Available online on 15.02.2026 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

Open Access Full Text Article

Research Article

Histopathological Evaluation of Placental Changes in Women with Excessive Gestational Weight Gain

Özkan Yükselmiş, Murat Akkuş *, Fırat Aşır

Department of Histology and Embryology, Medical Faculty, Dicle University, 21280, Sur, Diyarbakır, Turkey

Article Info:

Article History:

Received 26 Nov 2025
Reviewed 12 Jan 2026
Accepted 03 Feb 2026
Published 15 Feb 2026

Cite this article as:

Yükselmiş Ö, Akkuş M, Aşır F, Histopathological Evaluation of Placental Changes in Women with Excessive Gestational Weight Gain, Journal of Drug Delivery and Therapeutics. 2026; 16(2):164-167 *DOI: http://dx.doi.org/10.22270/jddt.v16i2.7574*

For Correspondence:

Murat Akkuş, Department of Histology and Embryology, Medical Faculty, Dicle University, 21280, Sur, Diyarbakır, Turkey

Abstract

Objective: Excessive weight gain during pregnancy has been associated with adverse maternal and fetal outcomes; however, its effects on placental histomorphology remain incompletely characterized. The present study aimed to evaluate placental histopathological alterations in women who gained excessive weight during pregnancy using Hematoxylin–Eosin (H&E) staining.

Materials and Methods: Placental tissues were obtained postpartum from 45 normal-weight pregnant women (BMI <30 kg/m^2) and 45 women with excessive gestational weight gain/obesity (BMI ≥30 kg/m^2). All samples underwent routine histological processing and were stained with H&E. Placental sections were examined under light microscopy for villous architecture, syncytiotrophoblast integrity, stromal changes, fibrin deposition, vascular congestion, and syncytial knot formation.

Results: Placentae from the control group exhibited preserved villous architecture, intact syncytiotrophoblast layers, and normal stromal and vascular morphology. In contrast, placentae from the high-BMI group demonstrated marked histopathological alterations, including villous structural disorganization, stromal degeneration, increased fibrin deposition, prominent vascular congestion, and an increased number of syncytial knots.

Conclusion: Excessive weight gain during pregnancy is associated with significant placental histomorphological alterations detectable by routine H&E staining. These structural changes may reflect impaired uteroplacental circulation and reduced placental functional capacity, potentially contributing to an adverse intrauterine environment.

Keywords: Gestational weight gain, placenta, hematoxylin–eosin, histopathology, villous degeneration

INTRODUCTION

Pregnancy is a dynamic physiological process requiring complex maternal adaptations to support fetal growth and development[1]. The placenta plays a central role in this process by mediating nutrient exchange, gas transfer, metabolic regulation, and immunological tolerance between the mother and fetus [2]. Preservation of placental structural integrity is therefore essential for optimal pregnancy outcomes. In recent decades, excessive gestational weight gain and maternal obesity have emerged as major public health concerns[3]. These conditions have been linked to increased risks of gestational diabetes, hypertensive disorders, cesarean delivery, and long-term metabolic disease in offspring[4]. Growing evidence suggests that maternal metabolic stress adversely affects placental development, leading to morphological and functional abnormalities[5].

Histopathological studies have reported that excessive maternal weight gain may be associated with villous immaturity, increased fibrin deposition, stromal edema, vascular congestion, and altered trophoblastic morphology[6]. Such changes can impair uteroplacental blood flow and compromise fetal oxygen and nutrient delivery[7]. However, detailed histomorphological evaluations focusing exclusively on routine H&E findings remain limited[8].

Therefore, the present study aimed to investigate placental histopathological changes associated with excessive gestational weight gain using Hematoxylin–Eosin staining, providing a structural basis for understanding placental dysfunction in this population.

MATERIALS AND METHODS

Study Population and Tissue Collection

This study was conducted at Dicle University Faculty of Medicine, Department of Obstetrics and Gynecology. Placental tissues were collected from women aged 18–40 years following delivery. The control group consisted of 45 normal-weight pregnant women (BMI <30 kg/m^2), while the study group included 45 women with excessive gestational weight gain/obesity (BMI ≥30 kg/m^2). Pregnancies complicated by systemic diseases, gestational diabetes, preeclampsia, infection, or other obstetric complications were excluded.

Placental samples not required for routine pathological diagnosis were collected immediately after delivery for histological analysis.

Histological Processing and Hematoxylin–Eosin Staining

Placental tissue samples were fixed in 10% neutral buffered formalin for 24 hours, followed by routine dehydration, clearing, and paraffin embedding. Serial sections of 4 µm thickness were cut using a microtome and mounted on glass slides. Standard Hematoxylin–Eosin staining was performed according to established protocols.

RESULTS

Hematoxylin–Eosin Findings

Control Group (Figure 1): Placental sections from the control group demonstrated well-preserved chorionic villi with regular contours and uniform size. The syncytiotrophoblast layer was continuous and morphologically intact. The villous stroma appeared compact, with no evidence of degeneration, edema, or excessive fibrin deposition. Vascular structures were patent and evenly distributed, and intervillous spaces were well defined. Syncytial knot formation was minimal and within physiological limits.

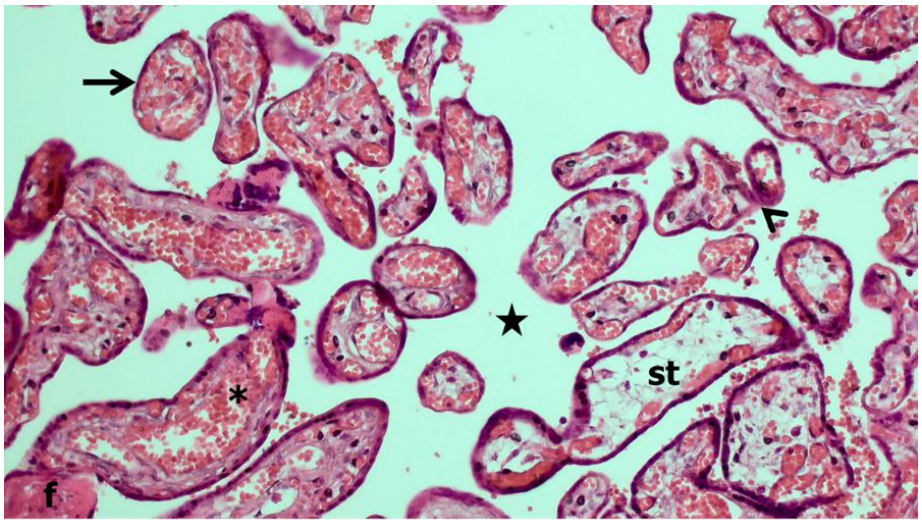

Figure 1: Placental histology of the control group. Arrow: chorionic villus; arrowhead: syncytial knot; st: stroma; asterisk: villous capillary; f: fibrin deposition; star: intervillous space. Scale bar: 50 µm; Magnification: ×20.

High BMI Group (Figure 2): Placentae obtained from women with excessive gestational weight gain exhibited pronounced histopathological alterations. Chorionic villi appeared irregular, enlarged, and structurally disorganized. Stromal degeneration, including vacuolization and loss of normal connective tissue architecture, was frequently observed. Marked vascular congestion with erythrocyte accumulation was evident within villous capillaries and intervillous spaces. Fibrin deposition was increased in both villous and intervillous regions. Additionally, syncytial knots were more numerous and prominent compared with the control group.

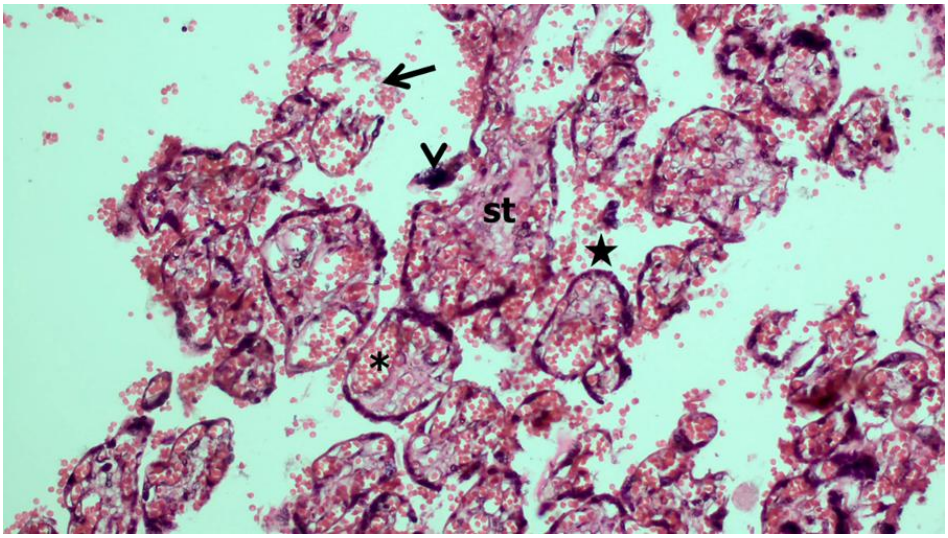

Figure 2: Placental histopathological alterations in the high-BMI group. Arrow: chorionic villus; arrowhead: syncytial knot; st: stroma; asterisk: villous capillary; f: fibrin deposition; star: intervillous space. Scale bar: 50 µm; Magnification: ×20.

DISCUSSION

The present study demonstrates that excessive weight gain during pregnancy is associated with distinct placental histopathological alterations identifiable using routine Hematoxylin–Eosin staining. Compared with normal-weight pregnancies, placentae from women with high BMI exhibited significant villous structural disruption, stromal degeneration, vascular congestion, increased fibrin deposition, and enhanced syncytial knot formation. These findings are consistent with previous reports indicating that maternal metabolic stress adversely affects placental development[9-12]. Villous disorganization and stromal degeneration may reduce the effective surface area available for maternal–fetal exchange, while vascular congestion suggests impaired uteroplacental blood flow [13]. Increased fibrin deposition is commonly interpreted as a marker of placental injury and hypoxic stress and has been associated with reduced placental efficiency [14].

The observed increase in syncytial knots may reflect accelerated trophoblastic turnover or chronic placental stress [15]. Although syncytial knot formation can be a physiological feature of term placentae, excessive accumulation is often associated with placental hypoxia and impaired perfusion [16,17]. Taken together, these histopathological changes suggest that excessive gestational weight gain creates a suboptimal intrauterine environment by compromising placental structure [18]. Such alterations may contribute to adverse fetal outcomes and support the concept that maternal metabolic status plays a critical role in shaping placental morphology.

In conclusion, routine H&E staining provides valuable insight into placental structural changes associated with excessive maternal weight gain. Recognition of these alterations may enhance understanding of placental dysfunction in high-risk pregnancies and underscores the importance of optimal weight management during gestation.

CONCLUSION

Excessive gestational weight gain is associated with distinct histopathological alterations in the placenta. Routine Hematoxylin–Eosin examination revealed disruption of chorionic villous architecture, stromal degeneration, increased fibrin deposition, vascular congestion, and enhanced syncytial knot formation in placentae from the high-BMI group compared with controls. These structural changes suggest compromised placental integrity and may reflect an adverse intrauterine environment, underscoring the importance of appropriate maternal weight management during pregnancy.

Acknowledgements: This study was part of doctoral thesis of Dr. Özkan Yükselmiş at department of Histology and Embryology, Dicle University.

Ethical Approval: This study was approved by the Dicle University Faculty of Medicine Non-Interventional Clinical Research Ethics Committee (Approval date: 16 April 2025; Approval number: 2025/150). All procedures were conducted in accordance with the ethical standards of the institutional and national research committees and with the principles of the Declaration of Helsinki.

Informed Consent: Informed consent was obtained from all participants prior to inclusion in the study.

Conflict of Interest: The author declares that there is no conflict of interest regarding the publication of this thesis.

Funding: This research was funded by Dicle University Scientific Research Platform (DÜBAP) by project number: TIP.25.029.

References

1. Burton GJ, Jauniaux E. What is the placenta? Am J Obstet Gynecol. 2015;213(4 Suppl):S6-S8. https://doi.org/10.1016/j.ajog.2015.07.050 PMid:26428504

2. Benirschke K, Burton GJ, Baergen RN. Pathology of the Human Placenta. 6th ed. New York: Springer; 2012. https://doi.org/10.1007/978-3-642-23941-0 PMCid:PMC3381679

3. Poston L, Caleyachetty R, Cnattingius S, Corvalán C, Uauy R, Herring S, Gillman MW. Preconceptional and maternal obesity: epidemiology and health consequences. Lancet Diabetes Endocrinol. 2016;4(12):1025-1036. https://doi.org/10.1016/S2213-8587(16)30217-0 PMid:27743975

4. Institute of Medicine (US); National Research Council. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington (DC): National Academies Press; 2009.

5. Desoye G. The human placenta in diabetes and obesity: friend or foe? Diabetes Care. 2018;41(7):1362-1369. https://doi.org/10.2337/dci17-0045 PMid:29934479

6. Kristiansen O, Roland MC, Zucknick M, Reine TM, Kolset SO, Henriksen T, Lekva T, Michelsen T. Maternal body mass index and placental weight: a role for fetal insulin, maternal insulin and leptin. J Endocrinol Invest. 2022;45(11):2105-2121. https://doi.org/10.1007/s40618-022-01842-2 PMid:35781790 PMCid:PMC9525437

7. Challier JC, Basu S, Bintein T, Minium J, Hotmire K, Catalano PM, Hauguel-de Mouzon S. Obesity in pregnancy stimulates macrophage accumulation and inflammation in the placenta. Placenta. 2008;29(3):274-281. https://doi.org/10.1016/j.placenta.2007.12.010 PMid:18262644 PMCid:PMC4284075

8. Mayhew TM. A stereological perspective on placental morphology in normal and complicated pregnancies. J Anat. 2009;215(1):77-90. https://doi.org/10.1111/j.1469-7580.2008.00994.x PMid:19141109 PMCid:PMC2714641

9. Kelly AC, Powell TL, Jansson T. Placental function in maternal obesity. Clin Sci (Lond). 2020;134(8):961-984. https://doi.org/10.1042/CS20190266 PMid:32313958 PMCid:PMC8820171

10. Brouwers L, Franx A, Vogelvang TE, Houben ML, van Rijn BB, Nikkels PG. Association of maternal prepregnancy body mass index with placental histopathological characteristics in uncomplicated term pregnancies. Pediatr Dev Pathol. 2019;22(1):45-52. https://doi.org/10.1177/1093526618785838 PMid:29969058 PMCid:PMC6604681

11. Redman CW, Sargent IL. Placental stress and pre-eclampsia: a revised view. Placenta. 2009;30 Suppl A:S38-S42. https://doi.org/10.1016/j.placenta.2008.11.021 PMid:19138798

12. Kingdom JC, Kaufmann P. Oxygen and placental villous development: origins of fetal hypoxia. Placenta. 1997;18(8):613-621. https://doi.org/10.1016/S0143-4004(97)90000-X PMid:9364596

13. Burton GJ, Jauniaux E. Oxidative stress. Best Pract Res Clin Obstet Gynaecol. 2011;25(3):287-299. https://doi.org/10.1016/j.bpobgyn.2010.10.016 PMid:21130690 PMCid:PMC3101336

14. Friis CM, Roland MC, Godang K, Voldner N, Bollerslev J, Henriksen T. Adiposity-related inflammation: effects of pregnancy. Obesity (Silver Spring). 2013;21(1):E124-E130. https://doi.org/10.1002/oby.20120 PMid:23505192

15. Huppertz B. The anatomy of the normal placenta. J Clin Pathol. 2008;61(12):1296-1302. https://doi.org/10.1136/jcp.2008.055277 PMid:18755720

16. Benirschke K, Kaufmann P. Pathology of the Human Placenta. 5th ed. New York: Springer; 2006.

17. Santos ED, Hernández MH, Sérazin V, Vialard F, Dieudonné MN. Human placental adaptive changes in response to maternal obesity: sex specificities. Int J Mol Sci. 2023;24(11):9770. https://doi.org/10.3390/ijms24119770 PMid:37298720 PMCid:PMC10253453

18. Barker DJ. The fetal and infant origins of adult disease. BMJ. 1990;301(6761):1111. https://doi.org/10.1136/bmj.301.6761.1111 PMid:2252919 PMCid:PMC1664286