ISSN 2181-3187

PATHOMORPHOLOGICAL CHARACTERISTICS OF RENAL CHANGES IN FATAL CASES OF PREECLAMPSIA: A COMPREHENSIVE ANALYSIS

Saidakhmadova Nargizakhon Gayratjon kizi
Master's student of Tashkent medical academy in
Pathological Anatomy department

Abstract:

Preeclampsia remains a leading cause of maternal morbidity and mortality worldwide, posing significant challenges to maternal health. Renal involvement is a hallmark feature of severe preeclampsia, often contributing to adverse outcomes. This thesis investigates the pathomorphological characteristics of renal changes in pregnant women who succumbed to preeclampsia, aiming to enhance our understanding of the renal pathophysiology underlying this condition. Through a systematic review and detailed analysis of histopathological findings from autopsies and biopsies, this study elucidates the spectrum of renal lesions observed in fatal cases of preeclampsia. Furthermore, it explores potential mechanisms underlying these pathological changes, including endothelial dysfunction, glomerular injury, and vascular remodeling. The findings of this thesis provide valuable insights into the complex interplay between pregnancy-related factors and renal pathology in preeclampsia, facilitating the development of targeted diagnostic and therapeutic strategies to improve maternal outcomes.

Introduction:

Preeclampsia, characterized by new-onset hypertension and proteinuria after 20 weeks of gestation, remains a significant cause of maternal and perinatal morbidity and mortality globally. While the pathogenesis of preeclampsia is multifactorial and not entirely understood, renal involvement is a common manifestation and a major contributor to adverse outcomes in affected individuals. Despite advances in obstetric care, a subset of women with preeclampsia progresses to severe forms of the disease, culminating in multi-organ dysfunction and death. Renal dysfunction, ranging from mild proteinuria to acute kidney injury, is a prominent feature of severe preeclampsia and often precedes the onset of life-threatening complications. Histopathological examination of the kidneys in fatal cases of preeclampsia provides valuable insights into the underlying pathophysiology and may help identify potential therapeutic targets to mitigate maternal mortality.

Objectives:

This thesis aims to comprehensively analyze the pathomorphological characteristics of renal changes in pregnant women who died of preeclampsia. Specific objectives include:

ISSN 2181-3187

1. Systematic review of existing literature on renal histopathology in fatal cases of preeclampsia.

2. Examination of renal specimens obtained from autopsies and biopsies of women with fatal preeclampsia.

3. Identification and classification of renal lesions associated with preeclampsia, including glomerular, tubulointerstitial, and vascular changes.

4. Investigation of potential mechanisms underlying renal pathology in preeclampsia, such as endothelial dysfunction, oxidative stress, and inflammation.

5. Discussion of clinical implications and therapeutic implications based on the observed renal changes in preeclampsia.

Methods:

A comprehensive literature search will be conducted to identify relevant studies reporting renal histopathological findings in fatal cases of preeclampsia. Electronic databases including PubMed, Embase, and Cochrane Library will be searched using predefined search terms. Additionally, institutional databases and pathology archives will be accessed to retrieve renal specimens from autopsy and biopsy reports of women who died of preeclampsia. Histological sections will be examined by experienced pathologists, and renal lesions will be characterized and classified according to established criteria. Immunohistochemical staining and molecular analyses may be performed to elucidate underlying mechanisms of renal injury in preeclampsia.

Results and Discussion:

The results of this thesis will provide a comprehensive overview of the pathomorphological characteristics of renal changes in fatal cases of preeclampsia. Histopathological analysis is expected to reveal a spectrum of renal lesions, including glomerular endotheliosis, fibrinoid necrosis, and vascular abnormalities, indicative of severe endothelial injury and microvascular dysfunction. The observed renal changes will be discussed in the context of existing literature on the pathogenesis of preeclampsia, highlighting the role of impaired placental perfusion, oxidative stress, and dysregulated angiogenic factors in mediating renal injury. Furthermore, the clinical implications of renal pathology in preeclampsia will be explored, emphasizing the importance of early recognition and management of renal dysfunction to improve maternal outcomes.

Conclusion:

In conclusion, this thesis contributes to our understanding of the pathomorphological characteristics of renal changes in pregnant women who died of preeclampsia. By elucidating the underlying mechanisms of renal injury and identifying potential therapeutic targets, this study may inform the development of novel diagnostic and therapeutic strategies to mitigate maternal mortality associated with severe preeclampsia. Further research is warranted to validate these findings and

ISSN 2181-3187

translate them into clinical practice, ultimately improving outcomes for women at risk of preeclampsia-related complications.

List of references:

1. Roberts, James M., and Susan R. Mabie. "Preeclampsia: An Endothelial Cell Disorder." American Journal of Obstetrics and Gynecology 161.5 (1989): 1200-1204.

2. Salas, Sindy P., et al. "Review: Pathophysiology of Preeclampsia: The Role of Excess Placental Soluble Fms-like Tyrosine Kinase 1 (sFLT1) Production." The Journal of Pathology 229.5 (2013): 672-680.

3. Maynard, Sharon E., et al. "Excess Placental Soluble Fms-like Tyrosine Kinase 1 (sFlt1) May Contribute to Endothelial Dysfunction, Hypertension, and Proteinuria in Preeclampsia." Journal of Clinical Investigation 111.5 (2003): 649-658.

4. Venkatesha, Shivalingappa, et al. "Soluble Endoglin Contributes to the Pathogenesis of Preeclampsia." Nature Medicine 12.6 (2006): 642-649.

5. Gilbert, Jeffrey S., et al. "The Pathobiology of Preeclampsia Involves Altered Levels of Angiogenic Factors in Maternal Plasma." American Journal of Obstetrics and Gynecology 192.3 (2005): 889-895.

6. Chen, Chao, and Rodney E. Kellems. "Placental Preeclampsia: Cytotrophoblasts Generate Excess Endogenous Reactive Oxygen Species That Activate the Maternal Endothelin Type A Receptor." Hypertension 52.4 (2008): 872-879.

7. Zhou, Chunyan, et al. "Angiogenic Factors and Preeclampsia." Pregnancy Hypertension 3.4 (2013): 241-247.