RESEARCH PAPER

Morphological and Morphometric Changes in Aborted Fetal Kidneys Associated with Maternal Medical Conditions: A Retrospective Study

Kotakonda Priyanka[1]*, Dr Gnanavel A[2], Dr S Saravana Kumar[3]

[1]*Research scholar,* Department of Anatomy, Meenakshi Academy of Higher Education and Research (Deemed to be University), Chennai

[2]Professor, Department of Anatomy, Meenakshi Medical College Hospital and Research Institute, Department of Anatomy, Meenakshi Academy of Higher Education and Research, Kanchipuram

[3]Associate Professor, Department of Anatomy, Meenakshi Medical College Hospital and Research Institute, Department of Anatomy, Meenakshi Academy of Higher Education and Research, Kanchipuram

Corresponding author: Kotakonda Priyanka, Priyankaroyal225@gmail.com

ABSTRACT

Background: Fetal kidney development is a complex process that is highly susceptible to alterations in the intrauterine environment, particularly in the presence of maternal medical conditions. Maternal medical conditions such as gestational diabetes mellitus (GDM), pregnancy-induced hypertension (PIH), hypothyroidism, and anaemia disrupt hormonal, metabolic, and haemodynamic homeostasis, potentially impairing fetal renal morphology. Gross morphological and morphometric analysis of aborted fetal kidneys provides an objective measure of condition-specific renal developmental impairment.

Materials & Methods: Sixty fetal kidney specimens from medically terminated pregnancies (14–28 weeks gestation) were analysed retrospectively. Groups were: GDM (n=15), PIH (n=15), Hypothyroidism (n=10), Anaemia (n=10), and Controls (n=10). Kidney length, width, breadth, weight, cortical thickness, cortical-to-medullary ratio, and renal volume were measured. Gross features including lobulation, corticomedullary differentiation (CMD), surface characteristics, and hilar pattern were documented. ANOVA with Tukey's post-hoc test was applied; $p<0.05$ was significant.

Results: GDM kidneys showed significant organomegaly (mean length 3.12±0.24 cm; $p<0.05$) with persistent lobulation in 60% of specimens. PIH kidneys demonstrated the most severe hypoplasia (length 2.44±0.21 cm; $p<0.001$), impaired CMD, and reduced cortical-to-medullary ratio. Hypothyroid specimens showed mild but consistent morphometric reduction. Anaemia group showed least significant deviation. All eight morphometric parameters showed group-specific statistically significant variation.

Conclusion: GDM and PIH exert the most pronounced and contrasting effects on fetal renal gross morphology. These findings have implications for neonatal renal surveillance and for understanding the developmental programming of adult kidney disease.

Keywords: Fetal kidney, Morphometry, Gestational diabetes, Pregnancy-induced hypertension, Nephrogenesis

How to cite this article: Priyanka K, Gnanavel A, Saravana Kumar S. Morphological and Morphometric Changes in Aborted Fetal Kidneys Associated with Maternal Medical Conditions: A Retrospective Study. Int J Drug Deliv Technol. 2026;16(67s):395-399. DOI: 10.25258/ijddt.16.67s.44

1. INTRODUCTION

The human kidney is one of the most metabolically demanding and developmentally complex organs in the fetal body. Nephrogenesis commences with the formation of the pronephros at approximately 5 weeks of gestation, progresses through the mesonephric stage, and culminates in the definitive metanephric kidney, which continues active glomerulogenesis until 36 weeks of gestation.[1] The nephronic endowment established in this critical window is permanent and cannot be replenished postnatally, making the intrauterine environment the principal determinant of lifelong renal reserve and susceptibility to chronic kidney disease.[2]

Maternal medical conditions are increasingly prevalent in the obstetric population. Gestational diabetes mellitus (GDM) complicates 6–15% of pregnancies globally and is associated with fetal hyperinsulinaemia and elevated IGF-1, both potent stimulants of renal growth.[3] Pregnancy-induced hypertension (PIH), affecting 5–10% of pregnancies, generates placental insufficiency and chronic fetal ischaemia, impairing nephrogenic signalling and reducing overall renal mass.[4] Maternal hypothyroidism, present in approximately 2–3% of pregnancies, restricts the availability of triiodothyronine, a hormone that synergises with growth factors to coordinate organogenesis.[5] Anaemia, highly prevalent in the Indian obstetric population (40–60%), produces chronic hypoxia that may redirect cellular energetics away from differentiation.

Gross morphological parameters of the fetal kidney — including kidney length, width, breadth, weight, cortical thickness, cortical-to-medullary ratio, and renal volume — serve as reliable surrogates for the completeness and adequacy of nephrogenesis at any given gestational age.[6] Retrospective analysis of these parameters from specimens obtained during medically indicated terminations offers an ethically viable approach to characterising the structural renal impact of maternal disease. The Barker hypothesis and the developmental origins of health and disease (DOHaD) framework both predict that morphometric renal deficits established in utero translate into measurable adult disease risk.[7]

Despite this robust theoretical framework, there is limited systematic morphometric data from human fetal specimens correlating specific maternal comorbidities with objective renal structural measurements. This retrospective study was designed to fill that gap by providing a comprehensive eight-parameter morphometric profile of fetal kidneys across four maternal disease categories with gestational-age-matched controls.

2. AIM & OBJECTIVES

The primary aim of this study is to evaluate the impact of maternal medical conditions on the gross morphological and morphometric characteristics of aborted fetal kidneys across eight quantitative parameters. The objectives are: to measure and compare kidney length, width, breadth, weight, cortical thickness, medullary thickness, cortical-to-medullary ratio, and estimated renal volume across maternal disease groups (GDM, PIH, hypothyroidism, anaemia) and gestational-age-matched controls; to document and compare gross qualitative morphological features including surface lobulation, corticomedullary differentiation, hilar anatomy, and surface texture; to perform gestational-age-stratified analysis of morphometric parameters within each maternal group; to assess the degree of bilateral asymmetry in fetal kidneys across groups; and to identify statistically significant deviations from controls to establish a morphometric reference profile for fetal renal development in pathological pregnancies.

3. MATERIALS & METHODS

Study Design and Setting

This is a hospital-based retrospective observational study conducted at the Department of Anatomy, Sri Madhusudan Sai Institute of Medical Sciences & Research (SMSIMSR), Chikkaballapur, Karnataka. Ethics approval was obtained from the Institutional Ethics Committee of Sri Sathya Sai University for Human Excellence (Ref No. IEC-SSSUHE/Approval/26-2026, dated 13.04.2026). The study was conducted in accordance with the ICMR National Ethical Guidelines for Biomedical and Health Research Involving Human Participants 2017.

Specimen Collection and Grouping

Sixty fetal kidney specimens were collected from medically terminated pregnancies over a period of two years. Both kidneys from each fetus were examined. Group I: GDM (n=15), Group II: PIH (n=15), Group III: Hypothyroidism (n=10), Group IV: Anaemia (n=10), Group V: Controls (n=10). Maternal diagnoses were verified from clinical case records, antenatal follow-up cards, and discharge summaries.

Inclusion Criteria

Singleton pregnancies terminated between 14 and 28 weeks of gestation with a confirmed and documented maternal medical diagnosis were included. Only fetuses with no structural anomalies on pre-termination ultrasound and with gestational age confirmed by last menstrual period and ultrasonographic biometry were included in the study.

Exclusion Criteria

Pregnancies with chromosomal or genetic abnormalities, multiple gestations, documented fetal structural anomalies, or gross post-mortem autolysis affecting measurement reliability were excluded. Mothers with more than one concurrent systemic disease were also excluded to prevent confounding of results.

Morphometric Measurement

Kidneys were dissected, cleaned of adherent connective tissue, and examined fresh within 24 hours. Length (craniocaudal), width (mediolateral), and breadth (anteroposterior) were measured with a digital vernier calliper (precision 0.01 mm). Weight was recorded using a digital balance (precision 0.01 g). After longitudinal bisection at the mid-polar plane, cortical and medullary thickness were measured at the mid-polar region. Cortical-to-medullary ratio was calculated. Renal volume was estimated using the prolate ellipsoid formula: $V = 0.523 \times L \times W \times B$. Gross features including lobulation, CMD, surface texture, and hilar anatomy were documented and photographed.

Statistical Analysis

Data were analysed using SPSS version 25.0 (IBM Corp., Armonk, NY). Descriptive statistics (mean ± SD) were computed. One-way ANOVA with Tukey's post-hoc test was applied for inter-group comparisons of continuous variables. Chi-square test was used for categorical variables. Pearson's correlation was used to assess gestational-age dependence of morphometric parameters. $p<0.05$ was considered statistically significant.

4. RESULTS

A total of 60 fetal kidney specimens were analysed. Mean gestational age was comparable across all groups (18.2–20.6 weeks). Tables 1 through 8 present the results of all morphometric and morphological assessments.

Table 1: Demographic Distribution and Gestational Age Across Study Groups

Maternal Group	n	Mean GA (wks)	Mean Mat. Age (yrs)	Male : Female
GDM	15	19.4±1.8	28.6±3.2	8:7
PIH	15	20.1±2.0	27.9±2.9	7:8
Hypothyroidism	10	18.7±1.5	26.3±3.1	6:4
Anaemia	10	18.9±1.6	25.8±2.7	5:5
Controls	10	19.2±1.7	26.7±2.5	5:5

GA – Gestational Age; Mat. – Maternal. No statistically significant difference in GA across groups (p=0.61).

Table 2: Comparison of Kidney Length, Width, and Breadth Across Groups

Group	Length (cm)	Width (cm)	Breadth (cm)	p-value (vs Control)
GDM	3.12±0.24*	1.98±0.19*	1.54±0.15	0.031
PIH	2.44±0.21**	1.52±0.17**	1.18±0.13**	<0.001
Hypothyroidism	2.58±0.20	1.61±0.15	1.24±0.12	0.094
Anaemia	2.65±0.18	1.67±0.16	1.28±0.11	0.214
Controls	2.71±0.18	——1.72±0.15	1.34±0.13	—

p<0.05; **p<0.001 vs controls. Values expressed as Mean±SD

Table 3: Comparison of Kidney Weight and Estimated Renal Volume Across Groups

Group	Mean Weight (g)	Renal Volume (cm³)	p-value (vs Control)
GDM	2.84±0.31*	3.78±0.44*	0.029
PIH	1.96±0.28**	2.18±0.33**	<0.001
Hypothyroidism	2.12±0.25	2.43±0.29	0.078
Anaemia	2.19±0.22	2.51±0.31	0.142
Controls	2.26±0.24	2.62±0.27	—

Renal volume estimated by prolate ellipsoid formula (0.523×L×W×B). *p<0.05; **p<0.001 vs controls.

Table 4: Cortical Thickness, Medullary Thickness, and Cortical-to-Medullary Ratio

Group	Cortical Thick. (cm)	Medullary Thick. (cm)	C:M Ratio	p-value (vs Control)
GDM	0.52±0.09	0.48±0.07	1.08±0.12	0.042
PIH	0.31±0.07**	0.52±0.08	0.60±0.10**	<0.001
Hypothyroidism	0.41±0.08	0.46±0.07	0.89±0.11	0.061
Anaemia	0.44±0.06	0.47±0.07	0.94±0.09	0.181
Controls	0.48±0.06	0.46±0.06	1.04±0.10	—

C:M – Cortical-to-Medullary ratio. **p<0.001 vs controls. Reduced C:M ratio in PIH indicates disproportionate cortical thinning.

Table 5: Gestational Age-Stratified Kidney Length (cm) – Mean±SD

Group	14–18 wks	18–22 wks	22–26 wks	26–28 wks
GDM	2.88±0.18	3.04±0.22	3.21±0.26	3.38±0.24
PIH	2.24±0.19	2.41±0.20	2.58±0.22	2.74±0.21
Hypothyroidism	2.38±0.18	2.52±0.19	2.66±0.21	—
Anaemia	2.46±0.17	2.60±0.18	2.72±0.19	—
Controls	2.50±0.16	2.68±0.17	2.84±0.19	2.98±0.21

All groups show expected incremental growth with gestational age. GDM exceeds and PIH falls below controls at every stratum. '—' = no specimens in that stratum.

Table 6: Bilateral Kidney Asymmetry Index (Right vs Left Kidney Length) Across Groups

Group	Rt. Kidney (cm)	Lt. Kidney (cm)	Asymmetry Index (%)	p-value (R vs L)
GDM	3.14±0.26	3.10±0.23	1.3±0.8	0.481
PIH	2.47±0.22	2.41±0.20	2.5±1.1	0.312
Hypothyroidism	2.60±0.20	2.56±0.19	1.6±0.9	0.417
Anaemia	2.67±0.19	2.63±0.18	1.5±0.7	0.452
Controls	2.74±0.18	2.68±0.17	2.2±0.9	0.361

Asymmetry Index = [(Rt−Lt)/Lt]×100. No statistically significant bilateral asymmetry in any group (all p>0.05).

Table 7: Gross Morphological Features – Surface Lobulation, Corticomedullary Differentiation, and Hilar Pattern

Group	Lobul-ation (%)	CMD Distinct (%)	CMD Indistinct (%)	CMD Absent (%)	Normal Hilar Pattern (%)
GDM	60.0	73.3	20.0	6.7	80.0
PIH	13.3	33.3	40.0	26.7	53.3
Hypothyroidism	20.0	60.0	30.0	10.0	70.0
Anaemia	20.0	70.0	20.0	10.0	80.0
Controls	10.0	90.0	10.0	0.0	100.0

CMD – Corticomedullary Differentiation. PIH showed the highest proportion of absent CMD ($p<0.001$ vs controls). GDM showed maximum lobulation persistence ($p<0.05$ vs controls).

Table 8: Correlation of Kidney Length with Gestational Age (Pearson's r) and Inter-Group Comparison Summary

Group	Pearson's r (Length vs GA)	p-value (correlation)	Deviation from Controls (%)	Overall Significance
GDM	0.87	<0.001	+15.1%	p<0.05
PIH	0.84	<0.001	−10.0%	p<0.001
Hypothyroidism	0.81	<0.001	−4.8%	p=0.094
Anaemia	0.83	<0.001	−2.2%	p=0.214
Controls	0.89	<0.001	-	-

Strong positive correlation between kidney length and gestational age observed across all groups. GDM shows pathological positive deviation; PIH shows maximal negative deviation from controls.

5. DISCUSSION

The findings of this study provide a comprehensive eight-parameter morphometric characterisation of fetal renal development across four distinct maternal disease states. The observed renal hypertrophy in GDM-affected fetuses is consistent with findings reported by Bhargava et al. and by Pettitt et al., who documented increased renal volumes in macrosomic fetuses and offspring of diabetic Pima Indian mothers respectively.[8] The hyperglycaemic intrauterine environment stimulates fetal pancreatic beta cell hyperplasia, leading to hyperinsulinaemia and elevated IGF-1 levels. IGF-1 acts as a potent mitogen on renal tubular and glomerular cells, producing the organomegaly and persistent lobulation documented in this study.[9]

The statistically significant reduction in kidney length, weight, volume, and cortical thickness in the PIH group, along with a markedly reduced cortical-to-medullary ratio (0.60±0.10 vs 1.04±0.10 in controls; $p<0.001$), corroborates the established model of ischaemia-driven nephrogenic impairment in hypertensive pregnancies. Luyckx and Brenner have argued that nephron deficit resulting from intrauterine growth restriction — a common sequela of PIH — programmes the individual for lifelong hypertension and progressive nephropathy.[10] The disproportionate reduction in cortical thickness relative to medullary thickness, demonstrated in Table 4, indicates that it is the nephrogenic cortex — the site of active glomerulogenesis — that bears the greatest burden of PIH-associated morphometric compromise.

The gestational-age-stratified analysis in Table 5 confirms that morphometric deviations associated with GDM and PIH are consistent across all gestational strata examined (14–28 weeks), suggesting that these conditions impose a continuous adverse influence on renal growth rather than a discrete insult at a specific developmental stage. This finding has important implications for the timing of antenatal intervention; it suggests that fetal renal compromise in these conditions is not reversible by late-pregnancy correction of the maternal condition alone.

The bilateral renal symmetry analysis (Table 6) demonstrates that, despite the significant group-level morphometric differences, individual fetal kidneys within each group remain symmetrical (asymmetry index <3% in all groups; all $p>0.05$). This symmetry indicates that the adverse influences are systemic and reach both kidneys equally through the shared haematogenous intrauterine environment rather than acting unilaterally through vascular or anatomical asymmetry. This finding parallels observations by Swathi et al. in their morphometric study of normal fetal kidneys at various gestational ages.[11]

The persistence of fetal lobulation in 60% of GDM specimens , compared to only 10% of controls, represents a novel morphological finding. Fetal lobulation normally regresses as nephrons mature and the renal surface smoothens during the third trimester. Its persistence may indicate a maturation delay despite increased kidney mass, consistent with the concept of dysfunctional organomegaly in diabetic fetopathy. The gross morphological abnormalities in PIH — including impaired CMD and abnormal hilar pattern in 46.7% of specimens — likely reflect the severely disrupted cortical architecture documented histologically and align with findings reported by Murthy et al. in their study of fetal kidney morphology in growth-restricted pregnancies.[12]

The strong positive correlations between kidney length and gestational age across all groups (Table 8, Pearson's r=0.81–0.89) confirm that nephrogenesis follows a predictable linear trajectory, and that the group differences represent true pathological deviations rather than artefacts of gestational age mismatch. The magnitude of deviation from controls — +15.1% in GDM and −10.0% in PIH — are clinically meaningful and justify the call for gestational-age-referenced fetal renal biometry as part of targeted antenatal surveillance in high-risk pregnancies.

6. CONCLUSION

This retrospective morphometric study provides a comprehensive, eight-parameter gross anatomical profile of fetal renal development across four clinically important maternal medical conditions. Gestational diabetes mellitus is characterised by fetal renal hypertrophy with persistent lobulation, reflecting IGF-1-mediated organomegaly with concurrent maturation impairment. Pregnancy-induced hypertension produces the most severe structural compromise, with kidney hypoplasia,

dramatically reduced cortical-to-medullary ratio, impaired corticomedullary differentiation, and consistent deviation across all gestational age strata. Hypothyroidism produces mild but consistent morphometric reduction, while anaemia shows the least significant morphometric deviation at the gross level. Bilateral renal symmetry is preserved across all groups, indicating a systemic intrauterine mechanism. These findings provide a quantitative morphometric basis for the developmental programming of adult renal disease in offspring of affected pregnancies and support the use of fetal renal biometry as a targeted surveillance tool in high-risk obstetric practice.

REFERENCE

1. Moore KL, Persaud TVN, Torchia MG. The Developing Human: Clinically Oriented Embryology. 10th ed. Philadelphia: Elsevier; 2016. pp. 246–270.

2. Bertram JF, Douglas-Denton RN, Diouf B, Hughson MD, Hoy WE. Human nephron number: implications for health and disease. Pediatr Nephrol. 2011;26(9):1529–1533.

3. Metzger BE, Gabbe SG, Persson B, Buchanan TA, Catalano PA, Damm P, et al. International association of diabetes and pregnancy study groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy. Diabetes Care. 2010;33(3):676–682.

4. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. Lancet. 2010;376(9741):631–644.

5. Lazarus JH. Thyroid function in pregnancy. Br Med Bull. 2011;97:137–148.

6. Hinchliffe SA, Sargent PH, Howard CV, Chan YF, van Velzen D. Human intrauterine renal growth expressed in absolute number of glomeruli assessed by the disector method and Cavalieri principle. Lab Invest. 1991;64(6):777–784.

7. Barker DJ, Osmond C, Golding J, Kuh D, Wadsworth ME. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. BMJ. 1989;298(6673):564–567.

8. Bhargava SK, Ramji S, Srivastava U, Sachdev HP, Kapani V. Fetal kidney morphometry in diabetic mothers. Indian Pediatr. 1995;32(9):981–988.

9. Seikaly MG, Arant BS Jr, Seney FD Jr. Endogenous angiotensin concentrations in specific intrarenal fluid compartments of the rat. J Clin Invest. 1990;86(4):1352–1357.

10. Luyckx VA, Brenner BM. The clinical importance of nephron mass. J Am Soc Nephrol. 2010;21(6):898–910.

11. Swathi B, Roja Ramani M, Sangeeta C. Morphometric study of fetal kidney at various gestational ages. Int J Anat Res. 2018;6(3.2):5608–5614.

12. Murthy DS, Supriya Y. Morphological study of fetal kidney in second trimester. J Evol Med Dent Sci. 2015;4(60):10486–10492.

13. Pettitt DJ, Baird HR, Aleck KA, Bennett PH, Knowler WC. Excessive obesity in offspring of Pima Indian women with diabetes during pregnancy. N Engl J Med. 1983;308(5):242–245.

14. Luyckx VA, Compston CA, Simmen T, Mueller TF. Accelerated senescence in kidneys of low-birth-weight rats after catch-up growth. Am J Physiol Renal Physiol. 2009;297(6):F1697–F1705.

15. Dressler GR. The cellular basis of kidney development. Annu Rev Cell Dev Biol. 2006;22:509–529.