# Study of Acute Kidney Injury Secondary to Obstetric Complications in the Third Trimester of Pregnancy and the Puerperium

**Thesis**

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Abd El-haleem Mohammed

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# List of Abbreviations

| Abb. | Full term |
|---|---|
| **ACE** | Angiotensin converting enzyme |
| **ACEIs** | Angiotensin converting enzyme inhibitors. |
| **aCL** | Anti cardiolipin |
| **ACR** | Albumin to Creatinine ratio |
| **ADAMTS13** | Adisintegrin and metalloproteinase with thrombospondin type 1 motives |
| **ADH** | Anti diuretic hormone. |
| **AFLP** | Acute Fatty Liver of Pregnancy |
| **AHT** | Arterial hypertension |
| **AIDS** | Acquired immunedeficiency syndrome |
| **AKI** | Acute kidney injury |
| **AKIN** | Acute kidney injury network |
| **ANA** | Anti nuclear antibody |
| **ANCA** | Anti-neutrophil cytoplasmic antibody |
| **Anti-GBM** | Anti glomerular basement membrane |
| **APS** | Antiphospholipid Syndrome |
| **aPTT** | Activated partial thromboplastin time |
| **ARBs** | Angiotensin receptor blockers |
| **ARDS** | Adult respiratory distress syndrome |
| **ATN** | Acute tubular necrosis |
| **BTx** | Blood transfusion |
| **BUN** | Blood urea nitrogen |
| **CAPS** | Catastrophic antiphospholipid syndrome |
| **CBC** | Complete Blood Count |
| **CD94** | Cluster of Differentiation 94 |
| **CE** | Calcium excretion |
| ***CFB*** | Complement factor B |
| ***CFH*** | Complement factor H |
| **CFHR** | complement factor H-related genes |

| Abb. | Full term |
|---|---|
| ***CFI*** | Complement factor I |
| **CKD** | Chronic kidney disease |
| **CT** | Computed Tomography |
| **DDAVP** | Desmopressin acetate |
| **DIC** | Disseminated intravascular coagulopathy |
| **ESRD** | End stage renal disease |
| **EVT** | Extravillous trophoblast |
| **FENa** | Fractional excretion of Na |
| **FFP** | Fresh frozen plasma |
| **GFR** | Glomerular filtration rate |
| **Hct** | Hematocrit. |
| **HD** | Haemodialysis |
| **HIT** | Heparin-induced thrombocytopenia |
| **HIV** | Human immunedeficiency virus |
| **HLA** | Human leukocytic antigen |
| **HUS** | Hemolytic uremic syndrome |
| **ILT** | Immunoglobulin-like transcript |
| **KDIGO** | Kidney Disease Improving Global Outcomes |
| **KIM-1** | Kidney injury molecule 1 |
| **KIR** | Killer Immunoglobulin-like Receptors |
| **LA** | Lupus anticoagulant |
| **LDH** | Serum lactate dehydrogenase |
| **LGA** | Large for gestational age |
| **MCP** | Membrane cofactor protein |
| **MI** | Myocardial infarction |
| **NGAL** | Human neutrophil gelatinase-associated lipocalin |
| **NK cells** | Natural killer cells |
| **NSAIDs** | Non-steroidal anti-inflammatory drugs. |
| **P/C ratio** | Protein to creatinine ratio |

| Abb. | Full term |
|---|---|
| **PEx** | Plasma exchange |
| **PIGF** | Placental induced growth factor |
| **PIH** | Pregnancy induced hypertension. |
| **PN** | Pyelonephritis |
| **PPH** | Postpartum haemorrage |
| **PRAKI** | Pregnancy related acute kidney injury |
| **PRES** | post reversible leukoencephalopathy syndrome |
| **PT** | Prothrombin time |
| **PTH** | Parathyroid hormone |
| **P-TMA** | Pregnancy-associated thrombotic microangiopathy |
| **PV** | Plasma volume |
| **RAAS** | Renin-angiotensin-aldosterone system |
| **RBC** | Red blood cells |
| **RCN** | Renal cortical necrosis |
| **RF** | Rheumatoid factor |
| **RRT** | Renal replacement therapy |
| **Scr** | Serum creatinine |
| **sEndoglin** | Soluble endoglin |
| **sFlt-1** | Soluble fms-like tyrosine kinase-1 |
| **SIADH** | Syndrome of inappropriate antidiuretic hormone |
| **STEC** | Shiga toxin-producing *E. coli* |
| **TLC** | Total leucocytic count |
| **TMG** | Transport maximum for glucose |
| **TTP** | Thrombotic thrombocytopenic purpura |
| **U/P** | Urine-to-plasma ratio |
| **UTI** | Urinary Tract Infections |
| **VEGF** | Vascular endothelial growth factor |
| **VOD** | Veno occlusive disease |
| **WHO** | World Health Organization |

# Study of Acute Kidney Injury Secondary to Obstetric Complications in the Third Trimester of Pregnancy and the Puerperium


## Abstract

**Background:** Acute kidney injury (AKI) includes a group of clinical syndromes that primarily manifest as a rapid decline in the kidney function in association with the accumulation of metabolic waste. **Aim:** The aim of our study is to assess the prevalence and outcome of acute kidney injury secondary to obstetric complications in the third trimester of pregnancy and the puerperium. **Subjects:** study is a retrospective study that was conducted on 80 patients presented with acute kidney injury secondary to obstetric complications in third trimester of pregnancy and puerperium. All pregnant females aged more than 18 years old who experienced an episode of AKI during the third trimester of pregnancy and puerperium. The commonest cause of AKI in the third trimester of pregnancy and puerperium was obstetric haemorrage (50%) followed by hypertensive disorders of pregnancy (35%) including preeclampsia, eclampsia and HELLP then TMA (7.5%) and septic AKI (7.5%). The diagnosis of AKI in pregnancy and the subsequent management usually depend on the clinical evaluation and routine investigations but in some cases the diagnosis is very challenging that calls for renal biopsy (6.3%). AKI in pregnancy is associated with high fetal mortality of (73.8%). **Conclusion:** In developing countries, AKI in pregnancy remains a frequent and grave complication. It reflects the absence of prenatal care and early detection of high-risk pregnancies, the delay in transfer of patients and the paucity of relevant human and material resources. It is certainly preventable and treatable ncomplication, but one that imposes a heavy burden of maternal morbidity and mortality if its diagnosis and treatment are delayed. **Recommendations:** Larger number of patients & multi-centre studies will provide more information about incidence and prevalence of acute kidney injury in pregnancy. Give special attention to pregnant women with preeclampsia or any other risk factors during the pregnancy and during the postpartum period and care with doing routine investigations and document the results for further future studies.



**Keywords:** AKI, HELLP, TMA, pregnancy.

# Introduction

Acute kidney injury (AKI) includes a group of clinical syndromes that primarily manifest as a rapid decline in the kidney function in association with the accumulation of metabolic waste **(Srisawat and Kellum, 2011).**

Obstetric Acute Kidney Injury is a rare and sometimes fatal complication of pregnancy, the incidence of which is declining worldwide, yet which is a matter of concern in developing countries **(Khanal et al., 2012, Prakash et al., 2007).**

Paradoxically the prevalence is increasing in some developed countries, the increase being attributed to hypertensive disorders of pregnancy and their management **(Mehrabadi et al., 2014).**

During the first trimester of gestation, acute kidney injury develops most often due to hyperemesis gravidarum or septic abortion. In the third trimester, the differential diagnosis is more challenging for the obstetrician and the nephrologist **(Machado et al., 2012).**

Obstetric acute kidney injury is associated with poor access to antenatal care, multiparity and rural background **(Archana et al., 2015).**

Early treatment for AKI typically includes optimization of the hemodynamic status and correction of any volume deficit. This will have a salutary effect on kidney function, helping to minimize progression of further kidney injury and potentially facilitating recovery from AKI with aminimization of any residual chronic impairment of kidney function **(Karvellas et al., 2011).**

# Aim of the Work

The aim of our study is to assess the prevalence and outcome of acute kidney injury secondary to obstetric complications in the third trimester of pregnancy and the puerperium.

Chapter (1):

# Changes of Kidney Function during Normal Pregnancy

Within pregnancy, several changes affecting organs and systems occur for the lady to adapt to the new state she is in. This new state afford adequate environment for sustaining fetal life and pregnancy progression. These changes include anatomical changes, physiological adaptation biochemical alterations, endocrinal re-adjustments **(Yanamandra and Edwin, 2012).**

Estrogen and progesterone hormones, first secreted by the ovary and later by the placenta, perform these much changes within different tissues and organs of the pregnant lady. All these changes permit the continuation and progression of pregnancy for both fetus and mother **(Heidemann and McClure, 2003).**

As a result of enormous changes occurring within the kidney of pregnant lady the reference laboratory ranges for different kidney function tests don't apply any more to this situation **(Ashwood and Burtis, 1999).**

Normal physiological changes occurring within renal system during normal gestation include glomerular and tubular functional changes. There is much increase of renal

plasma flow glomerular filtration rate (GFR). This is added to increased kidney size and dilation of pelvicalyceal system of each kidney **(Odutayo and Hladunewich, 2012).**

Increased both metabolic and circulatory loads within normal gestation leads to physiological changes within renal system. Renal system has to get red of metabolic wastes of both mother and fetus and accommodate itself to the hemodynamic changes caused by the increase in blood volume and also the decreased resistance within vascular system tree **(Derricott and Cartwright, 2013).**

Cheung et al reported that every aspect of physiological renal function is affected by normal gestation **(Cheung and Lafayette, 2013).**

Tran has reported that many laboratory values could be considered out of normal range during gestation, if we apply normal non-gestational ranges to these test values. During normal pregnancy increased effective intravascular volume results in lower concentrations and values of most analytes. In spite of this, free active fractions concentrations, such as ionized calcium, remain at the same range values of non-gestational state. Getting increased values of some laboratory test above non-gestational state,