An increase of VSM is described in pregnancies at high altitude, pre-eclampsia, maternal heart failure, and maternal anaemia. VSM deficiency was reported in pre-eclampsia, materno–fetal rhesus incompatibility, maternal diabetes, low birth weight and stillbirths
Villous stromal fibrosis and sclerosis[12 13]	Extensive stromal fibrosis occurs in terminal villous deficiency, in IUGR, and in avascular villi as a result of stem vessel thrombosis, and in CMV infection
Villous oedema[12 13 31]	Placentas from pregnancies with hydrops fetalis may show a combination of immaturity and oedema. Villous oedema occurs in infections (syphilis, CMV, toxoplasma), in cases of fetal hydrops, and in hydatidiform moles. It is correlated with neurological impairment and cerebral palsy. May be normal if focal
Dysmaturity/immaturity[12 44]	A failure of villous maturation was found to be associated with fetal hypoxia, IUGR, maternal diabetes, and materno–fetal rhesus incompatibility. Failure of maturation can lead to intrauterine death

Table 1 Continued

Disorder	Clinicopathological correlation
Advanced maturation/maturitas praecox[12 13]	Accelerated maturation can be seen in prematurely delivered placentas in pre-eclampsia. It is considered to be an ischaemic feature
Mesenchymal dysplasia[45 46]	Associated with Beckwith-Wiedeman syndrome
Perivillous fibrin[32], extensive perivillous fibrin deposition	When more than 20–30% of the villous tissue and functional placenta is involved it is associated with IUGR and fetal death. In these cases often 70–80% of the villous population is enveloped by fibrin. The maternal serum AFP values is raised, sometimes extremely so
Maternal floor infarct[12 13 39 47], Gitter infarct	Massive basal plate perivillous fibrin deposition is termed ''maternal floor infarct'' and is associated with high mortality and IUGR. Massive perivillous fibrin deposition in a netlike pattern is the ''Gitter infarct''. Neither of these is an infarct. Massive perivillous fibrin can recur (18%) and is associated with IUGR and fetal death
Villitis	
Acute villitis[12 13]	Clinical consequences depend on the type of the pathogenic agent. Acute villitis is usually associated with severe maternal infection, preterm delivery and might lead to intrauterine infection and IUD
Chronic villitis: basal, parenchymal, granulomatous, and VUE[12 13 32]	Chronic villitis: more often of unknown aetiology (VUE) than known. Fetal infections causing chronic villitis: CMV, toxoplasma, connatal syphilis. Chronic villitis is associated with: IUGR and/or stillbirth VUE: IUGR, preterm birth, is often recurrent!
Chronic histiocytic intervillositis, (chronic perivillositis)[48–50]	Associated with raised maternal serum AFP, recurrent abortion, IUGR, preterm delivery. Malaria infection should be excluded
Abnormalities of the fetal vessels	
Fetal chorionic vessel thrombosis and avascular villi; stem vessel thrombosis (single/or multiple); recanalisation of chorionic vessels[8 31 32 51 52]	Extensive avascular villi as a result of fetal vessel thrombosis was reported in association with stillbirth, IUGR, maternal and fetal coagulopathy, and fetal thromboembolic disease leading to cerebral palsy
Intimal fibrin cushion[37 53]	Neonatal asphyxia, association with disseminated capillary thrombi of fetal vessels. The severity of the fetal consequences depends more on the accompanying vascular lesions, mainly on fetal vessel thrombosis
HEV and haemorrhagic villitis[12 13 54–56]	HEV was reported to be a postmortem artefact and was doubted as being a specific disease entity. HEV was found to be associated with meconium staining and postmaturity. Earlier reports revealed an association with stillbirth, IUGR, neurological disability, maternal hypertension. Can occur in live births, associated with perinatal complications, fetal distress, IUGR. Interlesional relations exist between thrombotic, chronic inflammatory, and chronic vaso-occlusive lesions
Chorangiosis[13 57 58]	Perinatal death, congenital malformation, and cerebral palsy were found to be associated with chorangiosis as a response to low grade tissue hypoxia. Although others have supported this observation, it is still unclear how chronic hypoxia results in increased vascularisation. The importance of this alteration needs further investigation
Abnormalities of the maternal vessels[13]	
Failure of physiological adaptation of maternal vessels, uteroplacental vessel fibrinoid necrosis,[12] acute atherosis, uteroplacental vessel thrombosis[59]	Uteroplacental or decidual arteriopathy is closely related to pregnancy induced hypertension, maternal essential hypertension, and pre-eclampsia, and results in fetal complications such as IUGR, SGA, and stillbirth. It is associated with APA, SLE, and thrombophilia
Haemorrhages of the placenta	
Intervillous haemorrhage and intervillous thrombus	Most often intervillous haemorrhage is related to a maternal vessel lesion and is of maternal origin. Its consequence can be fetal compromise or death depending on the functional placenta parenchyma loss and the rest of the unaffected placenta
Kline's haemorrhage[12 13]	In some cases, intervillous haemorrhage and thrombus is a sign of fetal bleeding into the maternal circulation, as described by Kline. Only a few of these alterations lead to a large amount of fetal blood loss and stillbirth or severe anaemia followed by ischaemic lesions of parenchymal organs
Twin placenta, chorionicity	It is important to know the type of twinning because the twin-to-twin transfusion syndrome is associated with diamniotic monochorionic placentas
Angiomas	
Angioma of the placenta (chorangioma)[12 13 58]	Large lesions often lead to cardiac failure, hydrops, and death of the fetus. Transplacental bleeding and fetomaternal transfusion have been also described, leading to anaemia. Chorangioma was reported to be associated with pre-eclampsia, multiple gestation, premature delivery, fetal thrombocytopenia, and fetal angioma (Kasabach-Merritt syndrome)
Angioma in the cord[60 61]	The lesion can be associated with raised AFP, fetal DIC, and fetal hydrops; fetal death has been described too

AFP, α fetoprotein; APA, anti-phospholipid syndrome; CMV, cytomegalovirus; DIC, disseminated intravascular coagulopathy; HEV, haemorrhagic endovasculitis; IUD, intrauterine death; IUGR, intrauterine growth restriction; NOS, not otherwise specified; RBC, red blood cell; SGA, small for gestational age; SLE, systemic lupus erythematosus; VSM, vasculo–syncytial membrane; VUE, villitis of unknown aetiology.

chorionic layer), whereas that of a dichorionic placenta is thicker, because it contains two chorionic layers between the amniotic sacs. The dividing membrane can be sampled as a membrane roll or in ''T section'' form. A properly oriented T section is the best sample to prove chorionicity. Fetal vessel anastomoses and inter-twin blood transfusion occur normally in monochorionic placentas. Imbalance in the blood flow may lead to acute or chronic twin-to-twin transfusion. Acute transfusion occurs either during labour or after the death of either of the twins, and frequently results in severe neurological damage or the death of the co-twin. Chronic twin-to-twin transfusion manifests as discordant fetal growth, oligohydramnios in the donor, and polyhydramnios in the recipient fetus, and is often associated with poor fetal outcome.[15] Arterio–arterial anastomoses (AAA) may have a protective role against chronic twin-to-twin transfusion syndrome, but may be the route of acute blood loss after compromise or the death of one twin. Veno–venous anastomoses (VVA) are associated with poor outcome.[16] The anatomical background of chronic twin-to-twin transfusion syndrome seems to be a unidirectional arteriovenosus shunt between the donor and the recipient twin. Injection studies can be performed in fresh specimens to clarify the type of the anastomosis.[17] In fixed placentas, arteries may be identified by the fact that they are always superficial to the veins. Arterio–venous anastomosis (AVA) may be identified by the presence of an impaired vessel from one twin feeding an area drained by the co-twin. In monochorionic diamniotic placentas, it may be useful to record the sites of insertion and distance between the cord insertions, the relative size of the placental territories serving each twin, the number and minimum diameter of superficial anastomoses (AAA/VVA), and the number and direction of deep anastomoses (AVA).

''A properly oriented T section is the best sample to prove chorionicity''

It is recommended that fused dichorionic placentas should be separated. Evidence of a vanished twin might be found in singleton or twin placentas. This varies in appearance from an amorphous, fibrotic plaque to a well formed fetus papyraceous. Histological and *x* ray examinations are helpful to identify calcification.[18]

RECOGNISED CLINICOPATHOLOGICAL CORRELATIONS

Table 1 summarises the clinical relevance of placental abnormalities.

CONCLUSION

We recommend that relevant placentas are discussed regularly at perinatal mortality or morbidity meetings. This could reveal new clinicopathological correlations, would increase appreciation of the profession, and would serve team building and communication between the different medical teams. We have presented an algorithm of indications for placental examination and discussed the methods of histopathological examination. Common placental lesions with their clinicopathological correlation are reviewed. Our intent is to outline the acknowledged entities with their clinical consequences. Often, the clinicopathological correlation appears to be strong, significant, and well documented. In other instances, lesions may have a tendency to occur with clinical conditions and in the rest of the cases there is only an anecdotal association. A major problem with the literature related to the placenta is that most of it has been produced based solely on abnormal placentas, so that for many features it is not clear what is pathologically abnormal and what is a normal variant. Basic studies are necessary to analyse normal placentas statistically and to identify the normal variants of histological lesions during the course of pregnancy.

It is also apparent that because function depends on the reserve capacity of the placenta, several findings can be judged only in the clinical context: the importance of a particular lesion depends on its localisation and on the extent of the lesion (the proportion of the placenta involved and the size and the condition of the uninvolved placenta). Some features can be within normal limits in term placentas, whereas earlier in pregnancy they may be pathological. In addition, the assessment of the lesions is even more complex because several pathological conditions can coexist in the same placenta.

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Authors' affiliations

B Hargitai, No 1 Department of Obstetrics and Gynecology, Semmelweis University Budapest, Baross u. 27, 1088 Budapest, Hungary
T Marton, Department of Histopathology, Birmingham Women's Hospital, Metchley Park Road, Edgbaston, Birmingham B15 2TG, UK
P M Cox, Department of Histopathology, Birmingham Women's Hospital

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