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

#### **Thesis**

Submitted for Partial Fulfillment of Master Degree in Internal Medicine

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## Contents

Subject	age No.
List of Tables	<del></del> I
List of Figures	III
List of Abbreviations	IV
Introduction	1
Aim of the Work	3
Review of Literature	
Chapter (1): Changes of kidney function during	
normal pregnancy	
Chapter (2): Obstetric complications and AKI	
Chapter (3): Prerenal AKI and acute tubular	,
necrosis at third trimester of pregnancy and	
puerperium	38
Chapter (4): Intrinsic renal and postrenal AKI in	
third trimester of pregnancy and puerperium	49
Chapter (5): Guidelines in treatment of AKI	
secondary to obstetric and puerperial	
complications	
Subjects and Methods	
Results	
Discussion	128
Summary	143
Conclusion	147
Recommendations	148
References	149
Arabic Summary	

## Tist of Tables

Table No.	Title	Page No.
	Tables of Review of Literature	
Table (1)	Staging of AKI	15
Table (2)	Aetiological classification of AKI	16
Table (3)	Pregnancy-Related Causes of AKI	19
Table (4)	Conditions that predispose women to sepsis and septic shock syndrome during pregnancy	32
Table (5)	Guidelines for urinary indeces of prerenal azotemia and ARF	48
Table (6)	Classification of hypertensive disorders of pregnancy	50
Table (7)	Severe complications of preeclampsia	52
Table (8)	Reported frequency of signs and symptoms of HELLP syndrome	60
Table (9)	Molecular-based classification of TMA syndromes	64
<b>Table (10)</b>	Frequency and severity of laboratory findings among imitators of preeclampsia-eclampsia	78
	Tables of Results	
<b>Table (11)</b>	Demographic data of patients presented with pregnancy related AKI	109
<b>Table (12)</b>	Different Pregnancy complications that have impact on kidney function	110
<b>Table (13)</b>	Different clinical types of pregnancy related AKI	112

Table No.	Title	Page No.
<b>Table (14)</b>	Obstetric history of pre-eclampsia	113
<b>Table (15)</b>	Types of AKI according to urine output at time of presentation	114
<b>Table (16)</b>	AKI classification according to KDIGO 2012	115
<b>Table (17)</b>	Clinical Assessment of patients at time of presentation	116
<b>Table (18)</b>	Urine analysis of patients at time of presentation.	118
<b>Table (19)</b>	Laboratory investigations of patients at time of presentation.	119
<b>Table (20)</b>	Autoimmune markers of patients.	120
<b>Table (21)</b>	Renal Biopsy done for patients.	121
<b>Table (22)</b>	Duration of hospital admission	122
<b>Table (23)</b>	Lines of treatment received by the patients	123
<b>Table (24)</b>	Lines of treatment received by the patients	124
<b>Table (25)</b>	Different aspects of renal outcome.	125
<b>Table (26)</b>	Maternal outcome	126
<b>Table (27)</b>	Fetal outcome	127

## List of Figures

Figure No.	Title	Page No.
	Figures of Review of Literature	
Fig. (1)	Clinical phases of acute kidney injury	43
Fig. (2)	Stage based management of AKI	90
Fig. (3)	AKI risk assessment	93
Fig. (4)	Algorithmic Approach to the Management of AKI in Pregnancy	95
	Figures of Results	
Fig. (5)	Different Pregnancy complications that have impact on kidney function	111
Fig. (6)	Different clinical types of pregnancy related AKI	112
Fig. (7)	Obstetric history of pre-eclampsia	113
Fig. (8)	Type of AKI according to urine output at time of presentation	114
Fig. (9)	AKI classification according to KDIGO 2012	115
Fig. (10)	Renal Biopsy done for patients	121
Fig. (11)	Lines of treatment received by the patients	123
Fig. (12)	Lines of treatment received by the patients	124
Fig. (13)	Maternal and fetal outcome	126

## Tist of Abbreviations

Abb.	Full term
ACE	Angiotensin converting enzyme
<b>ACEIs</b>	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
<b>Anti-GBM</b>	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
<b>FENa</b>	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
<b>PIGF</b>	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
<b>VEGF</b>	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 readjustments (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,