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Epidemiology, Causes and Outcome of Obstetric Acute Kidney Injury

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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

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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 $\geq 354 \mu\text{mol/l}$ ($\geq 4 \text{ mg/dl}$) with an acute increase of at least $44 \mu\text{mol/l}$ (0.5 mg/dl)	Anuria for 12 h
AKIN Stage		
1	Serum creatinine increase $\geq 26.5 \mu\text{mol/l}$ ($\geq 0.3 \text{ mg/dl}$) 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 $\geq 354 \mu\text{mol/l}$ ($\geq 4.0 \text{ mg/dl}$) with an acute increase of at least $44 \mu\text{mol/l}$ (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-abortal sepsis, and improved prenatal care. In the past, obstetric AKI used to be mostly due to post-abortal 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 abortal 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.

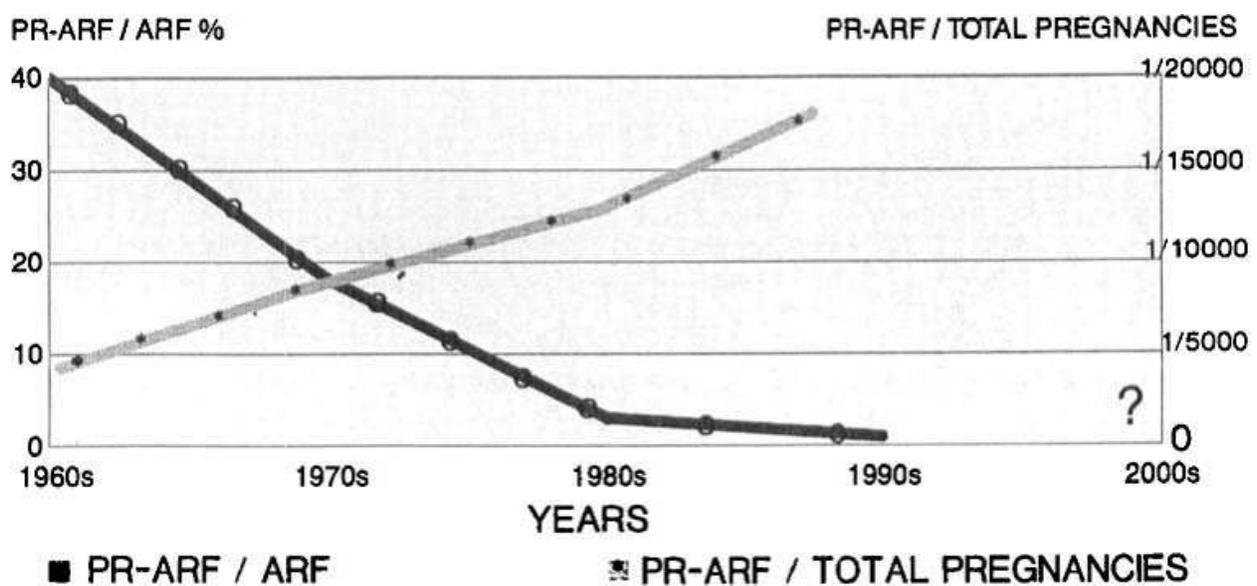


Fig. 1. Incidence of pregnancy related AKI.

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 Najjar 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

- **Acute or massive blood loss**
- **Abortion**
- **Ectopic**
- **Hydatiform mole**
- **Severe dehydration**
- **Ac. Pyelonephritis**
- **Hyperemesis gravidarum**
- **ATN resulting from a septic abortion**

ARF Late in Pregnancy/Postpartum

- **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

- **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 ($\mu\text{mol/L}$)	<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 ($\mu\text{mol/L}$)	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-abortal 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).

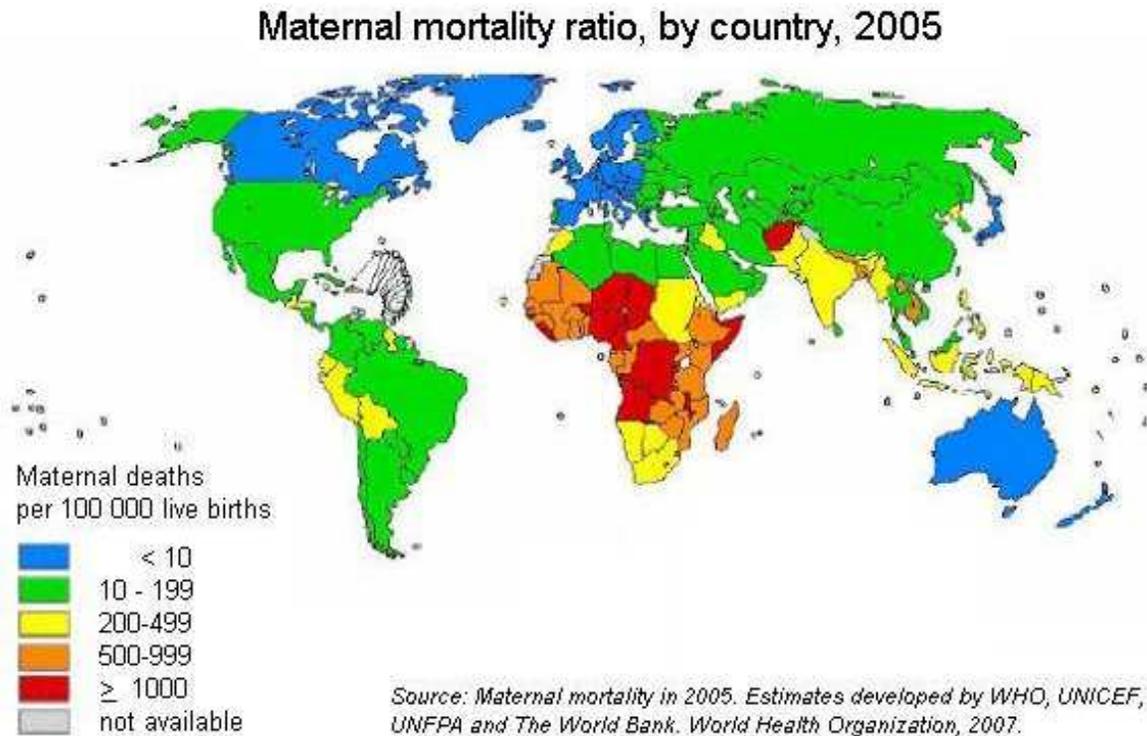
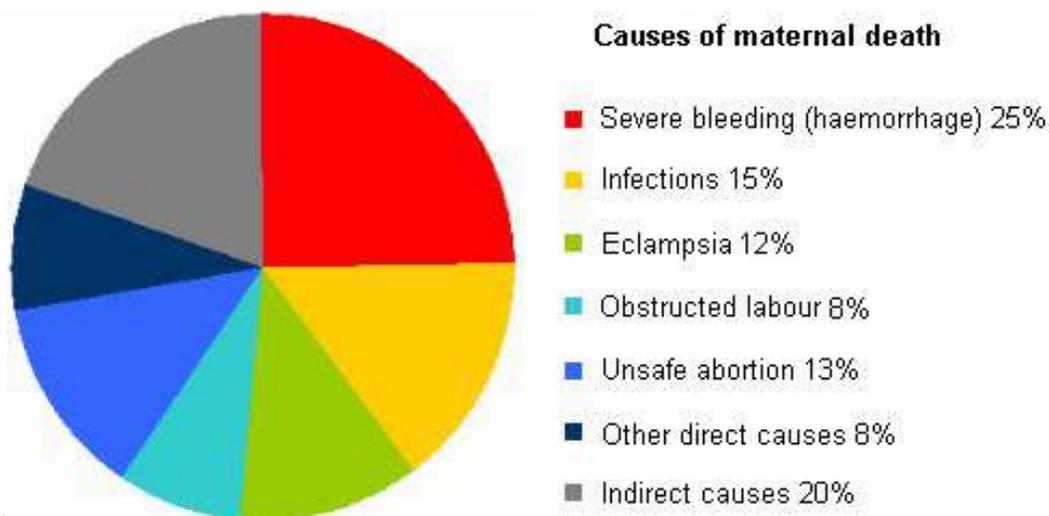


Fig. 2. Maternal mortality ratio by country.



Source: The World Health Report 2005. Make every mother and child count. Geneva, World Health Organization, 2005.

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 or 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 abortal 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

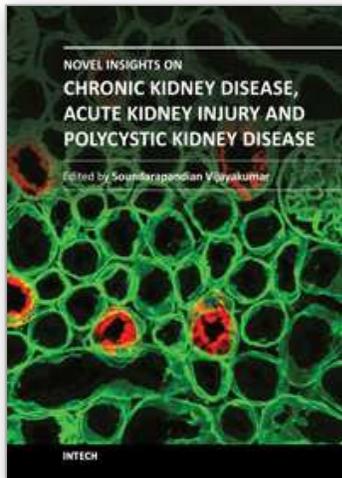
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