

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

186,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Epidemiology, Causes and Outcome of Obstetric Acute Kidney Injury

Namrata Khanal^{1*}, Ejaz Ahmed² and Fazal Akhtar²

¹Middlemore Hospital, Auckland,

²Department of Renal Medicine,

Sindh Institute of Urology and Transplantation, Karachi,

¹New Zealand

²Pakistan

1. Introduction

Acute kidney injury (AKI) is a clinical syndrome denoted by an abrupt decline in glomerular filtration rate (GFR) sufficient to decrease the elimination of nitrogenous waste products (urea and creatinine) and other uremic toxins (Jefferson et al, 2010). AKI is a not very common yet serious complication occurring in pregnancy. The incidence and the mortality rates associated with obstetric acute kidney injury (also known as pregnancy related acute renal failure; PRARF) have decreased over the last few decades especially in developed countries (Prakash et al, 2007; Stratta et al, 1996). There are several factors which lead to this improvement and will be discussed later in the chapter. Since the term AKI is now widely used in place of acute renal failure (Ricci et al, 2011); for the ease of description we have used obstetric AKI in place of PRARF in this chapter.

Obstetric AKI can occur at any stage of pregnancy; ante-partum or post-partum and may be AKI occurring as a coincidence during pregnancy or AKI due to causes specific to pregnancy.

Although obstetric AKI is vanishing from developed world (Stratta et al, 1996), it is still a frequent cause of maternal morbidity and mortality in the developing nations. Poverty, lack of awareness and difficulties (e.g. lack of transport) accessing obstetric care all are responsible for this additional burden (World Health Organization [WHO], 2009). This also increases the disparity in reported number of cases and its actual occurrence contributing to scarcity of literature even in recent time.

1.1 Definition and epidemiology of Acute Kidney Injury (AKI) and obstetric AKI

Insight into the occurrence and consequences of kidney disease has rapidly progressed. More than 30 different definitions have been used for defining AKI in the literature, creating much confusion and making comparisons difficult (Bellomo et al, 2001). Recently, consensus

* Corresponding Author

definitions and classification systems have been proposed for AKI. RIFLE criteria stratify AKI into five stages (Table 1). This will eventually allow consistency across studies such that results can be compared (Ricci et al, 2011).

System	Serum creatinine criteria	Urine output criteria
RIFLE class		
Risk	Serum creatinine increase to 1.5-fold OR GFR decrease >25% from baseline	<0.5 ml/kg/h for 6 h
Injury	Serum creatinine increase to 2.0-fold OR GFR decrease >50% from baseline	<0.5 ml/kg/h for 12 h
Failure	Serum creatinine increase to 3.0-fold OR GFR decrease >75% from baseline OR serum creatinine ≥354 μmol/l (≥4 mg/dl) with an acute increase of at least 44 μmol/l (0.5 mg/dl)	Anuria for 12 h
AKIN Stage		
1	Serum creatinine increase ≥26.5 μmol/l (≥0.3 mg/dl) OR increase to 1.5–2.0-fold from baseline	<0.5 ml/kg/h for 6 h
2	Serum creatinine increase >2.0–3.0-fold from baseline	<0.5 ml/kg/h for 12 h
3	Serum creatinine increase >3.0-fold from baseline OR serum creatinine ≥354 μmol/l (≥4.0 mg/dl) with an acute increase of at least 44 μmol/l (0.5 mg/dl) OR need for RRT	<0.3 ml/kg/h for 24 h OR anuria for 12 h OR need for RRT

Small but important differences are observed between the two systems. A time constraint of 48 h for diagnosis (using either serum creatinine levels or urine output) is required in AKIN criteria. GFR decreases are used for diagnosis only in RIFLE criteria. In both systems, only one criterion (creatinine or urine output) has to be met to qualify for a given class or stage of AKI. Classes L and E of the RIFLE criteria are not reported. Owing to the wide variation in indications for and timing of initiation of RRT, individuals who receive RRT are considered to have AKIN Stage 3 AKI irrespective of their serum creatinine level and urine output.^{6,15} Abbreviations: AKI, acute kidney injury; AKIN, AKI Network; GFR, glomerular filtration rate; RIFLE, Risk, Injury Failure, Loss, End-stage renal disease; RRT, renal replacement therapy.

Table 1. Classification and staging systems for AKI.

AKI is estimated to occur in as many as 4%-20% of hospital admissions (Waikar et al, 2008) and in approximately 5-6% of critically ill patients with the period prevalence ranging from 1-25% (Uchino et al, 2005). Similarly, the period prevalence for acute renal replacement therapy in ICU is around 4-5% (Uchino et al, 2005). Septic shock in itself attributes to 50-60% of the cases. Hospital mortality in critically ill patients with AKI is equally higher at approximately 60% and has been reported to range from 28-90% (Uchino et al, 2005; Bellomo et al, 2004).

Incidence of pregnancy related AKI used to be 24-40% of all AKI in 60's which decreased to 2-3% in 80's (Fig 1) (Stratta, 1996). Interestingly enough, its incidence was already decreasing in 1963 as compared to 1959 when AKI occurred in 1 in 5000 pregnancies and 1 in 1400 pregnancies respectively in developed countries (Smith et al, 1965; Knapp & Hellman, 1959).

The incidence of obstetric AKI has further declined over last 4 decades (Prakash et al, 2010). This improvement is due to improved availability of safe and legal abortion, more widespread and aggressive antibiotic use decreasing the incidence of post-abortion sepsis, and improved prenatal care. In the past, obstetric AKI used to be mostly due to post-abortion sepsis (Gul et al, 2004). Four decade long retrospective review of pregnancy related AKI cases was published from a centre in Italy. They reported virtual non-existence of post abortion sepsis, while AKI from obstetric complications like amniotic fluid embolism, extensive haemorrhage and prolonged intrauterine death etc. had decreased. AKI associated with preeclampsia and eclampsia remained stable until 1987 however its incidence decreased dramatically thereafter contributing towards the improved maternal mortality rate. The decreased occurrence was more evident in developed nations (Stratta et al, 1996). In 1995, the maternal mortality ratio in Africa was estimated to be over 1000 per 100 000 pregnancies and in Europe 28 per 100 000 pregnancies (Hill et al., 2001). In a recent review on AKI amongst all hospital admissions, pregnancy related AKI has not been listed as one of the causes of AKI. Authors have included critically ill patients with AKI in this review and have reviewed around 170 published literatures (Waikar et al, 2008). It may not be an over statement to say that these cases are declining in incidence furthermore.

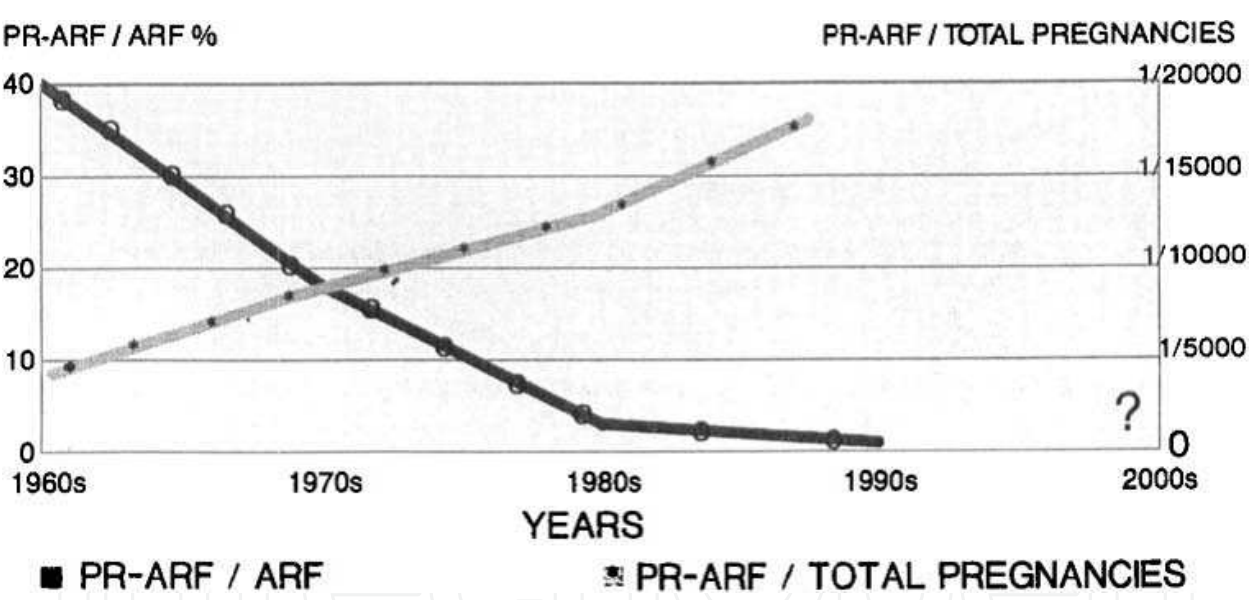


Fig. 1. Incidence of pregnancy related AKI.

However in recent reports published from developing countries; the frequency of obstetric AKI have been reported to be varying between 4-15% (Sivakumar et al, 2011). In a single centre study from India the incidence of AKI was reported as 1 in 56 births (Prakash et al, 2010) in contrast to 1 in 20000 births as reported from Italy (Stratta et al, Ren Fail 1996). Similar studies from individual centre from various developing countries have been published over the last decade emphasizing the fact that obstetric AKI is still prevalent in the developing or poor income nations (Table 2). However due to the absence of meta-analysis on obstetric AKI especially from these countries it is difficult to precisely estimate its actual incidence.

Author	Year published	Study popn	Obs-AKI (%)	Septic abortion	APH/PPH	Pre Eclampsia/HELLP	DIC	Puerperal sepsis	Misc.
Chugh et al	1976	325	22.1	31	12	8	9	9	12
Prakash et al	1995	426	13.9	45	2	4	-	5	3
*Prakash et al	2006	190	-	130	11	33	-	16	-
Kilari et al	2006	41	4.24	4	7	10	-	12	8
Goplani et al	2008	70	9.06	14	27	20	23	43	17
Saleem Najar et al	2008	40	7.02	20	8	6	4	-	6
Hassan et al	2009	130	33	-	24	5	4	12	-
*Khalil et al	2009	60	-	3	22	10	11	1	4
*Khanal et al	2010	50	-	3	36	14	-	16	18
Prakash et al	2010	106	20.75	Only included patients with preeclampsia					
**Prakash et al	2010	4758	1.78	NA	16	30	14	21	18
Agida et al	2010	46	13	Only included patients with preeclampsia					
*Erdemoglu et al	2010	75	-	11	9	57	-	-	-
Sivakumar et al	2011	59	4.36	28	11	18	-	-	11

* Study population included all pregnant women with AKI, causes listed existed together or individually **only included patients in third trimester; NA: not applicable; this series had sepsis as cause of AKI in 16 (18.8%) patients

Table 2. Incidence and causes of Obstetric AKI in developing countries.

1.2 Causes of AKI (Table 3)

First of all, any cause which can give rise to AKI in non-pregnant women of reproductive age group (pre-renal, renal, and post renal or obstructive) may contribute to AKI in pregnancy. They can be coincidental simultaneous occurrences in pregnant women. As in any evaluation of AKI, causes not related to pregnancy must thus be considered and excluded. Pregnancy unrelated causes of AKI represent only about 5% of all obstetric AKI (Krane, 1988).

A second group of patients are those with underlying chronic renal disease that worsens during pregnancy, suggesting the development of acute renal failure superimposed on their chronic diseases. Glomerular diseases might be diagnosed for the first time in pregnancy and AKI may occur as a result of rapidly progressive glomerulonephritis. Examples of disorders

that may show a decline in the GFR include chronic glomerular diseases, lupus nephritis, diabetic nephropathy, and the chronic interstitial nephritides. Discussion on renal function during pregnancy in women with underlying renal disease is beyond the scope of this chapter.

Finally, there are causes of renal failure specific to pregnancy, which are relatively more commonly encountered in gravid women. AKI seems to have bimodal distribution during pregnancy in first trimester and third trimester respectively.

Acute renal Failure in During early pregnancy <ul style="list-style-type: none">• Acute or massive blood loss-Abortion-Ectopic-Hyaditiform mole• Severe dehydration-Ac. Pyelonephritis-Hyperemesis gravidarum• ATN resulting from a septic abortion
ARF Late in Pregnancy/Postpartum <ul style="list-style-type: none">• Thrombotic microangiopathy-TTP-HUS-Severe preeclampsia usually with HELLP syndrome• Renal Cortical Necrosis resulting from-Placenta previa-Prolonged intrauterine foetal death-Amniotic fluid embolism• Intrinsic renal disease/autoimmune diseases• Acute pyelonephritis-ATN from septicaemia or hypotension-ARF from micro abscesses• Acute fatty liver of pregnancy
Obstructive Causes <ul style="list-style-type: none">-Mild-moderate hydronephrosis is normal-Occasionally, degree of obstruction sufficient to cause ARF-Nephrolithiasis if solitary functioning kidney

Table 3. Causes of Acute kidney Injury in Pregnancy.

Prerenal azotemia is the most common cause of both community and hospital-acquired AKI. It is an appropriate physiological response to renal hypo perfusion (Blantz, 1998). Similarly, the most common cause of AKI specifically in first trimester of pregnancy is prerenal azotemia owing to hyperemesis gravidarum or vomiting from acute pyelonephritis. In late pregnancy volume contraction may be secondary to blood loss (Pertuiset & Grunfled, 1994). Antepartum and postpartum haemorrhage have been reported as major causes of AKI in pregnancy (Smith et al, 1965; Kennedy et al, 1973; Ali et al, 2004). Pregnant women who sustained AKI receive blood transfusion frequently; this further emphasizes the frequency of significant haemorrhage in these patients (Ali et al, 2004; Khanal et al, 2010). Uterine bleeding secondary to abortion or septic abortions are still chief causes of obstetric AKI in developing countries.

Pregnancy-specific conditions such as preeclampsia, HELLP syndrome, acute fatty liver of pregnancy (AFLP), haemolytic uremic syndrome/ thrombotic thrombocytopenic purpura independently or in combination cause uterine bleeding ante-partum or post-partum haemorrhage. These conditions are frequently associated with complications like abruptio-placentae, hepatic infarction, hepatic rupture, intra-abdominal bleeding, and puerperal sepsis all of which can be further complicated by AKI. This occurs frequently in third trimester (Krane, 1988; Maynard et al, 2007; Prakash, 2010). Preeclampsia is a multi-system disorder unique to human pregnancy characterised by hypertension and involvement of one or more other organ systems and/or the foetus. American College of Obstetrics and Gynaecology, requires blood pressures $>140/90$ mm Hg on two occasions combined with urinary protein excretion >300 mg/d for the diagnosis of preeclampsia. Preeclampsia occurs in 3-5% of pregnancies and is associated with increased maternal and fetal mortality especially in developing countries. It is a leading cause of premature deliveries in developed countries thus increases the neonatal morbidity (Society of Obstetric Medicine of Australia and New Zealand [SOMANZ], 2009).

Abruptio placentae and puerperal sepsis; may also occur independent of these conditions and can be complicated by AKI. Obstetric complications such as septic abortion and placental abruption are associated with severe acute tubular necrosis (ATN) and bilateral cortical necrosis. Acute cortical necrosis, usually involves bilateral renal cortex, may occur as a consequence of irreversible or severe ATN. It has been found to be associated with poor renal outcome in longer term. Bilateral cortical necrosis is most often a complication of abruptio-placentae (36 per cent in the series of Chugh); while in other studies it was associated with disseminated intravascular coagulation. It presents as acute renal failure in other conditions too but, unlike acute tubular necrosis, total and persistent anuria is almost constant (Kleinknecht et al, 1973; Chugh, 1976). The diagnosis can be established either by renal biopsy or, better, by selective renal angiography. Other imaging studies (plain radiograph, ultrasound scan, CT-scan of abdomen and nuclear renal scan) may also be helpful. Renal cortical necrosis which occurs as a consequence of AKI in pregnancy continues sporadically (Naqvi et al, 1996; Prakash et al, 2007). With overall decrease in the incidence of AKI in pregnancy and improved overall management; incidence of cortical necrosis is decreasing even in the developing countries. Whenever present it is associated with irreversible renal failure (Khanal, 2010). Sepsis is still a major cause including septic abortions and puerperal sepsis in several studies published from India over last decades (Sivakumar, 2011).

Less common and miscellaneous causes of obstetric AKI include:

Obstructive uropathy; obstruction may occur in gravidas due to polyhydramnios, incarcerated gravid uterus, or can occur even in women with otherwise uncomplicated gestation due to retroverted uterus. Rarely, acute urinary tract obstruction in pregnancy is induced by a kidney stone, and it seldom causes renal failure (Strothers & Lee, 1992; Scarpa et al, 1996).

Amniotic fluid embolism which occurs primarily in multi-para after prolonged labour can cause AKI. Those with underlying renal parenchymal disease even without advanced chronic kidney disease are more prone to develop acute tubular necrosis (ATN) especially due to super imposed pre-eclampsia (Pertuiset & Grunfeld, 1994). Thrombotic thrombocytopenic purpura-haemolytic uremic syndrome (TTP-HUS) can easily be confused

with severe preeclampsia, usually with the HELLP syndrome (haemolysis with a microangiopathic blood smear, elevated liver enzymes, and a low platelet count) (McCrae et al, 1992) both can cause AKI.

There are anecdotal reports on sporadic cases of acute glomerulonephritis (GN) including post infectious GN, good-pasture's syndrome, lymphoma, drug nephrotoxicity, incompatible blood transfusions and endocarditis causing AKI during pregnancy (Pertuiset & Grunfeld, 1994). Acute interstitial nephritis (from antibiotics or non-steroidal anti-inflammatory drugs etc.) may coincidentally occur during pregnancy and result in AKI.

1.3 Changes in renal anatomy and physiology in normal pregnancy:

To understand the pathophysiology and proper management of renal problems in pregnancy it is important that we are familiar with the anatomical and physiological changes that occur during normal pregnancy.

1.3.1 Renal tract anatomy

The kidneys enlarge during normal pregnancy, increasing by 1 to 2 cm in length and in volume by up to 70% towards term, due to tissue hypertrophy and expansion of both interstitial and vascular compartments. More important from a clinical perspective is the increase in size of the renal pelvices and ureters. By third trimester about 80% of the pregnant women have hydronephrosis which is easily evident by ultrasound, more on the right than on the left (Baylis & Davison, 2010; Brown et al, 2010). A number of factors are thought to be important in this change. Progesterone, a smooth muscle relaxant, reduces ureteric tone and peristalsis. The asymmetric dilation of the pelvicalyceal system suggests extrinsic compression by the enlarging uterus at the pelvic brim, hypertrophy of surrounding connective tissue (Waldeyer's sheath) and kinking due to ligaments or compression by iliac blood vessels (Brown et al, 2010). The clinical consequence of these changes can be urinary stasis; increasing the risk of bacterial growth and asymptomatic bacteriuria of pregnancy. If the changes are in extreme and precipitate the over distention syndrome, with massive dilation they may present with symptoms like recurrent severe flank pain, increasing serum creatinine, hypertension, or even reversible acute kidney injury (Khanna & Nguyen, 2001). In case of presentation with over distention symptoms that suggest renal colic but no stone detectable by ultrasound or by radiographic imaging; it is imperative to exclude urinary tract infection and to avoid the temptation to drain the system using nephrostomy tubes. Furthermore it is important to remember that acute renal failure as a consequence of ureteric obstruction in pregnancy is uncommon; and that ureteric dilation is part of normal pregnancy and it is not usually possible to distinguish between this and pathologic dilation (Brown et al, 2010).

1.3.2 Renal physiology

1.3.2.1 Systemic haemodynamics

Detailed discussion of systemic haemodynamics of normal pregnancy is beyond the scope of this chapter. Briefly, changes start as early as the first trimester with reduced systemic vascular resistance, increase in cardiac output by 40-50% and resting tachycardia by 24th week (Davison & Dunlop, 1980). There is progressive expansion of the plasma and

extracellular fluid volume, reaching a maximum of 1.25 litres at times. The volume of the total extracellular fluid space is determined principally by sodium and hence water accumulation. The combination of increased cardiac output and peripheral vasodilatation means that organ blood flow increases in pregnancy, with the most dramatic changes occurring in the kidney and skin circulation throughout gestation and in the uterus in the second part of the pregnancy. These changes result in a small reduction in arterial blood pressure, typically reaching a nadir in the mid-trimester of about 10 mmHg systolic, rising towards pre-pregnancy levels at term (Brown & Gallery, 1994). Table 4 illustrates changes in some common indices during pregnancy.

	Non-Pregnant	Pregnant
Hematocrit (%)	41	33
Plasma creatinine (μmol/L)	<120	<90
mg/dl	<1.3	<1
Plasma urea (mmol/L)	11.2-31	9-12
mg/dl	4-11	3.2-4.4
Plasma albumin (g/L)	35-45	25-35
(g/dl)	3.5-4.5	2.5-3.5
Plasma uric acid mg/dl (μmol/L)	4 (240)	3.2 (190) early
		4.3 (260) late
Plasma bicarbonate (mmol/L, meq/l)	22-28	18-20
Urinary protein excretion (mg/d)	<150	<300

Table 4. Changes in some common indices during pregnancy (Modified from Source: Baylis & Davison, 2010; Brown et al 2010).

1.3.2.2 Renal haemodynamics

There is approximately 25% increase in glomerular filtration rate (GFR) by 4 weeks. This reaches a nadir of ~50% by mid pregnancy and is maintained until the last few weeks of pregnancy after which it starts to decrease however still remaining above the non-pregnant level. This leads to fall in serum creatinine (see Table 4). More pronounced is the increased renal plasma flow and decline in filtration fraction; both return to non pregnant level towards the term (Baylis & Davison, 2010).

In pre-eclampsia both renal plasma flow (RPF) and GFR decreases. There is salt retention and contraction of plasma volume as compared to normal pregnancy (Brown & Gallery, 1994). Although the absolute values of RPF and GFR may remain above non-pregnant level, this is probably the most likely mechanism of hypofiltration in this condition. The endothelium is involved inclusive of glomerulus, causing vascular endothelial cell dysfunction (glomerular endotheliosis) and results in swollen bloodless glomeruli and the loss of glomerular barrier size and charge selectivities (Baylis & Davison, 2010). This pattern is also seen in normal pregnancy and not pathognomonic of pre-eclampsia (Strevens, 2003).

1.3.3 Pathophysiology of AKI in preeclampsia/ eclampsia and other microangiopathies

In severe pre-eclampsia/ eclampsia, renal failure is most probably due to acute tubular necrosis (Sibai et al, 1993). ATN in these women is frequently due to haemorrhage; for

reasons explained in previous sections. AKI may also complicate postpartum eclampsia. HELLP syndrome (haemolysis, elevated liver enzymes and low platelet count) is a variant of severe pre-eclampsia, which usually resolves following delivery (Weinstein, 1985) and occurs in around 20% of the severe pre-eclampsia cases (Sibai et al, 1993). AKI may also occur as a direct consequence of disseminated intravascular coagulation (one of the dreaded complications in upto 20% of women with HELLP syndrome), and sepsis (Sibai et al, 1993).

Similarly, other microangiopathies like acute fatty liver of pregnancy which occurs commonly in the third trimester and haemolytic uremic syndrome/ thrombotic thrombocytopenic purpura occurring ante- or post-partum share several pathophysiologic mechanisms which is difficult to differentiate and eventually may contribute to the development of AKI (Ganesan, 2011). Acute fatty liver of pregnancy is an obstetric emergency, which if untreated may progress to fulminant hepatic failure and proves life threatening to both mother and foetus. Variable degree of AKI has been reported in upto 90% of women with acute fatty liver of pregnancy, which is usually reversible with the recovery of liver failure (Hou, 2001). HUS/TTP occurs typically in the early postpartum period but delays of several months postpartum have also been reported. Renal failure was previously thought to be irreversible but complete and partial recovery does occur in about 30% of these cases (Beaufils, 2001).

1.3.4 Pyelonephritis, septic abortion and puerperal sepsis

Pathogenesis of sepsis-induced renal dysfunction is still poorly understood. Though it has been demonstrated that septic AKI can occur in the setting of marked Hyperaemia and vasodilatation; and renal ischemia is not necessary for the loss of GFR (Bellomo et al, 2008); various inflammatory factors have also been shown to be generated following ischemia which contributes to development of AKI and ATN (Kribben, 1999). Experimental studies continue to report newer concepts for pathogenesis of septic AKI (Bellomo, 2011). Similar studies in man are required to confirm these experimental findings.

Pregnant women are at greater risk of urinary tract infection due to the altered anatomy and urinary stasis as discussed in previous sections. Untreated timely and correctly this can lead to urosepsis. Acute pyelonephritis may occur as part of urinary tract infection and may be severe enough to cause AKI as a result of sepsis or prerenal azotemia from vomiting. Improved availability and better management of abortion has led to decrease in the incidence of post-abortion sepsis especially in the developed countries (Gul et al, 2004). Sepsis is still a major cause including septic abortions and puerperal sepsis in developing countries (Sivakumar, 2011; Khanal, 2010).

1.4 Prevention and model of care

A total of 99% of all maternal deaths occur in developing countries, where 85% of the population lives. More than half of these deaths occur in sub-Saharan Africa and one third in South Asia. Globally, around 80 per cent of maternal deaths are due to obstetric complications; mainly haemorrhage, sepsis, unsafe abortion, pre-eclampsia and eclampsia, and prolonged or obstructed labour (United Nations Children's Fund [UNICEF], 2003; United Nations Development Programme [UNDP], 2006). An estimated 21.6 million unsafe abortions took place worldwide in 2008, almost all in developing countries. Numbers of unsafe abortions have increased from 19.7 million in 2003 (Department of Reproductive

Health and Research, WHO, 2011). Complications of unsafe abortions account for 13 per cent of maternal deaths worldwide, and 19 per cent of maternal deaths in South America (Ahman et al 2002; WHO, 2004). Preeclampsia is associated with poor maternal outcome including maternal death even in developed countries (SOMANZ, 2009; Isler , 1999).

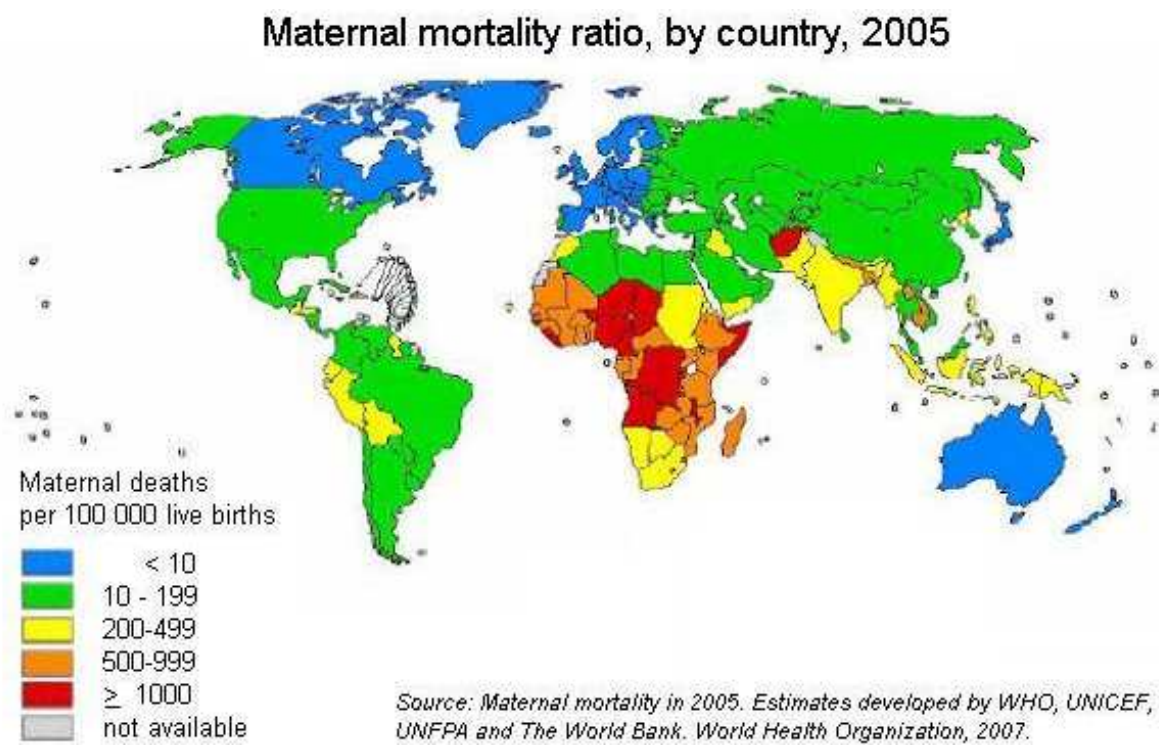


Fig. 2. Maternal mortality ratio by country.

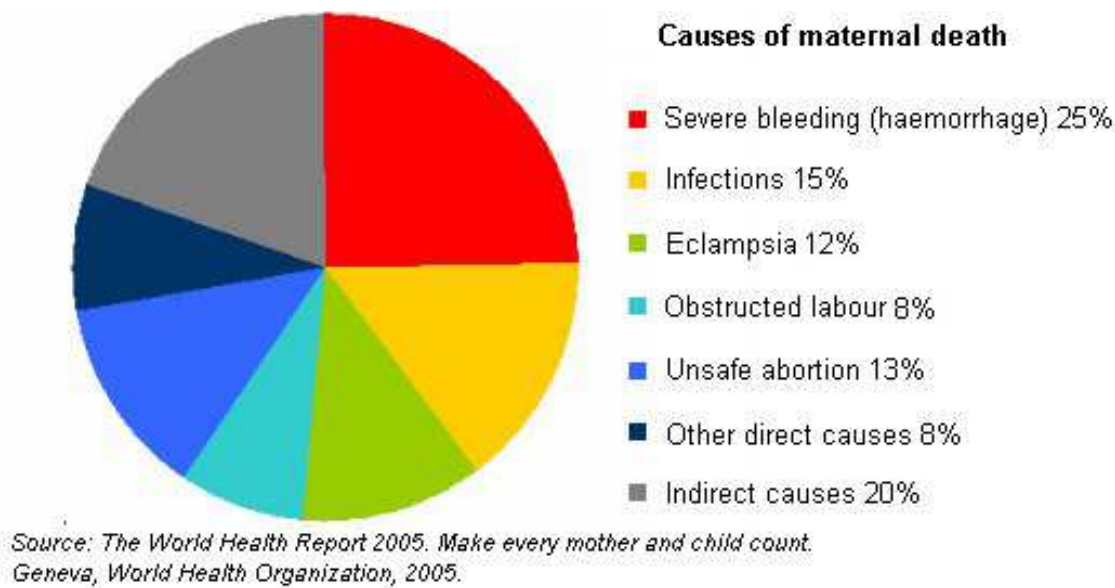


Fig. 3. Causes of maternal death.

One of the many complications contributing to this burden is AKI occurring as a consequence of these complications. It is very difficult to postulate any specific measure to prevent the occurrence of acute kidney injury. Adequate timely management of the

underlying condition which may be complicated by AKI is the only way to prevent it from happening. The physiologic changes in renal system that occur with pregnancy increasing the risk of infection are in itself non-modifiable, so is preeclampsia. Acute on chronic deterioration of renal function can probably be prevented by selecting women who are at lowest risk of progression of their existing kidney disease and perhaps counselling others for contraception is the only solution. Hence development of model of care is of utmost importance to reduce the maternal/ foetal mortality and morbidity; as has been long recognized by WHO (WHO, 2011); which is probably made worse by poor renal outcome in pregnant mothers. Widespread availability of improved prenatal care through midwives and timely recognition/ referral of high risk cases have decreased the incidence of pregnancy related AKI in developed world. An estimated 74 per cent of maternal deaths could be averted if all women had access to the interventions for preventing or treating pregnancy and birth complications, in particular emergency obstetric care (Barbinard & Roberts, 2006). There is need to improve the provision of quality services in developing countries. Factors such as poverty, gender inequalities, illiteracy, poor health systems, political instability, cultural barriers, and lack of infrastructure (e.g. lack of transport) in certain areas making it difficult to access the facility all contribute to increased burden (WHO, 2011).

Measures to decrease maternal mortality also aim to reduce the consequences women face as a result of these complications. AKI is one of them. Below are our recommendations in keeping with the guidelines proposed by WHO and UNFPA (United Nations family planning association) in Millennium Summit as Millennium development goal 5 (MDG5) which will probably help to decrease maternal mortality in developing countries:

Firstly, it is very important for the pregnant women to understand the benefits of seeking safe abortion, utilizing antenatal follow up and when possible avoid unplanned pregnancies. It is likely that the morbidity and mortality risk would be reduced with adequate antenatal and delivery care (Robinson & Wharrad, 2001; de Bernis et al., 2000). **Secondly**, increasing the number of health personnel in form of midwives and trained birth attendants and making them available for these populations, frequent free health camps at remote areas to identify the population at risk e.g. identifying women with underlying kidney disease, and education on family planning and easy access to contraceptive measures, increasing its availability and legalizing the abortions are beneficial. **Finally**, timely intervention when needed through experts when needed for e.g. timely administration of antibiotics for infection, adequate management of hypovolaemia, performing caesarean section when indicated, and vigilant post partum care of these women all are important steps to prevent complication and thus reduce maternal mortality. Increased use of contraception has an obvious and direct effect on the maternal death rate per 1000 women of reproductive age and on the lifetime risk of maternal death, by reducing the number of pregnancies (Royston & Armstrong, 1989). Unsafe abortions are entirely preventable, and yet continue to occur in almost all developing countries and in Eastern Europe. The evidence suggests that this can be greatly reduced when (Department of Reproductive Health and Research, WHO, 2011):

- Pregnancies can be planned through effective contraception;
- Counselling and services meet the unmet need for family planning, and appropriate method mix of contraception is offered to all women, including both married and unmarried women; and

- Safe abortion services are available and accessible.

In the meantime ill-effects of unsafe abortion should be prevented by:

- making safe abortions services available and accessible where abortion is not against the law;
- ensuring that permitted reasons for abortion are supported by the national legislative process and health systems;
- granting access to services for the management of complications arising from unsafe abortion; and
- Providing post abortion counselling and offering contraceptive services, this will also help to avoid repeat abortion.

Similarly, preeclampsia occurs in 3-5% of pregnancies and is associated with increased maternal and foetal mortality especially in developing countries. If women with these problems are identified in antenatal period, safe delivery can be planned before hand (WHO, Dept. of Reproductive Health and Research, Dept. of Maternal, Newborn, Child and Adolescent Health, Dept. of Nutrition for Health and Development, 2011). Some reports have been published where the authors have questioned the importance of presence of skilled birth attendance at delivery and suggested that perhaps partnership between midwives and doctors and timely referral is more important to achieve this target (Cross et al, 2010) of reducing maternal mortality by 2/3 in these regions. Despite efforts from national, international and global health organizations MMR declined by only 5% from 1990-2005. To achieve this target of decline in MMR by 2/3 by 2015 will require tremendous work in this area. Any improvement in the maternal mortality rate will eventually lead to decrease consequences like AKI from various complications during pregnancy in developing countries.

1.5 Management outline

1.5.1 Fluid and electrolyte balance (Maynard et al, 2010)

Timely recognition of the events and adequate replenishment of the fluid volume is essential to prevent more dreaded complication of acute tubular necrosis (ATN). Eventually when AKI ensues management depends on the underlying cause of renal dysfunction. To avoid/correct hypovolaemia and ascertain fluid balance is important at every stage. Where bleeding is the cause of hypovolaemia, measures to stop bleeding should precede all other procedures, this may necessitate termination of pregnancy, preterm delivery of the foetus and blood transfusion. Efforts should be made not to incite further insult by avoiding or minimising the use of nephrotoxic agents including radio-contrast dyes and various drugs as much as possible. If they have to be used; adjustment of the dose will be required. Equal attention has to be paid to ensure adequate electrolyte balance. Of all potassium requires regular monitoring. Hyperkalaemia demands urgent medical management and if persistent may be an indication for renal replacement therapy.

1.5.2 Appropriate management of sepsis

When infection coexists; use of appropriate antibiotics empirically is justified. Ensuring the safety of antibiotics during pregnancy is of utmost importance. Initial choice of antibiotics may vary according to the hospital protocol and prevalence of antibiotic resistance for the suspected organism. Antibiotic spectrum will often have to be broadened according to the severity of infection.

1.5.3 Management of preeclampsia/eclampsia/HELLP syndrome

Management of these syndromes is usually supportive and revolves around blood pressure control, use of magnesium sulphate to prevent seizures and timing of delivery. There are various guidelines proposed by different obstetric societies around the globe in regards to management. Of interest however are the emerging concepts on its long term cardiovascular and renal consequences (Mc Donald et al, 2008). Similarly acute fatty liver of pregnancy (AFLP) also demands supportive care and prompt delivery. Thrombotic thrombocytopenic purpura; which creates a diagnostic dilemma together with HELLP and AFLP is managed using plasma exchange. Studies have shown significant improvement in maternal mortality rates since its introduction (Martin et al, 2008).

1.5.4 Renal replacement therapy in pregnancy

When renal replacement therapy is indicated for medically not amenable acute complications like hyperkalaemia, fluid overload, metabolic acidosis, and uremic encephalopathy either of the modalities (haemodialysis; HD or peritoneal dialysis; PD) can be used during pregnancy however there are no head to head trials comparing the benefit of one over the other. However, studies do suggest that PD may interfere with utero-placental blood flow (Bui et al, 2003).

1.6 Outcome of obstetric AKI

Attempts have been made to derive factors to predict mortality associated with AKI. In general, AKI associated mortality seems to be variably increasing with increasing age, greater degree of illness severity at presentation, presence of chronic kidney disease, need for organ support in form of mechanical ventilation, hypotension or need for inotrope support and so on (Waikar et al, 2008). Degree of change in serum creatinine and need for dialysis as well are associated with increased mortality rates. These estimates however have not been analysed specifically in the setting of pregnancy related AKI.

Table 5 summarizes maternal mortality rates and renal outcome in pregnant women with AKI from different studies. In our experience prolonged duration of oliguria is associated with increased rate of dialysis dependency. Most of pregnant women with acute kidney injury come from rural areas and did not have antenatal check up (Khanal et al, 2010; Ahmad et al, 2001; Hassan et al, 2009). Antenatal check up not only helps in creating awareness among the pregnant mothers to seek help from midwives, it also brings high risk cases to notice and increases the likelihood of referral to experts on time. Sepsis including post abortion sepsis has been found to be associated with severe consequences and poor maternal outcome even in modern days in developing countries. This is probably from poor handling techniques and emphasizes the importance of need to increase the number of trained personnel like midwives to conduct delivery. Late referral to the tertiary care centres and delays in actually reaching the centre due to lack of infrastructure all contribute to the poor outcome. Significant and progressive improvement in mortality rates over decades is evident in table 6. This study analysed the data on pregnancy related AKI over 37 years. Although a progressive decline over each decade can be easily noticed, complications like HUS were associated with adverse maternal outcome. Thus close monitoring of high risk cases, timely recognition of complication, and institution of appropriate management intervention on time are of utmost importance (Stratta, 1996). A review on acute renal

failure in hypertensive pregnant women which was conducted over 12 years included 9600 women with hypertension. 31 of these women developed AKI, all were in the postpartum period. Of these there were 2 maternal deaths and 50% of the patients from the pre-eclamptic group required dialysis. All patients had acute tubular necrosis (ATN). In the chronic hypertensive group with super imposed pre-eclampsia, 42% required dialysis and 3 had cortical necrosis (Sibai et al, 1990).

Author	Year	Study Pop with AKI	Mortality (%)	Dialysis dependent	Partial renal recovery	Complete renal recovery
Knapp	1957	23/32000 deliveries	48	-	-	-
Smith	1965	70	32	-	-	-
Chugh	1976	72	55.6	-	-	-
Prakash	1995	59	27	-	-	-
Stratta	1996	84	31	7	-	-
Kilari	2006	41	24.39	-	4	21
Hassan	2009	43	16.2	6	12	18
Khalil	2009	60	15	5	6	28
Khanal	2010	50	8	25	11	14
Prakash	2010	85	20	1	5	59
Arora	2010	57	28.1	3	5	24
Erdemoglu	2010	75	10.6	33.3% (required dialysis)		
Sivakumar	2011	59	23.72	-	10.16	54.23

Table 5. Renal outcome in Obstetric AKI.

Years	1958- 1967	1968- 1977	1978- 1987	1988- 1994
ARF (AKI)	60	298	535	562
PR-ARF	26(43%)	40(13.4%)	15(2.8%)	3(0.5%)

Table 6. Total Number and Main Causes of PR_ARF (obstetric AKI) Observed in 37 Years at Department of Nephrology and Clinical Obstetrics Torino, Italy (P Stratta,1996).

Both ante partum or post partum haemorrhage can lead to pre renal azotemia. Haemorrhage due to abruptio placentae has been found to be associated with increased risk of irreversibility of renal function in some series due to the development of cortical necrosis (Turney et al, 1989; Sibai et al, 1993). It is unclear why BRCN occurs more frequently during pregnancy, but this complication has been associated with septic abortions, preeclampsia, abruptio placentae, postpartum accidents, and haemorrhage. Bilateral renal cortical necrosis has been frequently mentioned to be associated with irreversibility of the renal function (Turney et al, 1989).

Foetal outcome is also poor. Intra-uterine death and still birth has been reported as high as 30-70% (Ali et al, 2004; Prakash et al, 2007; Khanal et al, 2010). High incidence of foetal loss was associated with increased incidence of dialysis dependency in mothers. This could be owing to the increased severity of illness (Khanal et al, 2010). Perinatal mortality is

significantly low in neonates born to pregnant mothers without AKI as compared to those who developed AKI during pregnancy (Gul et al, 2004).

With the implementation of MDG 5 in developing countries, maternal mortality rates owing to all of these complications will hopefully improve along with improvement in foetal outcome.

1.7 Future perspective of obstetric acute kidney injury

Evident from the history, it is certain that maternal mortality rates from complications that can occur in pregnancy can be improved. Efforts have been put forward to increase the skilled birth attendants to assist delivery. However difficulties associated with overall health system and physical infrastructure, political instability and high illiteracy rates etc. creates hindrance to smooth development and therefore difficulty in achieving goals in developing countries.

Increased incidence of preterm deliveries associated with preeclampsia and its adverse long term renal/ cardiovascular outcome demand further research to understand the basis of the problem.

1.8 Recommendations

American college of obstetricians and gynaecologist guidelines
Society of Obstetric Medicine of Australia and New Zealand guidelines
Royal college of obstetricians and gynaecologists guidelines
WHO Guidelines on reproductive and sexual health

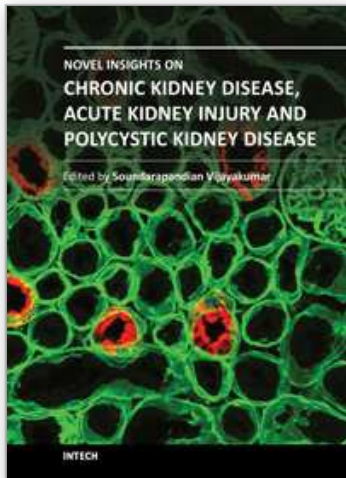
2. References

- Agida ET, Adeka BI, Jibril KA: Pregnancy outcome in eclamptics at the University of Abuja Teaching Hospital, Gwagwalada, Abuja: a 3 year review. *Niger J Clin Pract*; 13: 394-398.
- Ahmad W, Ziaulllah, Rizwanul-Haque M, Shari T. Acute renal failure: causes and outcome. *Proceeding Shaikh Zayed Postgrad Med Inst* 2001; 15:23-8.
- Ahman E, Shah I: Unsafe abortion: worldwide estimates for 2000. *Reprod Health Matters* 2002; 10: 13-17.
- Ali A, Zaffar S, Mehmood A, Nisar A. Obstetrical acute renal failure from Frontier Province: a 3 years prospective study. *J Postgrad Med Inst* 2004; 18:109-17.
- Babinard J, Roberts P. Maternal and Child Mortality Development Goals: What Can the Transport Sector Do? *The International Bank for Reconstruction and Development/The World Bank*; 2006
- Baylis C, Davison JM. Pregnancy and Renal Disease. In: Jurgen F, Johnson RJ, Feehally J. *Comprehensive clinical nephrology*. 4th ed. New York: Elsevier; 2010
- Beaufils M. Pregnancy. In: Davison A, Cameron J, Grunfeld, Ponticelli C, Ritz E, Winearls C, Ypersele C. *Oxford text book of clinical nephrology*. 3rd ed. Great Clarendon Street: Oxford university press; 2005: 1604-12.
- Bellomo R, Kellum J, Ronco C: Acute renal failure: time for consensus. *Intensive Care Med* 2001; 27: 1685-1688.
- Bellomo R, May C, Wan L: Acute renal failure and sepsis. *N Engl J Med* 2004; 351: 2347-2349; author reply 2347-2349.

- Bellomo R, Wan L, Langenberg C, Ishikawa K, May CN: Septic acute kidney injury: the glomerular arterioles. *Contrib Nephrol*; 174: 98-107.
- Blantz RC: Pathophysiology of pre-renal azotemia. *Kidney Int* 1998; 53: 512-523.
- Brown MA, Gallery ED: Volume homeostasis in normal pregnancy and pre-eclampsia: physiology and clinical implications. *Baillieres Clin Obstet Gynaecol* 1994; 8: 287-310.
- Brown MA, Mangos GJ, Peek M, Plaat F: Renal Disease. In: Powrie R, Greene MF, Camann W. *de Swiet's Medical Disorders in Obstetric Practice*. 5th ed. Blackwell; 2010: 182-209.
- Bui HT, Avjioglu E, Clarke A, Ellis D: Severe post-partum haemorrhage of the right kidney. *Aust N Z J Obstet Gynaecol* 2003; 43: 243-245.
- Chugh KS, Singhal PC, Sharma BK, Pal Y, Mathew MT, Dhall K, et al.: Acute renal failure of obstetric origin. *Obstet Gynecol* 1976; 48: 642-646.
- Cross S, Bell JS, Graham WJ: What you count is what you target: the implications of maternal death classification for tracking progress towards reducing maternal mortality in developing countries. *Bull World Health Organ*; 88: 147-153.
- Davison JM, Dunlop W: Renal hemodynamics and tubular function normal human pregnancy. *Kidney Int* 1980; 18: 152-161.
- de Bernis L, Dumont A, Bouillin D, Gueye A, Dompnier JP, Bouvier Colle MH (2000). Maternal morbidity and mortality in two different populations of Senegal: a prospective study (MOMA survey). *British journal of obstetrics and gynaecology*, 107(1): 68-74
- Department of Reproductive Health and Research, World Health Organization. Unsafe abortion: global and regional estimates of the incidence of unsafe abortion and associated mortality in 2008. 6th Ed. 2011
- Erdemoglu M, Kuyumcuoglu U, Kale A, Akdeniz N: Pregnancy-related acute renal failure in the southeast region of Turkey: analysis of 75 cases. *Clin Exp Obstet Gynecol*; 37: 148-149.
- Ganesan C, Maynard SE: Acute kidney injury in pregnancy: the thrombotic microangiopathies. *J Nephrol*; 24: 554-563.
- Goplani KR, Shah PR, Gera DN, Gumber M, Dabhi M, Feroz A, et al.: Pregnancy-related acute renal failure: A single-center experience. *Indian J Nephrol* 2008; 18: 17-21.
- Gul A, Aslan H, Cebeci A, Polat I, Ulusoy S, Ceylan Y: Maternal and fetal outcomes in HELLP syndrome complicated with acute renal failure. *Ren Fail* 2004; 26: 557-562.
- Hassan I, Junejo AM, Dawani ML: Etiology and outcome of acute renal failure in pregnancy. *J Coll Physicians Surg Pak* 2009; 19: 714-717.
- Hill K, Abou-Zhar C, Wardlaw T (2001). Estimates of maternal mortality for 1995. *Bulletin of the World Health Organization*, 79(3): 182-193.
- Hou HS. ARF in pregnancy. In: Molitoris BA, Finn WF. *Acute renal failure: a companion to Brenner & Rector's The kidney*. Philadelphia: W.B. Saunders; 2001: 304-11.
- Hunt P, De Mesquita JB. Reducing maternal mortality: The contribution of the right to the highest attainable standard of health. University of Essex: 2010
- Isler CM, Rinehart BK, Terrone DA, Martin RW, Magann EF, Martin JN, Jr.: Maternal mortality associated with HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome. *Am J Obstet Gynecol* 1999; 181: 924-928.
- Jefferson A, Thurman JM, Schrier RW. Pathophysiology and Etiology of Acute Kidney Injury. In: Jurgen F, Johnson RJ, Feehally J. *Comprehensive clinical nephrology*. 4th ed. New York: Elsevier; 2010

- Karumanchi SA, August P, Podymow T. Renal complications in normal pregnancy. In: Jurgen F, Johnson RJ, Feehally J. Comprehensive clinical nephrology. 4th ed. New York: Elsevier; 2010
- Kennedy AC, Burton JA, Luke RG, Briggs JD, Lindsay RM, Allison ME, et al.: Factors affecting the prognosis in acute renal failure. A survey of 251 cases. *Q J Med* 1973; 42: 73-86.
- Khalil MA, Azhar A, Anwar N, Aminullah, Najm ud D, Wali R: Aetiology, maternal and foetal outcome in 60 cases of obstetrical acute renal failure. *J Ayub Med Coll Abbottabad* 2009; 21: 46-49.
- Khanal N, Ahmed E, Akhtar F: Factors predicting the outcome of acute renal failure in pregnancy. *J Coll Physicians Surg Pak*; 20: 599-603.
- Khanna N, Nguyen H: Reversible acute renal failure in association with bilateral ureteral obstruction and hydronephrosis in pregnancy. *Am J Obstet Gynecol* 2001; 184: 239-240.
- Kleinknecht D, Grunfeld JP, Gomez PC, Moreau JF, Garcia-Torres R: Diagnostic procedures and long-term prognosis in bilateral renal cortical necrosis. *Kidney Int* 1973; 4: 390-400.
- Knapp RC, Hellman LM: Acute renal failure in pregnancy. *Am J Obstet Gynecol* 1959; 78: 570-577.
- Krane NK: Acute renal failure in pregnancy. *Arch Intern Med* 1988; 148: 2347-2357.
- Kribben A, Edelstein CL, Schrier RW: Pathophysiology of acute renal failure. *J Nephrol* 1999; 12 Suppl 2: S142-151.
- Martin JN, Jr., Bailey AP, Rehberg JF, Owens MT, Keiser SD, May WL: Thrombotic thrombocytopenic purpura in 166 pregnancies: 1955-2006. *Am J Obstet Gynecol* 2008; 199: 98-104.
- Maynard SE, Karumanchi SA, Thadhani R. Hypertension and kidney disease in pregnancy In: Brenner BM, Rector FC. The kidney. 7th ed. Philadelphia: W.B. Saunders; 2010: 1567-95
- McCrae KR, Samuels P, Schreiber AD: Pregnancy-associated thrombocytopenia: pathogenesis and management. *Blood* 1992; 80: 2697-2714.
- McDonald SD, Malinowski A, Zhou Q, Yusuf S, Devereaux PJ: Cardiovascular sequelae of preeclampsia/eclampsia: a systematic review and meta-analyses. *Am Heart J* 2008; 156: 918-930.
- Najar MS, Shah AR, Wani IA, Reshi AR, Banday KA, Bhat MA, et al.: Pregnancy related acute kidney injury: A single center experience from the Kashmir Valley. *Indian J Nephrol* 2008; 18: 159-161.
- Naqvi R, Akhtar F, Ahmed E, Shaikh R, Ahmed Z, Naqvi A, et al.: Acute renal failure of obstetrical origin during 1994 at one center. *Ren Fail* 1996; 18: 681-683.
- Pertuiset N, Grunfeld JP: Acute renal failure in pregnancy. *Baillieres Clin Obstet Gynaecol* 1994; 8: 333-351.
- Prakash J, Kumar H, Sinha DK, Kedelaya PG, Pandey LK, Srivastava PK, et al.: Acute renal failure in pregnancy in a developing country: twenty years of experience. *Ren Fail* 2006; 28: 309-313.
- Prakash J, Niwas SS, Parekh A, Pandey LK, Sharatchandra L, Arora P, et al.: Acute kidney injury in late pregnancy in developing countries. *Ren Fail*; 32: 309-313.
- Prakash J, Tripathi K, Pandey LK, Sahai S, Usha, Srivastava PK: Spectrum of renal cortical necrosis in acute renal failure in eastern India. *Postgrad Med J* 1995; 71: 208-210.

- Prakash J, Vohra R, Wani IA, Murthy AS, Srivastva PK, Tripathi K, et al.: Decreasing incidence of renal cortical necrosis in patients with acute renal failure in developing countries: a single-centre experience of 22 years from Eastern India. *Nephrol Dial Transplant* 2007; 22: 1213-1217.
- Ricci Z, Cruz DN, Ronco C: Classification and staging of acute kidney injury: beyond the RIFLE and AKIN criteria. *Nat Rev Nephrol*; 7: 201-208.
- Robinson JJ, Wharrad H (2001). The relationship between attendance at birth and maternal mortality rates: an exploration of United Nations' data sets including the ratios of physicians and nurses to population, GNP per capita and female literacy. *Journal of advanced nursing*, 34(4): 445-455.
- Royston E, Armstrong S (1989). *Preventing maternal deaths*. Geneva, World Health Organization
- Scarpa RM, De Lisa A, Usai E: Diagnosis and treatment of ureteral calculi during pregnancy with rigid ureteroscopes. *J Urol* 1996; 155: 875-877.
- Sibai BM, Ramadan MK. Acute renal failure in pregnancy complicated by hemolysis, elevated liver enzymes, and low platelets. *Am J Obstet Gynecol* 1993; 168 (6 pt 1): 1682-90.
- Sibai BM, Villar MA, Mabie BC. Acute renal failure in hypertensive disorders of pregnancy. Pregnancy outcome and remote prognosis in thirty-one consecutive cases. *Am J Obstet Gynecol* 1990; 162 (3): 777-83.
- Sivakumar V, Sivaramakrishna G, Sainaresh VV, Sriramnaveen P, Kishore CK, Rani Ch S, et al.: Pregnancy-related acute renal failure: a ten-year experience. *Saudi J Kidney Dis Transpl*; 22: 352-353.
- Smith K, Browne JC, Shackman R, Wrong OM: Acute Renal Failure of Obstetric Origin: An Analysis of 70 Patients. *Lancet* 1965; 2: 351-354.
- Stothers L, Lee LM: Renal colic in pregnancy. *J Urol* 1992; 148: 1383-1387.
- Stratta P, Besso L, Canavese C, Grill A, Todros T, Benedetto C, et al.: Is pregnancy-related acute renal failure a disappearing clinical entity? *Ren Fail* 1996; 18: 575-584.
- Strevens H, Wide-Svensson D, Hansen A, Horn T, Ingemarsson I, Larsen S, et al.: Glomerular endotheliosis in normal pregnancy and pre-eclampsia. *BJOG* 2003; 110: 831-836.
- Uchino S, Kellum JA, Bellomo R, Doig GS, Morimatsu H, Morgera S, et al.: Acute renal failure in critically ill patients: a multinational, multicenter study. *JAMA* 2005; 294: 813-818.
- Waikar SS, Liu KD, Chertow GM: Diagnosis, epidemiology and outcomes of acute kidney injury. *Clin J Am Soc Nephrol* 2008; 3: 844-861.
- Weinstein L: Preeclampsia/eclampsia with hemolysis, elevated liver enzymes, and thrombocytopenia. *Obstet Gynecol* 1985; 66: 657-660.
- World health organization. Population dynamics and reducing maternal mortality. 2009 URL:
http://www.who.int/making_pregnancy_safer/en/
http://www.somanz.org/pdfs/somanz_guidelines_2008.pdf
<http://www.unfpa.org/public/publications/pid/4968>
http://siteresources.worldbank.org/INTTRANSPORT/Resources/336291-1227561426235/5611053-1229359963828/tp12_maternal_health.pdf



Novel Insights on Chronic Kidney Disease, Acute Kidney Injury and Polycystic Kidney Disease

Edited by Dr. Soundarapandian Vijayakumar

ISBN 978-953-51-0234-2

Hard cover, 134 pages

Publisher InTech

Published online 07, March, 2012

Published in print edition March, 2012

This book offers novel insights on topics such as congenital obstructive nephropathy, cerebral-renal salt wasting, and the role of hemoglobin variability in clinical outcomes of CKD which are not very often discussed in the literature. With comprehensive and insightful reviews by eminent clinicians and scientists in the field, this book is a valuable tool for nephrologists.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Namrata Khanal, Ejaz Ahmed and Fazal Akhtar (2012). Epidemiology, Causes and Outcome of Obstetric Acute Kidney Injury, Novel Insights on Chronic Kidney Disease, Acute Kidney Injury and Polycystic Kidney Disease, Dr. Soundarapandian Vijayakumar (Ed.), ISBN: 978-953-51-0234-2, InTech, Available from: <http://www.intechopen.com/books/novel-insights-on-chronic-kidney-disease-acute-kidney-injury-and-polycystic-kidney-disease/epidemiology-causes-and-outcome-of-obstetric-acute-kidney-injury>

INTECH
open science | open minds

InTech Europe

University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China

Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

© 2012 The Author(s). Licensee IntechOpen. This is an open access article distributed under the terms of the [Creative Commons Attribution 3.0 License](https://creativecommons.org/licenses/by/3.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

IntechOpen

IntechOpen