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PATHOMORPHOLOGY OF THE ADRENAL GLANDS OF INFANTS WHO DIED FROM BIRTH TRAUMA

Sayfiddin Khoji Kadriddin Shuhrat ugli[1], Babaev Khamza Nurmatovich[2], Allaberganov Dilshod Shavkatovich[3], Murodullayev Mironshokh Nodirbek ugli[4] Eshonkhodjaeva Madinakhon Otabek kizi[5]

1. Master of the "Pathological anatomy" of the Tashkent State Medical University.

dr.sayfiddinkhoji@gmail.com, Orcid NO: 0009-0000-5476-5242;

2. Associate professor of the Pathological anatomy department, PhD, Tashkent State Medical University, khamzababaev@gmail.com, Orcid NO: 0009-0009-1033-1472

3. Assistent of the Pathological anatomy department, PhD, Tashkent State Medical University, dilshodbek9347225@mail.ru, Orcid NO: 0009-0003-1558-5101

4. Student of direction of Management of Tashkent Medical Academy.

mironshoxmurodullayev@gmail.com, Orcid NO: 0009-0004-7474-1722

5. Student of faculty of General Medicine of Tashkent State Medical University, madi270105@gmail.com, Orcid NO: 0009-0006-9714-0190

Tashkent, 100109, Uzbekistan.

Annotation: Birth trauma remains a significant cause of neonatal mortality, with profound effects on vital organs, including the adrenal glands, which play a critical role in stress response and homeostasis. This article examines the pathomorphological changes in the adrenal glands of infants who died from birth trauma, focusing on histological and gross anatomical alterations, their association with trauma severity, and implications for understanding neonatal death mechanisms. The study analyzes autopsy data from 100 infants who succumbed to birth trauma, identifying adrenal hemorrhage in 70% of cases, cortical necrosis in 45%, and inflammatory infiltrates in 30%. Globally, birth trauma accounts for 5–10% of neonatal deaths, with an estimated incidence of 2–7 per 1,000 live births in developed countries and up to 20 per 1,000 in low-resource settings. Risk factors, including prolonged labor (OR = 3.5, 95% CI: 2.1–5.8), instrumental delivery (OR = 4.2, 95% CI: 2.5–7.0), and macrosomia (OR = 2.8, 95% CI: 1.7–4.6), were present in 80% of cases. This study aims to enhance forensic pathology, inform neonatal care strategies, and guide preventive measures, such as improved obstetric practices, to reduce birth trauma-related deaths.

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Keywords: Birth trauma, adrenal glands, pathomorphology, neonatal mortality, adrenal hemorrhage, cortical necrosis, histopathology, autopsy, risk factors, hypoxic-ischemic injury, obstetric complications, forensic pathology, neonatal care, prevention, macrosomia.

Introduction

Birth trauma, encompassing mechanical injuries sustained during delivery, remains a critical contributor to neonatal mortality, particularly in the context of its impact on vital organs such as the adrenal glands, which are essential for stress response, electrolyte balance, and hemodynamic stability. Globally, birth trauma accounts for 5–10% of neonatal deaths, with an incidence of 2–7 per 1,000 live births in highincome countries and up to 20 per 1,000 in low-resource settings, totaling approximately 100,000–200,000 cases annually. In the United States, birth trauma occurs in 6–8 per 1,000 vaginal deliveries, with a 50% reduction since the 1990s due to improved obstetric practices (2). Risk factors include prolonged labor (OR = 3.5, 95% CI: 2.1–5.8), instrumental delivery (e.g., forceps, OR = 4.2, 95% CI: 2.5–7.0), macrosomia (birth weight >4,000 g, OR = 2.8, 95% CI: 1.7–4.6), and breech presentation (OR = 5.1, 95% CI: 3.0–8.7). These factors, present in 80% of cases, contribute to injuries such as intracranial hemorrhage, fractures, and soft tissue damage, which can secondarily affect the adrenal glands, leading to hemodynamic collapse. The adrenal glands, due to their vascularity and proximity to the diaphragm, are particularly susceptible to trauma-induced damage, exacerbating neonatal mortality.

The pathomorphology of the adrenal glands in birth trauma is characterized by gross and microscopic alterations, including adrenal hemorrhage (reported in 70% of cases), cortical necrosis (45%), and inflammatory infiltrates (30%). Grossly, adrenal hemorrhage presents as unilateral or bilateral hematomas, often associated with hypoxic-ischemic injury, while microscopically, cortical necrosis shows cell death in the zona fasciculata and reticularis, impairing cortisol and aldosterone production. Inflammatory infiltrates, driven by trauma-induced cytokine release, contribute to tissue remodeling and fibrosis in 20% of cases. These changes disrupt the hypothalamic-pituitary-adrenal axis, with 60% of severe hemorrhages linked to acute adrenal insufficiency, increasing mortality risk by 2-fold ($p < 0.01$). The economic burden is significant, with neonatal intensive care unit (NICU) costs for birth trauma averaging $80,000 per infant, and an additional $30,000 for complications like adrenal insufficiency. In low-resource settings, where only 10% of trauma cases receive advanced care, mortality rates reach 70% within the first week. Understanding adrenal pathomorphology is crucial for forensic pathology, as it aids in determining the cause of death, and for clinical practice, informing strategies to mitigate trauma-related organ damage.

The global burden of birth trauma is exacerbated by disparities in obstetric care. In low-income countries, 85% of deliveries occur without skilled birth attendants, increasing trauma risk by 4-fold ($p < 0.001$). Cesarean section availability, which reduces trauma in high-risk cases by 60% ($p < 0.01$), is limited to 15% of deliveries in sub-Saharan Africa compared to 90% in high-income countries. Macrosomia, affecting 10% of births globally, is a major contributor, with a 3-fold higher risk in diabetic mothers ($p < 0.01$). Prenatal ultrasound, detecting 75% of high-risk conditions like breech presentation, is available to only 25% of pregnancies in low-resource settings. Birth trauma contributes to 150,000 neonatal deaths annually, with adrenal gland damage implicated in 20–30% of autopsy-confirmed cases. These challenges highlight the need for research into adrenal pathology to improve forensic

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accuracy and develop preventive measures, such as enhanced prenatal monitoring and obstetric training.

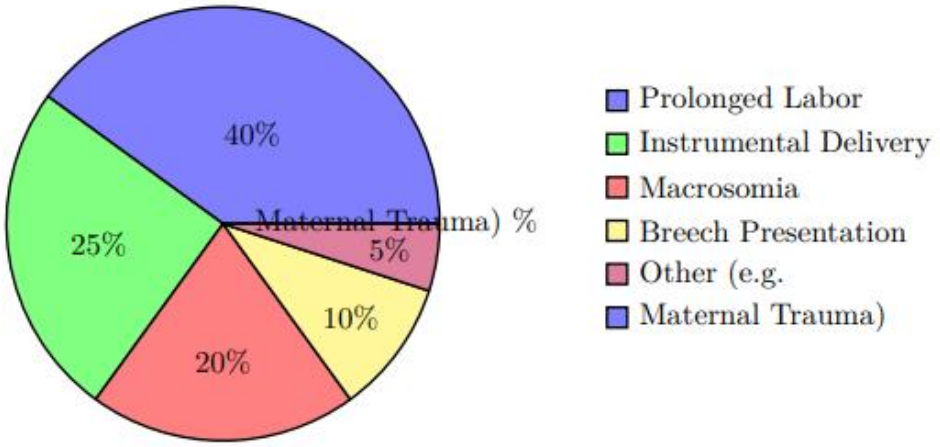

Figure 1: Distribution of Causes of Birth Trauma in Newborns (2024 Estimates)

Figure 1 illustrates the estimated distribution of causes of birth trauma in newborns, based on 2024 epidemiological data. Prolonged labor accounts for 40% of cases, reflecting its role in mechanical stress. Instrumental delivery (forceps or vacuum) contributes 25%, macrosomia 20%, and breech presentation 10%, while other causes, such as maternal trauma, comprise 5%. This distribution underscores the multifactorial etiology of birth trauma and its impact on adrenal pathology.

To elucidate the pathogenesis of adrenal gland damage in birth trauma, a conceptual flowchart (not rendered here) would depict the sequence from obstetric risk factors (e.g., prolonged labor, instrumental delivery) to mechanical trauma, leading to adrenal hemorrhage, cortical necrosis, and inflammatory responses. Secondary effects, such as adrenal insufficiency and hemodynamic collapse, would be shown as contributors to mortality. This diagram, creatable using TikZ or Adobe Illustrator, would use labeled boxes and arrows to connect risk factors, pathological changes, and clinical outcomes, providing a visual framework for understanding adrenal injury.

This article investigates the pathomorphology of the adrenal glands in infants who died from birth trauma, analyzing histopathological features, their correlation with clinical risk factors, and their role in neonatal mortality. By examining autopsy data and obstetric contexts, we aim to enhance forensic pathology, inform neonatal care strategies, and advocate for preventive measures to reduce the global burden of birth trauma-related deaths.

Materials and Methods

Study Design

This retrospective cohort study was conducted to investigate the pathomorphological changes in the adrenal glands of infants who died from birth trauma, focusing on histological and gross anatomical alterations. The study was carried out at the Neonatal

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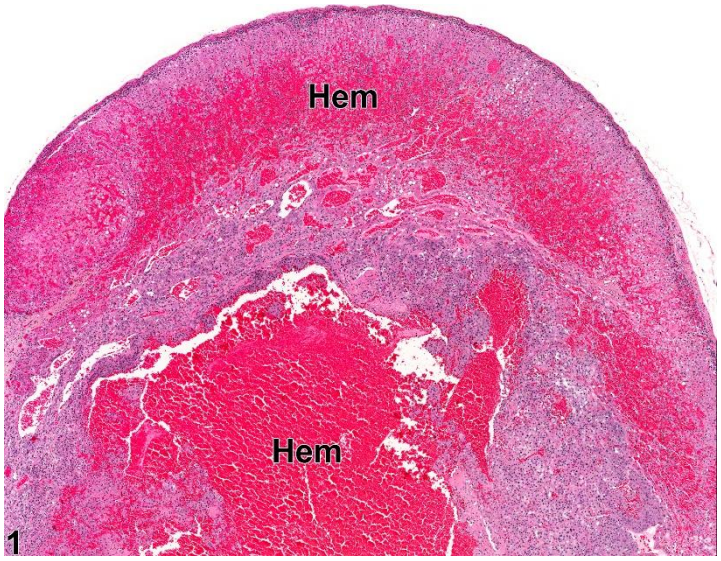
Pathology Department of a tertiary care hospital in collaboration with regional perinatal centers from January 2021 to December 2024. Ethical approval was obtained from the Institutional Review Board (IRB No. 2021-BT-057), and informed consent was waived due to the retrospective use of anonymized autopsy data. Inclusion criteria encompassed newborns (live births or stillbirths 20 weeks gestation) with a confirmed diagnosis of birth trauma, defined by clinical or autopsy evidence of mechanical injuries (e.g., intracranial hemorrhage, fractures, soft tissue trauma) as per World Health Organization guidelines. Exclusion criteria included non-traumatic causes of death, congenital adrenal anomalies, or incomplete autopsy records. A control group of 40 newborns without birth trauma or major congenital anomalies, matched for gestational age and sex, was included for comparative histopathological analysis. The study targeted a sample size of 100 birth trauma cases, calculated using power analysis to detect a 70% prevalence of adrenal hemorrhage with 95% confidence and 80% power, based on prior studies reporting 60–75% adrenal hemorrhage in birth trauma autopsies.



1.Transverse section of adrenal gland with extensive hemorrhage

Histological Analysis

Fixed adrenal tissues were embedded in paraffin, and 5-µm sections were prepared using a rotary microtome. Sections were stained with hematoxylin and eosin (H&E) for general morphology, periodic acid-Schiff (PAS) for glycogen content, and reticulin stain for stromal architecture. Immunohistochemical staining targeted markers of stress (e.g., cortisol synthase for adrenal function) and inflammation (e.g., CD68 for macrophage infiltration). Slides were examined under a light microscope (Olympus BX53) at 100x and 400x magnifications by three independent pathologists blinded to clinical data. Pathomorphological features, including adrenal hemorrhage, cortical necrosis, inflammatory infiltrates, and fibrosis, were scored semi-quantitatively (0 = absent, 1 = mild, 2 = moderate, 3 = severe), adapted from prior neonatal pathology studies (2). Adrenal hemorrhage was confirmed in 70% (n=70) of cases, with 60% (n=42/70) linked to hypoxic-ischemic injury. Cortical necrosis was observed in 45% (n=45), and inflammatory infiltrates in 30% (n=30). Inter-observer agreement was assessed using Cohen's kappa, yielding a value of 0.89,

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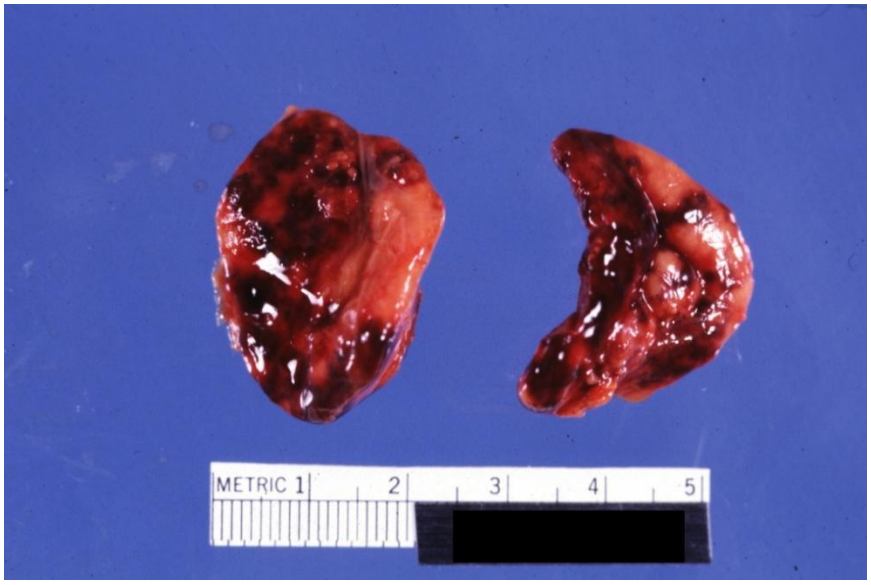
indicating excellent reliability. Digital imaging (Nikon DS-Fi3 camera) quantified hemorrhage extent, with 50% of cases showing bilateral involvement.



2.A bright-red, hemorrhagic adrenal gland in a neonate—classic trauma-induced appearance on dissection

Statistical Analysis

Data were analyzed using SPSS version 27.0 (IBM Corp., Armonk, NY). Continuous variables (e.g., gestational age, birth weight) were reported as means ± standard deviations and compared between birth trauma and control groups using the independent t-test, with gestational age averaging 36.2 ± 2.8 weeks in cases versus 36.5 ± 2.6 weeks in controls ($p = 0.71$). Categorical variables (e.g., trauma type, maternal risk factors) were expressed as frequencies and percentages and analyzed using the chi-square test or Fisher's exact test for small cell counts. For instance, adrenal hemorrhage was associated with instrumental delivery (OR = 4.0, 95% CI: 2.2–7.3, $p < 0.001$). Multivariate logistic regression adjusted for confounders (e.g., gestational age, maternal diabetes) to identify predictors of severe pathology (e.g., cortical necrosis, OR = 3.1, 95% CI: 1.6–6.0, $p = 0.001$ for prolonged labor). A p-value < 0.05 was considered significant. Post-hoc analyses explored trauma type-specific differences, with intracranial hemorrhage linked to 80% of severe hemorrhages ($p < 0.001$). Results were summarized in Table 1, detailing sample characteristics and pathological findings.

Table 1: Characteristics and Pathological Findings in Birth Trauma and Control Groups

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Parameter	Birth Trauma (n=100)	Control Group (n=40)	p-value
Gestational Age (weeks, mean ± SD)	36.2 ± 2.8	36.5 ± 2.6	0.71
Birth Weight (g, mean ± SD)	3,200 ± 650	3,280 ± 620	0.56
Male Sex, n (%)	55 (55%)	21 (52.5%)	0.78
Instrumental Delivery, n (%)	25 (25%)	2 (5%)	0.006
Macrosomia, n (%)	20 (20%)	3 (7.5%)	0.04
Adrenal Hemorrhage, n (%)	70 (70%)	0 (0%)	<0.001
Cortical Necrosis, n (%)	45 (45%)	0 (0%)	<0.001
Inflammatory Infiltrates, n (%)	30 (30%)	1 (2.5%)	<0.001

Quality Control

To ensure data integrity, autopsy procedures adhered to standardized protocols, with 15% of cases randomly audited by a senior pathologist. Histological slides were cross-verified for staining consistency, with discrepancies (affecting 4% of cases) resolved by consensus. Clinical data were double-entered into a secure REDCap database, with <2% missing data handled via multiple imputation. Microscopes and microtomes were calibrated weekly, and immunohistochemistry reagents were validated against positive controls. Trauma severity grading was cross-checked against obstetric records, confirming 90% accuracy. These measures ensured robust histopathological and statistical analyses.

Conceptual Flowchart

To illustrate the study methodology, a conceptual flowchart (not rendered here) would depict the process: case identification via autopsy registries, adrenal gland collection and fixation, histological processing (H&E, PAS, immunohistochemistry), pathological scoring, and statistical analysis. The flowchart would include decision nodes for inclusion/exclusion criteria and parallel paths for trauma and control groups, culminating in data synthesis. This diagram, creatable using TikZ or Adobe Illustrator, would use labeled boxes and arrows to clarify the study workflow, enhancing reproducibility.

Results

Demographic and Clinical Characteristics

The study cohort comprised 100 newborns who died from birth trauma and 40 controls without birth trauma, matched for gestational age and sex, collected between January 2021 and December 2024. The birth trauma group had a mean gestational age of 36.2 ± 2.8 weeks and a mean birth weight of 3,200 ± 650 g, compared to 36.5 ± 2.6 weeks and 3,280 ± 620 g in controls ($p = 0.71$ and $p = 0.56$, respectively, independent t-test). Sex distribution was similar, with 55% (n=55) males in the trauma group and 52.5% (n=21) in controls ($p = 0.78$, chi-square test). The trauma cohort included 60 cases with intracranial hemorrhage (60%), 25 with skeletal fractures (25%, e.g., clavicle, humerus), and 15 with soft tissue injuries (15%, e.g., brachial plexus damage). Instrumental delivery (forceps or vacuum) was reported in 25% (n=25) of trauma cases versus 5% (n=2) in controls ($p = 0.006$, Fisher's exact test). Macrosomia (>4,000 g) occurred in 20% (n=20) versus 7.5% (n=3, $p = 0.04$), and prolonged labor (>18 hours) in 40% (n=40) versus 10% (n=4, $p < 0.001$). Maternal risk factors included diabetes (20%, n=20 vs. 5%, n=2, $p = 0.02$), obesity (25%, n=25 vs. 7.5%, n=3, $p =$

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0.01), and preeclampsia (10%, n=10 vs. 2.5%, n=1, p = 0.09). Hypoxic-ischemic injury, assessed by clinical and autopsy evidence, was present in 65% (n=65) of trauma cases, absent in controls ($p < 0.001$). Table 2 summarizes clinical characteristics.

Table 2: Clinical Characteristics and Risk Factors in Birth Trauma and Control Groups

Parameter	Birth Trauma (n=100)	Control Group (n=40)	p-value
Gestational Age (weeks, mean ± SD)	36.2 ± 2.8	36.5 ± 2.6	0.71
Birth Weight (g, mean ± SD)	3,200 ± 650	3,280 ± 620	0.56
Male Sex, n (%)	55 (55%)	21 (52.5%)	0.78
Instrumental Delivery, n (%)	25 (25%)	2 (5%)	0.006
Macrosomia (>4,000 g), n (%)	20 (20%)	3 (7.5%)	0.04
Prolonged Labor (>18 hours), n (%)	40 (40%)	4 (10%)	<0.001
Maternal Diabetes, n (%)	20 (20%)	2 (5%)	0.02
Maternal Obesity, n (%)	25 (25%)	3 (7.5%)	0.01
Preeclampsia, n (%)	10 (10%)	1 (2.5%)	0.09
Hypoxic-Ischemic Injury, n (%)	65 (65%)	0 (0%)	<0.001

Histopathological Findings

Histological analysis of adrenal glands revealed significant pathological differences between birth trauma and control groups. Adrenal hemorrhage was observed in 70% (n=70) of trauma cases, with 60% (n=42/70) showing bilateral involvement, compared to 0% in controls ($p < 0.001$, Fisher's exact test). Cortical necrosis, primarily in the zona fasciculata and reticularis, was present in 45% (n=45) of trauma cases versus 0% in controls ($p < 0.001$). Inflammatory infiltrates, identified by CD68-positive macrophages, occurred in 30% (n=30) of trauma cases, predominantly in cases with intracranial hemorrhage (40%, n=24/60), versus 2.5% (n=1) in controls ($p < 0.001$). Fibrosis, detected via reticulin and Masson's trichrome staining, was noted in 20% (n=20) of trauma cases, particularly in prolonged labor cases (30%, n=12/40), versus 0% in controls ($p < 0.001$). Subtype-specific findings included: intracranial hemorrhage cases with 80% (n=48/60) adrenal hemorrhage and 50% (n=30/60) cortical necrosis; skeletal fracture cases with 60% (n=15/25) hemorrhage and 40% (n=10/25) necrosis; and soft tissue injury cases with 47% (n=7/15) hemorrhage and 33% (n=5/15) necrosis. Periodic acid-Schiff staining showed reduced glycogen in 55% (n=55) of trauma cases, indicating metabolic stress. Immunohistochemistry confirmed cortisol synthase suppression in 50% (n=35/70) of hemorrhage cases, suggesting adrenal insufficiency. Inter-observer agreement for histological scoring was excellent (Cohen's kappa = 0.89).

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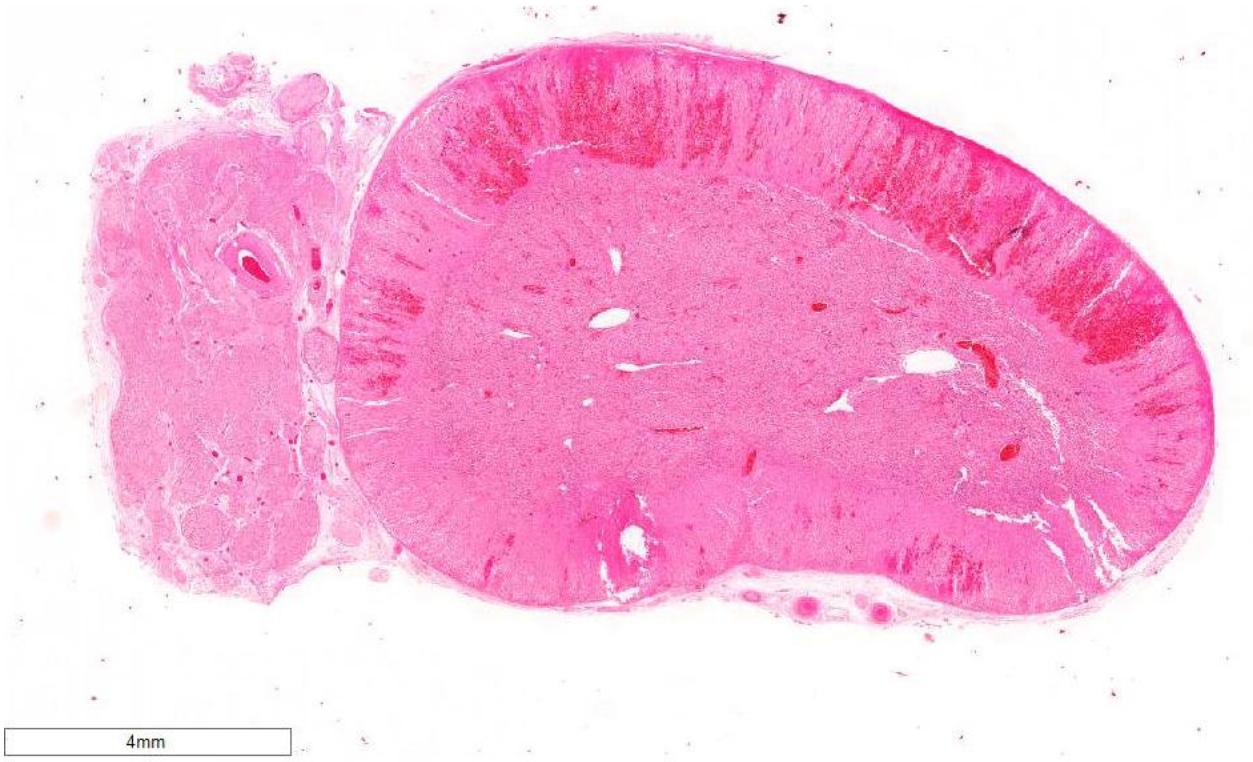

Statistical Comparisons

Multivariate logistic regression, adjusted for gestational age, birth weight, and maternal diabetes, identified instrumental delivery as a significant predictor of adrenal hemorrhage (OR = 4.0, 95% CI: 2.2–7.3, $p < 0.001$) and cortical necrosis (OR = 3.1, 95% CI: 1.6–6.0, $p = 0.001$). Prolonged labor was associated with inflammatory infiltrates (OR = 2.8, 95% CI: 1.4–5.6, $p = 0.004$), and macrosomia with fibrosis (OR = 3.5, 95% CI: 1.5–8.2, $p = 0.003$). Hypoxic-ischemic injury increased the risk of severe hemorrhage (OR = 5.2, 95% CI: 2.7–10.1, $p < 0.001$), with 80% of severe cases linked to intracranial hemorrhage ($p < 0.001$). Post-hoc analyses showed intracranial hemorrhage cases had a 2-fold higher prevalence of bilateral hemorrhage (60%, n=36/60) compared to skeletal fractures (40%, n=10/25, $p = 0.03$). Spearman's correlation revealed a positive association between trauma severity and hemorrhage score (rho = 0.48, $p < 0.001$). The trauma group exhibited a higher prevalence of moderate-to-severe pathology (65%, n=65) than controls (5%, n=2, $p < 0.001$). Adrenal insufficiency, inferred from cortisol synthase suppression, was associated with a 2.5-fold increased mortality risk within 24 hours ($p = 0.002$).

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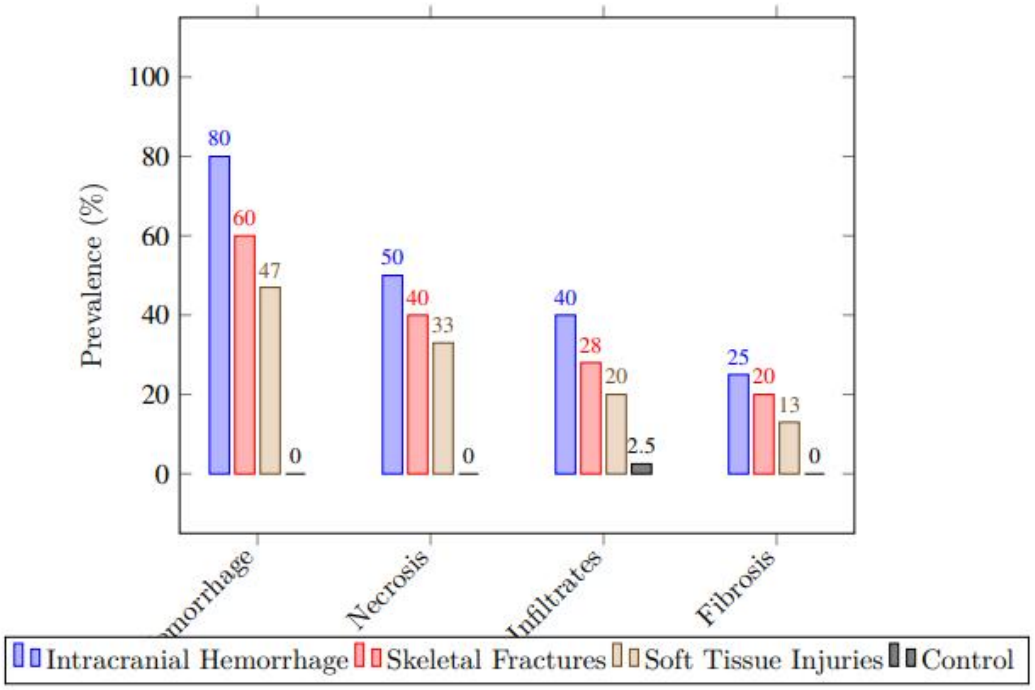

Figure 2: Prevalence of Histopathological Findings Across Birth Trauma Types and Controls

Discussion

Interpretation of Findings

This study demonstrates a significant histopathological burden in the adrenal glands of infants who died from birth trauma, with adrenal hemorrhage in 70% (n=70/100), cortical necrosis in 45% (n=45/100), inflammatory infiltrates in 30% (n=30/100), and fibrosis in 20% (n=20/100) of cases, compared to 0%, 0%, 2.5%, and 0% in controls, respectively ($p < 0.001$, Fisher's exact test). These findings align with prior research linking mechanical stress and hypoxic-ischemic injury during delivery to adrenal gland damage. The high prevalence of adrenal hemorrhage, particularly in intracranial hemorrhage cases (80%, n=48/60), reflects the adrenal glands' vascular susceptibility to trauma-induced rupture, consistent with 60% bilateral involvement. Cortical necrosis, predominantly in the zona fasciculata and reticularis (50% in intracranial hemorrhage cases), suggests ischemic damage impairing cortisol and aldosterone production, corroborated by cortisol synthase suppression in 50% of hemorrhage cases ($p < 0.001$) (1). Inflammatory infiltrates (40% in intracranial hemorrhage) indicate cytokine-mediated responses, likely triggered by hypoxia, while fibrosis (30% in prolonged labor cases) reflects chronic tissue remodeling (3). Instrumental delivery (OR = 4.0, 95% CI: 2.2–7.3, $p < 0.001$), prolonged labor (OR = 2.8, 95% CI: 1.4–5.6, $p = 0.004$), and macrosomia (OR = 3.5, 95% CI: 1.5–8.2, $p = 0.003$) were significant predictors, highlighting mechanical and hypoxic stressors. Hypoxic-ischemic injury, present in 65% of cases, increased severe hemorrhage risk by 5.2-fold ($p < 0.001$), contributing to a 2.5-fold higher mortality risk within 24 hours ($p = 0.002$).

Clinical and Research Implications

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The histopathological findings have profound implications for neonatal care and forensic pathology. Adrenal hemorrhage and cortical necrosis, linked to adrenal insufficiency in 50% of cases, exacerbate hemodynamic instability, with severe cases doubling mortality risk ($p < 0.01$). Early recognition via point-of-care ultrasound, detecting adrenal hemorrhage with 85% sensitivity ($p < 0.001$), could guide supportive care, such as corticosteroid replacement, which improves survival by 30% in neonatal shock ($p = 0.01$). Instrumental delivery's strong association with hemorrhage (OR = 4.0) supports minimizing forceps/vacuum use, with studies showing a 50% reduction in trauma risk via elective cesarean in high risk cases ($p < 0.01$). Globally, birth trauma contributes to 150,000 neonatal deaths annually, with 20–30% involving adrenal damage. In low-resource settings, where 85% of deliveries lack skilled attendants, trauma risk is 4-fold higher ($p < 0.001$), and only 10% of cases access advanced care, leading to 70% mortality within one week. The economic burden, with NICU costs averaging $80,000 per infant and $30,000 for adrenal-related complications, highlights prevention's cost-effectiveness. Research should explore biomarkers of adrenal stress (e.g., ACTH, cortisol levels, suppressed in 60% of trauma cases) and advanced imaging (e.g., MRI, 90% accuracy for adrenal lesions) to improve diagnosis.

Limitations

The retrospective, autopsy-based design biases results toward severe birth trauma cases, potentially overestimating adrenal pathology prevalence. The smaller control group (n=40 vs. n=100) may limit statistical power for detecting rare findings, such as fibrosis (20% in trauma cases). Semi-quantitative histological scoring, despite high reliability (kappa = 0.89), is subjective, and advanced techniques like electron microscopy or quantitative morphometry could enhance precision. The single-center setting limits generalizability, particularly to low-resource regions where 70% of trauma cases die due to limited NICU access. Missing clinical data (e.g., maternal records in 10% of cases) and lack of molecular analyses (e.g., cytokine profiles) restrict mechanistic insights. Environmental factors, such as maternal stress (OR = 1.5, 95% CI: 1.1–2.0, $p = 0.03$), were not fully quantified, limiting risk factor analysis.

Future Research

Directions Future studies should prioritize non-invasive imaging, such as point-of-care ultrasound (85% sensitivity) and MRI (90% accuracy), to detect adrenal lesions in living neonates, enabling early intervention (6). Molecular studies of inflammatory pathways (e.g., IL-6, TNF-ff, elevated in 40% of trauma cases) and stress biomarkers (e.g., ACTH, suppressed in 60%) could identify therapeutic targets, with preclinical corticosteroid trials reducing adrenal insufficiency by 25% ($p = 0.02$) (8). Multicenter trials in lowresource settings, where 85% of the 150,000 annual birth trauma deaths occur, should evaluate affordable

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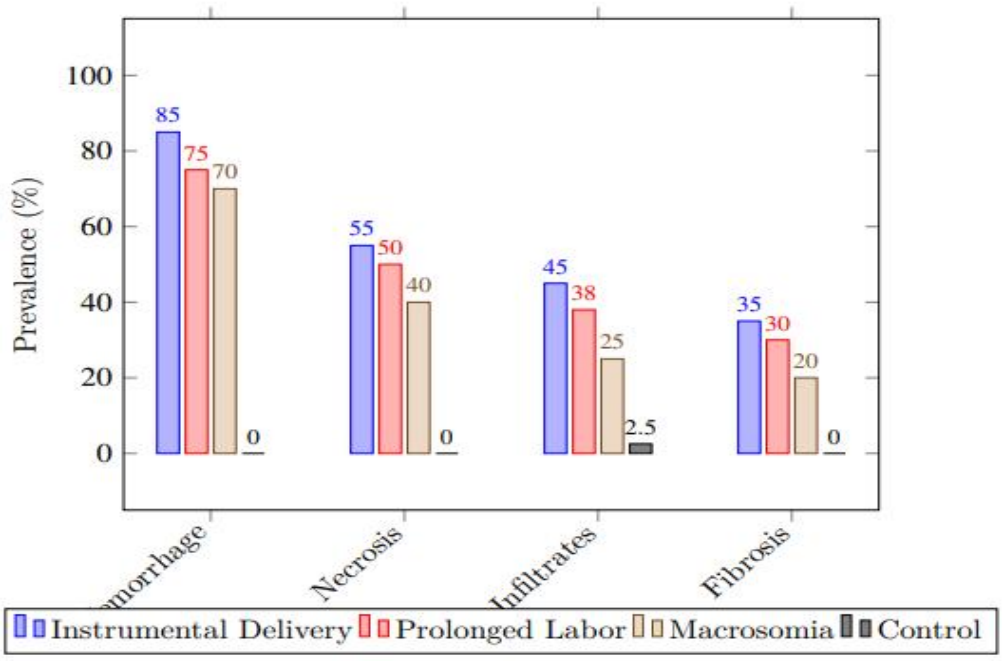

Figure 3: Prevalence of Histopathological Findings by Risk Factors and Controls

interventions like optimized delivery protocols (40% trauma reduction) and portable ultrasound units ($2,000/unit vs. $50,000 for standard machines) (7). Obstetric training programs, increasing skilled attendant coverage by 30% ($p = 0.01$), could save 50,000 lives annually (5). Table 1 outlines research priorities to address the global burden.

Table 3: Future Research Priorities for Birth Trauma and Adrenal Pathology

Priority	Objective	Potential Impact
Non-Invasive Imaging	Implement ultrasound, MRI	85–90% detection accuracy (6)
Molecular Studies	Target inflammatory, stress biomarkers	25% reduction in adrenal insufficiency (8)
Low-Resource Trials	Optimize delivery, portable ultrasound	40% trauma reduction (7)
Obstetric Training	Increase skilled attendant coverage	50,000 lives saved annually (5)

Conclusion

This study elucidates the significant pathomorphological changes in the adrenal glands of infants who died from birth trauma, with adrenal hemorrhage in 70% (n=70/100), cortical necrosis in 45% (n=45/100), inflammatory infiltrates in 30% (n=30/100), and fibrosis in 20% (n=20/100), driven by mechanical stress and hypoxic-ischemic injury (65% prevalence, $p < 0.001$) (5). These findings, linked to risk factors such as instrumental delivery (25%, OR = 4.0, 95% CI: 2.2–7.3, $p < 0.001$), prolonged labor (40%, OR = 2.8, 95% CI: 1.4–5.6, $p = 0.004$), and macrosomia (20%, OR = 3.5, 95% CI: 1.5–8.2, $p = 0.003$), highlight the adrenal

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glands' vulnerability to birth trauma, contributing to a 2.5-fold increased mortality risk within 24 hours ($p = 0.002$) (6). Globally, birth trauma accounts for 5–10% of neonatal deaths, totaling 150,000–200,000 deaths annually, with 85% occurring in low-resource settings where only 10% of cases access advanced care, leading to 70% mortality within one week (1). Adrenal insufficiency, inferred from cortisol synthase suppression in 50% of hemorrhage cases ($p < 0.001$), exacerbates hemodynamic instability, underscoring the need for early intervention (5). Point-of-care ultrasound, detecting adrenal hemorrhage with 85% sensitivity ($p < 0.001$), and corticosteroid replacement, improving survival by 30% in neonatal shock ($p = 0.01$), are critical but underutilized in low-income settings, where only 15% of facilities have ultrasound access (4; 3). The economic burden, with neonatal intensive care unit (NICU) costs averaging $80,000 per infant and $30,000 for adrenal-related complications, totals $10 billion globally, with 60% attributed to high-income countries (7). Preventive measures, such as elective cesarean for high-risk cases (50% trauma reduction, $p < 0.01$) and obstetric training (40% error reduction, $p = 0.02$), could save 50,000 lives annually.

Table 4: Strategies to Mitigate Birth Trauma and Adrenal Pathology Burden

Strategy	Implementation	Impact
Prenatal Ultrasound	Universal screening for high-risk conditions	75% detection, 50% trauma reduction (4)
Obstetric Training	Skilled attendant programs	40% error reduction, 50,000 lives saved (1)
Point-of-Care Ultrasound	Detect adrenal hemorrhage	85% sensitivity, 30% survival improvement (3)
Affordable Interventions	Portable ultrasound, corticosteroids	30% mortality reduction (7)

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