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Peripartum Acute Renal Failure.

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Abstract:

Acute renal failure is a rare challenging medical complication during pregnancy. Despite decreasing incidence mortality and morbidity of acute renal failure associate with pregnancy remains high. Management requires knowledge of the renal physiologic changes occurring in pregnancy and the relevant diagnoses, both pregnancy-specific

and those that may coincidentally occur with pregnancy.

Ideal medical care of this patients need a multidisciplinary approach considering maternal and fetal complication and timely specialist involvement.

Key Words: Acute Renal Failure, Pregnancy.

Introduction:

Renal disease may develop de novo during pregnancy. The usual causes are new-onset glomerulonephritis or interstitial nephritis, lupus nephritis, or acute renal failure (ARF). Rarely, obstructive uropathy develops as a result of stone disease or large uterine myoma that has increased in size during pregnancy.

Peripartum acute renal failure is an important clinical problem and despite decreasing incidence is associated with significant mortality and morbidity.

Definition, Incidence and Classification:

Acute renal failure is a syndrome characterized by rapid decline in glomerular filtration rate and retention of nitrogenous waste product such as BUN and creatinine [1]. The Acute Dialysis Quality Initiative developed a model for diagnosis of ARF, as shown in Figure 1 [2].

The hemodynamic changes affecting renal blood flow are coincident with and partially causative of some of the general cardiovascular changes of pregnancy. Very early decreases in peripheral vascular resistance in pregnancy are due in large part to decreased renal vascular resistance that may be related to the effects of maternal hormones such as relaxin.

This arteriolar underfilling is thought to lead to a systemic response including marked increases in cardiac output (approximately 50% above nonpregnant baseline) and plasma volume (approximately 40% above baseline).

There are also changes in glomerular filtration rate (GFR) and renal plasma flow. Both increase during the first half of pregnancy and subsequently level off, with increases on the order of 40–65% for GFR and 50–85% for renal plasma flow .

These changes predict the decreases in serum creatinine levels that are seen throughout gestation [3, 4].

Table 1 shows normal values for creatinine and other parameters in pregnancy.

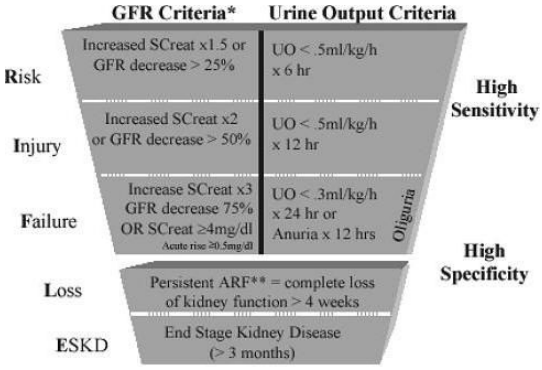

Figure 1. Diagnostic scheme for acute renal failure (ARF). The classification system includes separate criteria for creatinine and urine output. The criteria that lead to the worst classification should be used. Note that RIFLE-F is present even if the increase in serum creatinine (SCreat) is less than three-fold so long as the new SCreat is _4.0 mg/dL (350 _mol/L) in the setting of an acute increase of _0.5 mg/dL (44 _mol/L). The designation RIFLE-FC should be used in the case to denote "acute on chronic" disease. Similarly, when RIFLE-F classification is reached by urine output (UO) criteria, a designation of RIFLE-FO should be used to denote oliguria. The shape of the figure denotes the fact that more patients (high sensitivity) will be included in the mild category, including some who do not actually have renal failure (less specificity). In contrast, at the bottom, the criteria are strict and therefore specific, but some patients will be missed. GFR, glomerular filtration rate; ARF, acute renal failure. Reprinted with permission from Referece 2.

Table 1. Normal laboratory index in pregnancy

Index	Change and range in pregnancy
GFR	Increase about 40% over baseline
Creatinine clearance	Increase about 25% over baseline
BUN	9 - 10 mg/dl
Creatinine	0.5 – 0.8 mg/dl
Urineprotein	less than 300 mg in 24 hours urine
Plasma osmolality	decrease about 19 mOsm/kg

Traditionally, acute renal failure has been divided into three etiologies: prerenal, intrarenal and postrenal.

Early in pregnancy the most common problems are prerenal disease due to hyperemesis gravidarum or acute tubular necrosis, resulting from a septic abortion. Several different uncommon disorders can lead to acute renal failure later in pregnancy [5,6].

Because patients with chronic renal disease may present with worsening proteinuria, hypertension, and renal function, these disorders must be excluded from those conditions that cause acute deterioration of renal failure in otherwise normal women during pregnancy. Thus every patient presenting with acute renal failure during pregnancy should undergo a detailed history, physical examination, and laboratory assessment. Urine analysis and urine chemistry can be particularly useful in the categorization of the type of renal failure.

The overall incidence of Peripartum acute renal failure has decreased from 1/3000 to 1/15,000 –1/20,000. Similarly, the proportion of total cases of PR-ARF pregnancy has fallen from 20–40% in the 1960s to 2–10% in the 1980s [8].

In the developing world pregnancy related acute renal failure continues to account for 20% of total ARF cases and mortality rates remain as high as 50% [11].

The overall incidence of acute renal failure in pregnancy has decreased. Despite these declines, there has been little change in the overall mortality and long-term morbidity rates [7].

The consistency of mortality may be attributable to increased efficiency at prevention of cases of straightforward acute renal failure; the patients who continue to develop renal failure are sicker—often with multiple system organ failure [8]. Long-term prognosis has also remained fairly consistent over time, with full renal recovery rates of 60–90% [7].

Etiology

To identify the underlying etiology of ARF in pregnancy, it is important to consider both pregnancy-specific diagnoses as well as other etiologies relevant to reproductive age women that may be coincident with pregnancy.

Several general categories of pregnancy-specific ARF can be delineated: Hypovolemic ,Thrombotic microangiopathic, infectious and obstructive.

Hypovolemia:

Volume depletion can lead to ARF by causing prerenal ischemia. In pregnancy, the most common cause of volume depletion of this magnitude is obstetrical hemorrhage, which can occur at any gestational age.

Hyperemesis gravidarum is a common complication of pregnancy, with 70–85% of pregnant women experiencing some degree of nausea and vomiting [9] and 1–2% of women with severe symp-

toms,often including a loss of 5% of body weight [10].

In general, hyperemesis can be managed with oral antiemetic medications. In a small subset of patients, aggressive management with enteral and/or parenteral nutrition and hydration may be required. In rare cases, severe hypovolemia can result in prerenal ischemia and ARF.

Obstetrical hemorrhage can occur early in pregnancy due to spontaneous or induced abortion. More commonly, These processes can also be associated with consumptive coagulopathy, which can exacerbate the process, or disseminated intravascular coagulation, which can cause direct intrarenal damage.

Treatment of hemorrhage sufficient to cause prerenal ischemia includes volume support, replacement of blood products, and correction of coagulopathy. In the antepartum setting, delivery is indicated, either vaginally or by cesarean section, depending on the clinical picture from an obstetric perspective. In the postpartum setting, the underlying problem must be addressed. For surgical bleeding, exploratory laparotomy and repair may be indicated. In cases of uterine atony leading to hemorrhage, medical therapy with uterotonics, or if refractory, surgical intervention may be necessary [11].

Thrombotic Microangiopathy:

An important and difficult differential diagnosis is that of acute renal failure in late pregnancy in association with microangiopathic hemolytic anemia and thrombocytopenia. There are two main entities that must be considered: Thrombotic thrombocytopenic purpura-hemolytic uremic syndrome (TTP-HUS) and severe preeclampsia, usually with the HELLP syndrome (hemolysis with a microangiopathic blood smear, elevated liver enzymes, and a low platelet count) [12,13,14]. Sometimes glomerular involvements in the context of systemic lupus erythematous with or without antiphospholipid antibody syndrome, vasculitis syndromes and primary glomerular disease may present as thrombotic disease. The distinction between TTP-HUS and severe preeclampsia is important for therapeutic and prognostic reasons. However, the clinical and histologic features are so similar that establishing the correct diagnosis is often difficult. Most important are the history (e.g., preceding proteinuria and hypertension favor preeclampsia) and the time of onset [12,13,14]. Preeclampsia typically develops in the late third trimester, including the intrapartum period; only a few percent of cases develop in the postpartum period, usually in the first 24 to 48 hours. Preeclampsia does not occur before twenty weeks gestation in non-molar pregnancies.

Preeclampsia: Severe preeclampsia, which is much more common than TTP-HUS, is usually preceded by characteristic clinical features of hypertension, proteinuria, and severe edema. Renal failure is relatively unusual even with severe cases, unless there is significant bleeding with hemodynamic instability or marked disseminated intravascular coagulation (DIC). However, a mild degree of azotemia may occur, due in part to reduced permeability of the glomerular capillary wall [15]. Severe preeclampsia is

an indication for urgent delivery. The renal and extrarenal abnormalities typically begin to resolve spontaneously within two to three days postpartum and virtually complete recovery occurs within eight weeks postpartum [16]. In some cases, however, preeclampsia begins in the postpartum period without prior proteinuria and may be difficult to initially differentiate from postpartum HUS [14].Only the subsequent spontaneous recovery will point toward preeclampsia in this setting.

Generalized coagulopathy may be present when severe abruptio placentae, hepatic rupture, or liver failure complicate preeclampsia. Low levels of clotting factors, if present, are important diagnostically since they are almost always absent in TTP-HUS in which increased platelet consumption and thrombocytopenia are the primary abnormalities. for a review of the pathogenesis of these disorders which are felt to be part of a spectrum of disease, rather than distinct entities).

Thrombotic thrombocytopenic purpura-hemolytic uremic syndrome

TTP-HUS is characterized by the otherwise unexplained combination of thrombocytopenia and microangiopathic anemia, generally in association with renal disease [14].

these disorders occur in pregnancy at least as much as in the general population [17], and some authors report that pregnancy-related TTP/ HUS accounts for 10–25% of overall cases [18,19]. The overall incidence of TTP/HUS in pregnancy has been estimated to be 1/25,000 births [17].

Patients have been traditionally considered to have TTP when neurologic abnormalities are dominant and acute renal failure is minimal or not present, and considered to have HUS when acute renal failure is dominant and neurologic abnormalities are minimal or absent; however, these distinctions are frequently unclear and may not be important for management decisions. This clinical distinction may represent a true pathogenetic difference, as severe deficiency in the von Willebrand factor cleaving protease (ADAMTS13) is rarely present in patients with acute renal failure [20]. The thrombotic state in some patients antiphospholipid antibodies may be indistinguishable from TTP-HUS. The time of onset is variable. In one report of 13 pregnancies complicated by TTP-HUS, three developed before midpregnancy, eight peripartum, and two several weeks postpartum [21].

Postpartum disease may follow a normal pregnancy or be preceded by findings indistinguishable from preeclampsia [22,23]. ADAMTS13 levels tend to fall during the last two trimesters of pregnancy, which could contribute to the time course of development of TTP-HUS [24].

In addition to de novo disease, TTP-HUS that initially occurred in nonpregnant women has relapsed during a subsequent pregnancy and recurrent TTP-HUS has developed. However, most subsequent pregnancies are uncomplicated and most children survive [26].

The distinction between the HELLP variant of severe preeclampsia and TTP-HUS is difficult since both are characterized by microangiopathy and low platelet count. The presence of elevated liver

enzymes is strongly suggestive of HELLP syndrome, and is an uncommon feature of TTP-HUS. In contrast to patients with typical TTP, who have severe deficiencies of ADAMTS13, plasma levels of ADAMTS13 are only mildly or moderately reduced in patients with HELLP syndrome [27].

Acute fatty liver of pregnancy, a late pregnancy syndrome, has many features of both HUS-TTP and HELLP syndrome, and can be particularly difficult to identify,it is associated with acute renal failure in up to 60 percent of cases [28].

Most patients with renal failure have evidence of decreased renal perfusion or acute tubular necrosis Women with this disorder will have elevated liver enzymes, similar to those with HELLP syndrome; however, they frequently develop more severe azotemia and consumptive coagulopathy. The optimal therapy of TTP-HUS developing in association with pregnancy is the same as for patients who are not pregnant [29].

The patients were treated with plasma infusion with or without plasma exchange, similar to the regimen used in the management of other forms of TTP-HUS.The treatment of the HELLP variant of preeclampsia is also controversial. Delivery is indicated, and although some of the laboratory features may worsen immediately after delivery, they usually resolve within one week postpartum, without any specific therapy. Corticosteroids have been utilized with some success; however, large randomized clinical trials have not been performed.

Amniotic Fluid Embolism: Amniotic fluid embolism, a rare but dramatic clinical entity, can also be a cause of ARF durning pregnancy.Amniotic fluid embolism, which is also referred to as an anaphylactoid syndrome of pregnancy, is the acute onset of hypoxia, respiratory failure, cardiogenic shock, and DIC in pregnancy [30], occurring primarily during labor (70% of cases). The maternal mortality rate of amniotic fluid embolism is estimated to be _60%,with a similar perinatal mortality rate, and most survivors (85%) suffer neurologicsequelae [31]. Due to the syndrome's associated DIC, cardiac dysfunction, and hemorrhage causing intravascular volume depletion, ARF is a factor to consider in all immediate survivors, and careful attention to volume resuscitation is crucial.

Renal Cortical Necrosis: Bilateral renal cortical necrosis (or, in less severe cases, acute tubular necrosis) may be induced during pregnancy by abruptio placentae or other severe complications such as placenta previa, prolonged intrauterine fetal death, or amniotic fluid embolism [32,33]. Both primary DIC and severe renal ischemia (leading to endothelial damage and secondary fibrin deposition) have been proposed as the initiating event in this disorder. Affected patients typically have one of the above complications of pregnancy and then develop the abrupt onset of oliguria or anuria, frequently accompanied by gross hematuria, flank pain, and hypotension [32,33]. The triad of anuria, gross hematuria, and flank pain is unusual in the other causes of renal failure in pregnancy.

The diagnosis can usually be established by ultrasonography or CT scanning, which demonstrate hypoechoic or hypo-

dense areas in the renal cortex [32]. Renal biopsy or arteriography also can be performed, but these invasive procedures are not required in most cases. Renal calcifications on plain film of the abdomen also suggests renal cortical necrosis, but this is a late consequence of healing and is not visible for one to two months.No specific therapy has been shown to be effective in this disorder. Many patients require dialysis, but 20 to 40 percent have partial recovery with a creatinine clearance that stabilizes between 15 and 50 mL/min [33].

Postpartum Idiopathic ARF: Cases of ARF confined to the postpartum period that do not meet specific criteria for TTP, HUS, preeclampsia, or AFLP have been grouped into a category of idiopathic postpartum ARF [34,35]. Recently, it seems clear that these patients fall along the spectrum of Thrombotic microangiopathy with predominantly renal manifestations. There are no specific therapeutic principles regarding this entity; rather, patients should be treated according to their likely underlying etiology, with supportive care and plasmapheresis if clinically indicated [21,37].

Infection:

Infections leading to sepsis can result to hypotension and decreased renal perfusion, thus resulting in prerenal ischemia and potentially acute tubular necrosis. Pyelonephritis is the most common infectious complication of pregnancy. Although the incidence of asymptomatic bacteriuria is not increased in pregnancy, there is a higher likelihood of ascending infection and pyelonephritis [38].

Pyelonephritis can lead to sepsis and ARF in both the pregnant and nonpregnant host, there is an increased risk of ARF due to pyelonephritis in pregnancy independent of the presence or absence of sepsis [39].

These elevated risks can be attributed to several of the physiologic adaptations of the renal system to pregnancy, including ureteral dilation, bladder wall flaccidity, and increased sensitivity to bacterial endotoxin-induced tissue damage. The most common organisms involved in Pyelonephritis in pregnancy are Escherichia coli, Proteus mirabilis, and Klebsiella pneumoniae. Pseudomonas aeruginosa, group B streptococci, and enterococci are also potential pathogens [40].

Septic abortion was once a significant contributor to ARF in pregnancy but nowadays due to preventive measures it is a rare condition.

Obstruction:

The gravid uterus can provide significant compression of the genitourinary system, particularly in settings of uterine overdistention such as polyhydramnios, multiple gestation, or uterine fibroids. Although rare, there have been several case reports of PR-ARF in these scenarios [41–52].

Maternal genitourinary anomalies or prior surgery can also increase susceptibility to such processes, particularly in the setting of a unilateral kidney or collecting system [52].

Nephrolithiasis is another potential obstructive cause of ARF to consider, as the incidence of stones is the same as in a population of nonpregnant reproductive age women [53].

Approaches to treatment of obstructive PR-ARF include cystoscopy and retrograde ureteral stent placement or percutaneous nephrostomy [41, 42, 52]. If the clinical situation suggests that delivery would be appropriate, this may ameliorate genitourinary obstruction.[43, 46, 48–51]. Renal recovery is excellent and hemodialysis has been suggested as a temporizing measure until more definitive treatment of the obstruction can be achieved [49].

Conclusion:

Renal failure in pregnancy presents particular challenges, in that it occurs in a system physiologically altered from baseline and can occur due to disease processes that are specific to pregnancy and as yet incompletely understood.

It is crucial for physicians caring for these patients to have a broad knowledge of physiologic alterations in the renal system in pregnancy, to apply the best evidence-based diagnostic and therapeutic strategies for these disease processes, and to consider both maternal and fetal effects of disease and therapy.

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