Acta Scientific Women's Health (ISSN: 2582-3205)

Volume 8 Issue 5 May 2026

Research Article

Toward Healthier Babies: Pathologic Findings in Circummarginate Placentas with Clinical Implications

Sofie Banjasová[1], Octavia Butler[1] and Paul H Hartel[2,3]*

[1]*Department of Life Sciences, Atlantic Technological University, Ireland*

[2]*Department of Pathology, University Hospital Sligo, Ireland*

[3]*Department of Medicine, West Virginia University School of Medicine, USA*

***Corresponding Author:** Paul Hartel, Professor, Department of Pathology, University Hospital Sligo, Ireland.

DOI: 10.31080/ASWH.2026.08.0747

Received: April 14, 2026

Published: April 29, 2026

Abstract

Gross and histopathologic descriptions of circummarginate placentas compared with normal membrane placentas is virtually non-existent in pathology literature. We reviewed 50 circummarginate placentas from term pregnancies in the SUH catchment area from 2016-2026 and compared gross and histopathologic findings with 50 placentas with normal membrane insertion. Gross and histologic findings with the greatest difference in prevalence compared with controls included those associated with maternal vascular malperfusion such as increased peri villous fibrin deposition, distal villous hypoplasia, infarction, chronic chorionitis, retroplacental haematoma and villous oedema. Circummarginate placentas did not meaningfully differ from controls regarding prevalence of acute funisitis, acute chorioamnionitis, meconium staining, increased villous capillaries, intervillous thrombi, calcifications or increased syncytial knots. As circummarginate placental membranes are visible on ultrasound beginning at around 20 weeks, increased monitoring of pregnancies from this point may help identify, monitor and/or prevent clinical conditions associated with maternal vascular malperfusion such as pre-eclampsia, gestational diabetes and hypertension, growth restriction and autoimmune diseases/ thrombophilias.

Keywords: Circummarginate; Placenta; Maternal Vascular Malperfusion; Pre-Eclampsia

Introduction

Literature on circummarginate placentas is extremely sparse [1] and suggests they may be present in up to 25% of placentas, but have been regarded as clinically unimportant [2]. Gross and histopathologic descriptions of circummarginate placentas compared with normal membrane placentas are virtually non-existent in pathology literature. We reviewed 50 circummarginate placentas from term pregnancies in the SUH catchment area from 2016-2026 and compared gross and histopathologic findings with 50 placentas with normal membrane insertion. Clinical audit of circummarginate placentas in SUH catchment area was performed and anonymised data including maternal age and placental gross and histopathologic findings were reviewed for 50 circummarginate and 50 control placentas with normal membrane insertion. Data was obtained from electronic gross and histologic pathology reports. Maternal age and placental weight did not differ between placentas with circummarginate or normal membrane insertion. Gross and histologic findings with the greatest difference in prevalence compared with controls were increased peri villous fibrin deposition, distal villous hypoplasia,

infarction, chronic chorionitis, retroplacental haematoma and villous oedema. Circummarginate placentas did not meaningfully differ from controls regarding prevalence of acute funisitis, acute chorioamnionitis, meconium staining, increased villous capillaries, intervillous thrombi, calcifications or increased syncytial knots.

Methods

For 2026 Annual National Histopathology Quality Improvement Conference, clinical audit of circummarginate placentas in SUH catchment area was performed. Anonymised data including maternal age and placental gross and histopathologic findings were reviewed for 50 circummarginate and 50 control placentas with normal membrane insertion. Data was obtained from electronic gross and histologic pathology reports. Initial gross and histologic evaluation were performed by consultant histopathologists with a minimum of four haematoxylin and eosin-stained slides on each case including umbilical cord, foetal membrane, foetal and maternal surface and any lesions present.

Results and Discussion

Results are shown in Table 1. Circummarginate placentas (Figure 1) were seen in 3% of total placentas over a 10-year period (105/3640), excluding twin placentas. Maternal age ranged from 23-42 years (m = 33) and placental weights ranged from 321 to 761 grams (m = 465). There were two single artery umbilical cords. Gross and histologic findings with the greatest difference in prevalence compared with controls are shown in Table 1 and include those associated with maternal vascular malperfusion. Increased peri villous fibrin deposition was seen in 14 cases, distal villous hypoplasia in 12 (Figure 2), infarction in 12, chronic chorionitis in eight, retroplacental haematoma in seven and villous oedema in four. Circummarginate placentas did not meaningfully differ from controls regarding prevalence of acute funisitis, acute chorioamnionitis, meconium staining, increased villous capillaries, intervillous thrombi, calcifications or increased syncytial knots.

Membrane status	Maternal age, m years	Placental weight, m grams	Increased peri villous fibrin	Distal villous hypoplasia	Chronic chorionitis	Infarctions	Retroplacental haematoma	Increased villous oedema
Circummarginate	33	465	14/50 (28%)	12/50 (24%)	8/50 (16%)	7/50 (14%)	7/50 (14%)	4/50 (8%)
Normal	34	476	2/50 (5%)	0/50 (0%)	4/50 (8%)	2/50 (5%)	4/50 (8%)	0/50 (0%)

Table 1: Gross and histologic findings in circummarginate versus control placentas.

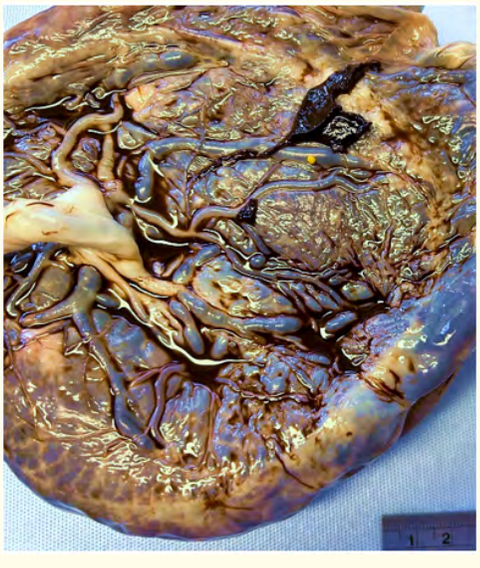

Figure 1: Circummarginate membranes insert inwards from the placental edge rather than at the margin, resulting in a flat transition to the villous chorion.

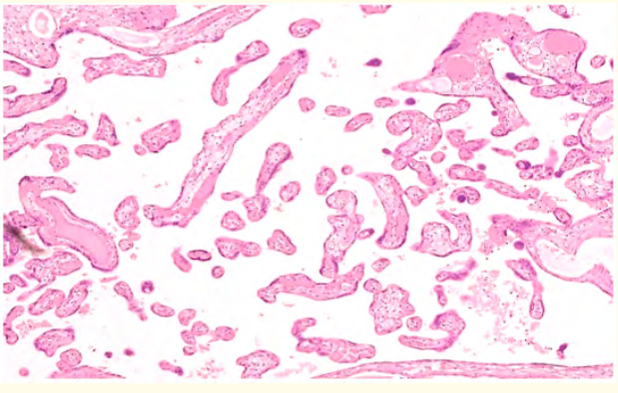

Figure 2: Distal villous hypoplasia, a manifestation of maternal vascular malperfusion.

Research into gross and histologic findings associated with circummarginate placentas is sparse in the pathology literature [1,2]. We reviewed 50 circummarginate placentas to document gross and histologic findings that may differ from placentas with normal membrane insertion and highlight any clinical utility these may have. The greatest difference in prevalence of pathologic findings we found in circummarginate placentas compared with controls were primarily those associated with maternal vascular malperfusion. Maternal vascular malperfusion can lead to serious medical consequences such as foetal growth restriction, pre-eclampsia or foetal death [3]. Maternal vascular malperfusion stems from inadequate spiral artery remodelling, spiral artery pathology or high-velocity malperfusion that may be detrimental to early placentation and placental function in later pregnancy [4-6]. Placental features considered to be indicative of maternal vascular malperfusion include both gross and microscopic findings [7]. Gross findings include infarction and retroplacental haemorrhage. Infarcts can be suspected by their generally pyramidal shape and usual involvement of the basal parenchyma or maternal floor of the placenta. Any infarction seen in a preterm placenta or at term greater than 5% of nonperipheral infarction should be described, while marginal infarcts may have less meaning. Placental abruption is a clinical diagnosis and the correct descriptor for the pathologic finding is retroplacental hemorrhage or retroplacental hematoma. Grossly, there is blood accumulation on the maternal surface, with congestion and/or hemorrhage of overlying parenchyma. Microscopically, there is blood accumulation beneath the decidua, compression of overlying intervillous space, villous crowding, congestion, and/or intravillous hemorrhage. Microscopic findings of maternal vascular malperfusion include abnormalities of villous development, such as distal villous hypoplasia. The latter is defined as a reduction in number of terminal villi, with remaining villi appearing small, thin, long, and poorly branched with an increased and widened intervillous space.

One of the most serious manifestations of maternal vascular malperfusion is preeclampsia. Preeclampsia can be defined as a multisystemic disorder occurring in 3–8% of pregnancies; it accounts for 16–18% of maternal and 40% of foetal and neonatal deaths. Preeclampsia is characterized by the new onset of hypertension (systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg), which occurs after the 20th week of gestation in pregnancy [8]. Preeclampsia is a placenta-driven, systemic disorder centred on vascular dysfunction. There is an evolving understanding of preeclampsia from a primarily hypertensive condition, [8] to a two-stage placental model, [9] and complex multi-system disease with long-term consequences [10,11].

Preeclampsia generally has two stages: early-onset preeclampsia and late-onset preeclampsia, with early-onset preeclampsia being the more severe of the two [8,9]. The condition originates from abnormal placental development, particularly impaired trophoblast differentiation and defective remodelling of uterine arteries, leading to maternal vascular malperfusion including reduced uteroplacental blood flow, placental hypoxia/ischemia, as well as the release of biologically active factors into the maternal circulation. The first stage involves poor placental development, and the second stage is the maternal response to this poor development. Preeclampsia can cause maternal headaches, vision loss, eclampsia or stroke, as well as the development of HELLP syndrome (haemolysis, elevated liver enzymes, and low platelet count), kidney damage or failure, congestive heart failure, [10,11] and pulmonary oedema. At the placental level, it disrupts key processes such as trophoblast proliferation, invasion, migration, angiogenesis, and vascular integrity, largely mediated through dysregulation of signalling pathways and microRNA expression.

The placenta typically becomes visible on ultrasound around 10 to 12 weeks of pregnancy, with clearer and more detailed assessments possible by the second trimester. Circummarginate placenta can be detected by ultrasound, usually during the second trimester, with a mean diagnosis age around 19–20 weeks, often identified during routine anatomic scans at 18-22 weeks [12].

Conclusion

Circummarginate placentas show a greater prevalence of gross and histologic findings associated with maternal vascular malperfusion than controls. As circummarginate placental membranes are visible on ultrasound beginning at 18-22 weeks, increased monitoring of pregnancies from this point may help prevent serious clinical conditions associated with maternal vascular malperfusion such as gestational hypertension and pre-eclampsia.

Conflict of Interest

No financial interest or other conflict of interest exists.

Bibliography

1. Wentworth P. "Circumvallate and circummarginate placentas. Their incidence and clinical significance". *American Journal of Obstetrics and Gynecology* 1.102 (1968): 44-47.

2. Benson RC., *et al.* "Circumvallate and circummarginate placenta. Unimportant clinical entities". *Obstetrics and Gynecology* 34.6 (1969): 799-804.

3. Khong TY., *et al.* "Inadequate maternal vascular response to placentation in pregnancies complicated by pre-eclampsia and by small-for-gestational age infants". *British Journal of Obstetrics and Gynecology* 93.10 (1986): 1049-1059.

4. Hempstock J., *et al.* "The contribution of placental oxidative stress to early pregnancy failure". *Human Pathology* 34.12 (2003): 1265-1275.

5. Jauniaux E., *et al.* "Trophoblastic oxidative stress in relation to temporal and regional differences in maternal placental blood flow in normal and abnormal early pregnancies". *American Journal of Pathology* 162.1 (2003): 115-125.

6. Burton GJ., *et al.* "Rheological and physiological consequences of conversion of the maternal spiral arteries for uteroplacental blood flow during human pregnancy". *Placenta* 30.6 (2009): 473-482.

7. Khong TY., *et al.* "Sampling and Definitions of Placental Lesions: Amsterdam Placental Workshop Group Consensus Statement". *Archives of Pathology and Laboratory Medicine* 140.7 (2016): 698-713.

8. Timofeeva AV, *et al.* "Prediction of Early- and Late-Onset Pre-Eclampsia in the Preclinical Stage via Placenta-Specific Extracellular miRNA Profiling". *International Journal of Molecular Sciences* 28.24.9 (2023): 8006.

9. Staff AC. "The two-stage placental model of preeclampsia: An update". *Journal of Reproductive Immunology* 134.135 (2019): 1-10.

10. Erez O., *et al*. "Preeclampsia and eclampsia: the conceptual evolution of a syndrome". *American Journal of Obstetrics and Gynecology* 226.2S (2022): S786-S803.

11. Gyselaers W., *et al.* "Preeclampsia: a gestational cardiorenal syndrome". *Journal of Physiology* 597.18 (2019): 4695-4714.

12. Agolah D. "Circumvallate placenta. Case study". *Radiopaedia.org* (2026).