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NARRATIVE REVIEW

Renal and cardiovascular repercussions in preeclampsia and their impact on fluid management: a literature review

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Abstract Preeclampsia is a multifactorial condition associated with significant morbidity and mortality. Fluid therapy in these patients is challenging since volume expansion may precipitate pulmonary edema, and fluid restriction may worsen renal function. Furthermore, cardiac impairment may introduce an additional component to the hemodynamic management. This article reviews the repercussions of preeclampsia on renal and cardiovascular systems and the development of pulmonary edema, as well as to discuss fluid management, focusing on the mitigation of adverse outcomes and monitoring alternatives. The literature review was carried out using PubMed, Embase, and Google Scholar databases from May 2019 to March 2020. Papers addressing the subjects of interest were included regardless of the publication language. There is a current trend towards restricting the administration of fluids in women with non-complicated preeclampsia. However, patients with preeclampsia may experience hemorrhagic shock, requiring volume resuscitation. In this case, hemodynamic monitoring is recommended to guide fluid therapy while avoiding complications.

Introduction

Preeclampsia is a multifactorial condition characterized by new-onset hypertension and proteinuria, or hypertension associated with significant organic dysfunction after 20-weeks of pregnancy.[1] Worldwide, it complicates 2% to 8% of pregnancies and is responsible for up to 14% of maternal deaths.[2,3] Preeclampsia is also a significant cause of morbidity; patients presenting with severe disease are at a higher risk of developing severe complications, including Acute Kidney Injury (AKI), cardiovascular diseases, and Pulmonary Edema (PE).[4]

The current knowledge in physiopathology of preeclampsia relies on abnormal placentation and imperfect invasion

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of the uterine spiral arteries by cytotrophoblast cells, causing inadequate blood flow and relative placental ischemia.[5,6] In consequence, a state of oxidative stress is established, leading to defective fetoplacental angiogenesis and endothelial dysfunction.[7]

The endothelial dysfunction in preeclampsia is associated with an increase in both peripheral vascular resistance and vascular permeability, which ultimately results in a state of relative intravascular hypovolemia.[8] Fluid therapy in this scenario may be challenging, especially during cesarean delivery. Although volume expansion may precipitate fluid overload and PE, fluid restriction may worsen tissue hypoperfusion and increase the risk of AKI (Fig. 1).[9–11] Furthermore, cardiac impairment may play an essential role in preeclampsia, introducing an additional component to the hemodynamic management.[12,13]

This article reviews the repercussions of preeclampsia on renal and cardiovascular systems, and the development of pulmonary edema, as well as to discuss fluid management, focusing on the mitigation of adverse outcomes and monitoring alternatives.

Methodology

The literature review was carried out by searching the PubMed, Embase, and Google Scholar databases from May 2019 to March 2020. Eligible studies were identified by using different combinations of the following search terms: ''preeclampsia'', ''pathogenesis'', ''acute kidney failure'', ''cardiovascular'', ''pulmonary edema'', and ''fluid management''.

Articles were initially selected after reviewing the title and the abstract. Papers addressing the subjects of interest were selected for a full review and included regardless of the publication language.

Preeclampsia-related renal repercussions

Preeclampsia is the leading cause of pregnancy-related AKI,[14,15] a condition associated with high rates of maternal mortality and fetal loss.[16] In the United States, between 1998 and 2009, up to 17% of deaths during hospitalized deliveries occurred among women with AKI.[11] Additionally, women who develop AKI during pregnancy, regardless of etiology, are at higher risk of poor outcomes, including obstetrical hemorrhage, placental abruption, and intensive care unit admission.[17,18] Approximately 2% of women with severe preeclampsia and 15% of women with HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome, a variant of severe preeclampsia, will develop AKI.[15,18–21]

The diagnosis of pregnancy-related AKI is challenging. During pregnancy, the physiological increase in Glomerular Filtration Rate (GFR) decreases the concentration of serum creatinine.[22,23] Thus, serum creatinine that is within the normal range for the general population may actually mask a significant impairment in renal function.[24] Pregnant women may experience 30% to 40% reduction in GFR before a significant increase in serum creatinine occurs.[15]

An additional issue is the absence of consensus regarding the diagnostic criteria for pregnancy-related AKI. Several classifications have been developed for application in the general population, including the RIFLE (Risk, Injury, Failure, Loss, and End-Stage Kidney Disease – ESKD)[25] and the AKIN (Acute Kidney Injury Network)[26,27] criteria. They are commonly used in the obstetric population, even without validation. The American College of Obstetricians and Gynecologists (ACOG) has its definition for AKI as a pregnancy-related hypertensive disorder (Table 1).[1]

AKI management during pregnancy is focused on providing supportive measures, dialysis, and correction of the underlying etiology.[28] Avoidance of nephrotoxic drugs and correction of complications, such as hypertension, hyperkalemia, and metabolic acidosis, should be promptly initiated along with judicious fluid management, aiming to provide adequate uteroplacental perfusion and fetal well-being while avoiding volume overload and PE.[29]

In the presence of uremic symptoms (encephalopathy, pericarditis, or neuropathy) or complications that are refractory to pharmacological interventions, renal replacement therapy is indicated. Dialysis prescription during pregnancy should minimize hemodynamic fluctuations; in this scenario, longer and more frequent sessions are preferred.[24,29] As many as 30% to 50% of those who develop AKI associated with HELLP syndrome will require dialysis temporarily.[30]

The identification of the underlying etiology of pregnancy-related AKI is essential; however, since several causes may share similar clinical and laboratory findings, the exact etiology may remain indefinite.[24] Regarding preeclampsia, diagnosis relies on the presence of Blood Pressure (BP) > 140/90 mmHg with proteinuria of $\geq$ 300 mg/day after 20 weeks of gestation in a previously normotensive woman or the evidence of organ damage.[31]

Specific treatment depends on the illness severity, fetal well-being, and gestational age. Before 24 weeks, pregnancy is discontinued because no fetal survival benefit is observed.[32] Between 24 and 32 weeks, expectant management is recommended[32,33]; after 32-weeks, delivery is the treatment of choice.[34] Development of severe preeclampsia, HELLP syndrome, or fetal compromise are indications for prompt delivery regardless of gestational age.[33]

In the past, preeclampsia-related AKI was considered completely reversed after delivery. Although the risk of ESKD after pregnancy is low, recent studies indicate that AKI during pregnancy increases the risk of long-term renal dysfunction.[18,22,35–37] A study that followed women with HELLP syndrome and AKI for up to one year after delivery reported that 21% of the patients needed dialysis.[35] Patients with preexisting hypertension or renal disease have a higher likelihood of requiring long-term dialysis.[38]

Preeclampsia-related cardiovascular repercussions

Preeclampsia and cardiovascular diseases share several predisposing conditions, such as obesity, smoking, sedentary lifestyle, diabetes, chronic kidney disease, chronic hypertension, and abnormal serum lipid profile.[39] For a long time, the overlap of risk factors for both illnesses was thought to be spurious. However, recent studies hypothesize that disorders of the cardiovascular system may have a direct effect

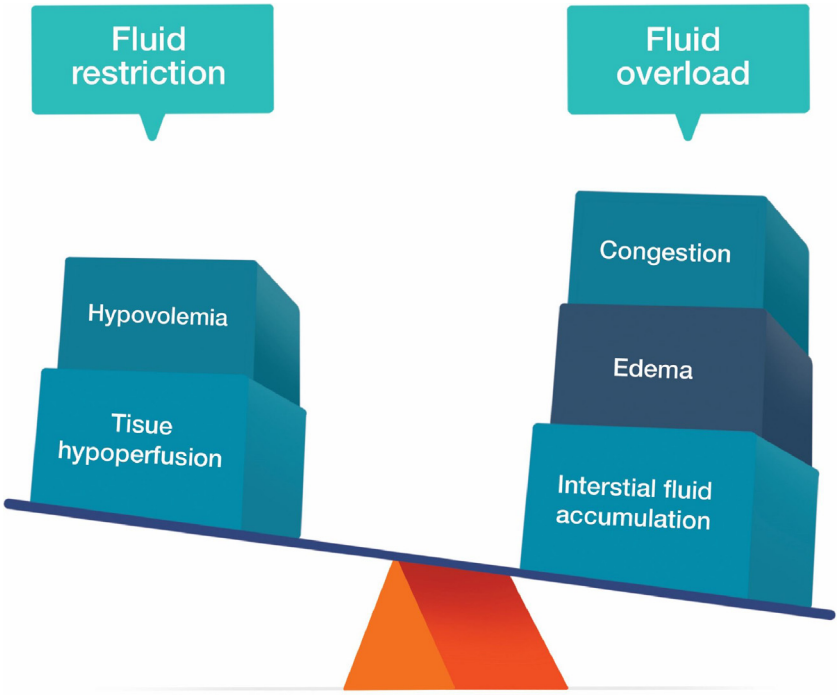

Figure 1 Considerations while choosing the fluid management strategy in women with preeclampsia.

Table 1 RIFLE, AKIN, and ACOG definitions for AKI.

RIFLE[25]	
Risk	1.5-fold increase in serum creatinine OR 25% decrease in GFR OR < 0.5 mL.kg^{-1}.h^{-1} for > 6 h
Injury	2-fold increase in serum creatinine OR 50% decrease in GFR OR UO < 0.5 mL.kg^{-1}.h^{-1} for > 12 h
Failure	3-fold increase in serum creatinine OR 75% decrease in GFR OR UO < 0.3 mL.kg^{-1}.h^{-1} for > 24 h OR no UO for 12 h
Loss of kidney function	Complete loss of kidney function (> 4 weeks)
ESKD	Complete loss of kidney function (> 3 months)
AKIN[26]	
Absolute increase in serum creatinine 0.3 mg.dL^{-1} or more OR 1.5-fold increase in baseline serum creatinine OR UO < 0.5 mL.kg^{-1}.h^{-1} for > 6 h	
ACOG[1]	
Baseline serum creatinine > 1.1 mg.dL^{-1} OR 2-fold increase in baseline serum creatinine in the absence of renal disease	

ESKD, End Stage Kidney Disease; GFR, Glomerular Filtration Rate; UO, Urinary Output.

on preeclampsia pathogenesis and that preeclampsia is an independent risk factor for cardiovascular disease.[12,13,40–42]

Preeclamptic women experience a derangement in renin-angiotensin-aldosterone system regulation when compared to their healthy counterparts. While refractoriness to angiotensin II is found in uncomplicated pregnancies,[43] an increase in vascular sensitivity to angiotensin II occurs in preeclampsia.[44,45] This imbalance, which is clinically expressed as high BP and peripheral arterial resistance, is present long before the diagnosis of preeclampsia.[39] Likewise, when compared to healthy women, those who develop preeclampsia may have a lower cardiac output and cardiac index, impaired myocardial relaxation, and increased prevalence of abnormal heart geometry even before conception.[46–48]

Besides the compelling evidence that preeclampsia has a cardiovascular etiology, the American Heart Association now recognizes that this condition is a risk factor for long-term postpartum cardiovascular diseases.[49] A cohort study that included over one million maternities demonstrated that preeclamptic women have a higher risk of major adverse cardiovascular events and that this risk remains significantly higher long after the delivery.[50] Another study found that recurrent preeclampsia is associated with increased rates of hypertension, ischemic heart disease, heart failure, and cerebrovascular accident.[51] There is also evidence for increased cardiovascular risk in the offspring of the preeclamptic mother.[52]

The cardiovascular management in uncomplicated preeclampsia is focused on BP control. Although BP thresholds and goals vary in international guidelines, there is a consensus that BP should be treated when it is severe (systolic BP $\geq$ 160 mmHg or diastolic BP $\geq$ 110 mmHg).[42] In complicated preeclampsia, delivery after stabilization of the patient is the only specific treatment.[31] During labor,

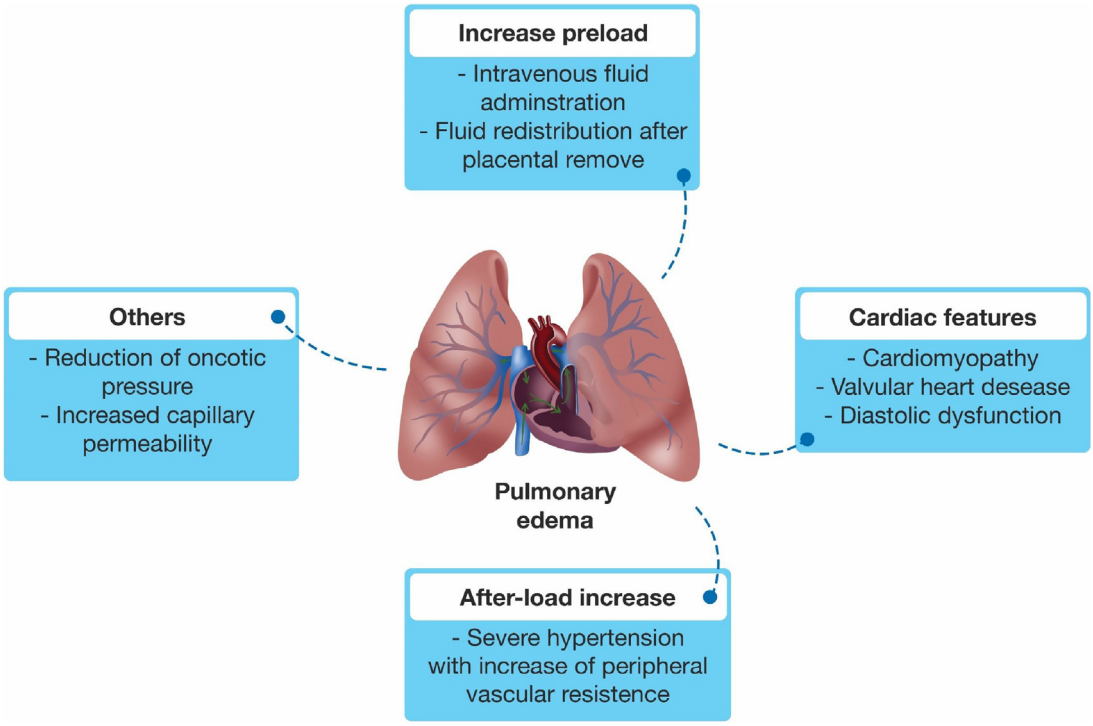

Figure 2 Multifactorial etiology of pulmonary edema in preeclampsia.

the use of invasive hemodynamic monitoring might be considered to guide fluid therapy, especially if severe cardiac disease, PE, persistent oliguria, and severe hypertension are present.[10,53]

In the postpartum period, women should be followed during the first 6 to 8 weeks; patients with a persistent need for antihypertensive medication require a referral to a cardiologist.[54] It is known that cardiovascular diseases have a slow progression, from asymptomatic to symptomatic stage. In this setting, the diagnosis of preeclampsia, which typically occurs in young women, poses an opportunity for early identification of high-risk patients when they are still in the asymptomatic stage. At this point, lifestyle and therapeutic interventions are more effective in controlling other cardiovascular risk factors.[42]

Pulmonary edema in preeclampsia

PE is the most common cardiopulmonary complication of preeclampsia, occurring in 3% to 5% of preeclamptic pregnancies, mainly in the peripartum or postpartum stage.[42,55,56] Despite the low rate of incidence, PE is a life-threatening event, being a frequent cause of admission to the intensive care unit and the leading cause of death among preeclamptic women.[57,58]

The etiology of PE in preeclampsia is multifactorial (Fig. 2). The decrease in oncotic pressure secondary to preeclampsia-related hypoproteinemia, the disruption in pulmonary endothelium leading to increased capillary permeability, and the increase in afterload due to severe hypertension all appear to play a role.[10,59] As previously discussed, preeclampsia may be associated with cardiac function impairment, which may contribute to PE.[60] PE is also related to preeclampsia severity, with higher rates among women who develop AKI, HELLP syndrome, and eclampsia.[18,61,62]

Nearly 70% of the PE events occur in the postpartum period.[61] Following the placental removal, a fluid shift from the extravascular space restores the intravascular volume, increasing the preload.[63,64] Furthermore, the inadvertent administration of fluid, frequently used to increase plasma volume or treat oliguria, may also exacerbate the preload and trigger PE.[10] It is recognizable that iatrogenic fluid administration is a major preventable cause of PE.[65]

The management of preeclampsia complicated by PE poses a significant challenge to the medical team. The superimposed issues of the physiological changes of pregnancy, the presence of the fetus, and the knowledge gaps regarding the physiopathology of preeclampsia all contribute to the high rates of morbidity and mortality associated with this condition.[10]

The initial goal while treating PE during preeclampsia relies on the reduction of BP with intravenous antihypertensive agents (Table 2).[42] Rapid reduction of blood pressure may be accomplished with the use of intravenous labetatol or hydralazine.[42,66] Some authors also recommend the use of nitroglycerine in hypertensive crises, despite the risk of aggravate the depletion of intravascular volume.[42,67,68] Furosemide should be used with caution because it may worsen the placental perfusion.[69]

In the case of left ventricular systolic dysfunction, inotropic support should be considered.[10] There is a gap in the literature regarding the choice of a specific inotrope to manage acute heart failure during pregnancy, especially due to safety concerns.[70] Therefore, the selection of an inotrope should be based upon the clinical scenario.

Table 2 Intravenous antihypertensive medications used in hypertensive pulmonary edema during preeclampsia.

Drug	Starting dose	Repeating doses and intervals	Maximum total dose	Comments
Labetalol[42,66]	20 mg	40 mg after 10 minutes	220 mg	Avoid in asthma, chronic obstructive airways disease, and heart failure; associated with neonatal bradycardia and hypoglycemia
Hydralazine[42,66]	5 mg	5–10 mg after 20 minutes	20 mg	Risk of sudden hypotension and maternal tachycardia; may need preloading or simultaneous fluid infusion
Nitroglycerine[10,42,67,68]	5 $\mu g.min^{-1}$	Gradually increase every 3–5 minutes	100 $\mu g.min^{-1}$	Considered the drug of choice by some authors; may aggravate the depletion of intravascular volume
Furosemide[10]	20–40 mg	40–60 mg after 30 minutes	120 mg	May worsen placental perfusion

Regarding ventilatory support, noninvasive modalities are initially preferred due to the risks associated with tracheal intubation in hypertensive pregnant women, such as intracerebral hemorrhage.[10,71,72]

After stabilization of the patient, consideration needs to be given to the delivery of the fetus if PE occurs in the antenatal period. Assessment of fetal well-being and multidisciplinary planning for safe birth is necessary.[10]

Fluid management and hemodynamic monitoring

Despite the lack of paramount evidence favoring a specific fluid management protocol, current guidelines advocate a restrictive approach, with additional fluid administration only recommended in selected scenarios (Table 3).[73–75]

Usually, preeclamptic women admitted for delivery may need fluid therapy for maintenance of water and electrolyte balance, or replacement of lost intravascular volume.[73] A routine fluid bolus should not be administered before neuraxial anesthesia, or to treat oliguria.[73,74,76,77] The available data do not suggest an absolute maternal or fetal benefit of colloids over crystalloids in preeclamptic women.[75,78,79]

Maintenance fluid therapy is required in uncomplicated patients who are expected to fast for several hours during labor. The infusion rate of crystalloids may be fixed 60 to 80 $mL.h^{-1}$, or calculated to match the Urinary Output (UO) combined with stool and insensible losses (lungs and skin).[73,80] It is also important to consider the volume used as a vehicle for drug administration when calculating the total amount of fluid. Given the low risk of complications following slow administration of intravenous fluids, hemodynamic monitoring may be based only upon clinical observation.[73]

Patients with severe preeclampsia may experience hemorrhagic shock due to several causes, such as placental abruption, operative blood loss, and rupture of subcapsular liver hematoma.[81–83] These women require immediate resuscitative measures, including intravenous volume replacement and blood typing.[84] The primary goal in this setting is to maintain a systolic BP above 90 mmHg.[73]

The shock index may also be considered as a predictor of adverse maternal outcomes. A threshold $\geq$ 0.9 indicates rigorous monitoring, $\geq$ 1.4 indicates an urgent need for intervention, and $\geq$ 1.7 indicates a high chance of adverse outcomes, including severe end-organ dysfunction and death.[85] UO is not a good predictor of fluid responsiveness in preeclamptic women and should not be routinely used as a therapeutic guide.[86]

The possibility of fluid overload and PE should be considered in the case of over-transfusion, especially when the fluid balance is above 2,000 mL.[58,73] However, inadequate resuscitation may increase the likelihood of AKI.[9] To balance the risks of both complications, hemodynamic monitoring is recommended to guide replacement therapy.[87]

Arterial line insertion is valuable in the presence of severe bleeding or difficult BP control. Other invasive methods, such as central venous pressure and pulmonary artery catheterization, are not routinely encouraged.[53,74] Ultimately, particular attention has been given to the use of noninvasive methods. Transthoracic echocardiography is recommended as a diagnostic and monitoring tool for hemodynamic complications, such as PE, severe arterial hypertension, and chest pain.[88] Lung ultrasound is another modality with growing importance. It can indicate both PE and increased left ventricular end-diastolic pressures.[89]

Conclusion and future directions

Preeclampsia is an important complication in pregnancy, resulting in significant rates of morbidity and mortality worldwide. Given its intricate and not still completely understood renal and cardiovascular repercussions, the management of fluid administration in preeclamptic women

Table 3 Intravenous fluid indications in preeclampsia.

Maintenance[73,80]	
60 to 80 mL.h^{-1} OR	Consider the volume used for drug administration
UO + stool + insensible losses	Monitoring based on clinical observation
Replacement[73,74,88,89]	
Titrate to systolic BP > 90 mmHg	Consider arterial line insertion if pressure control is difficult or there is severe bleeding
OR Shock index < 0.9	Consider noninvasive hemodynamic monitoring
Preload before neuraxial anesthesia[73,74,76,77]	
300 mL fluid challenge	Not routinely indicated
	Consider if high dose of anesthetic is administered
	Consider if hydralazine is the antenatal antihypertensive
Oliguria[73,74,77]	
300 mL fluid challenge	Not routinely indicated
Maintain UO $\geq$ 100 mL/4 h	If persistent oliguria, consider repeat the fluid challenge (in case of negative fluid balance)
	Consider noninvasive hemodynamic monitoring

BP, Blood Pressure; UO, Urinary Output.

can be challenging. Fluid restriction may precipitate or accentuate ischemic kidney lesions, while fluid overload may increase the hydrostatic pressure in the pulmonary capillaries, leading to PE.

Despite the lack of paramount evidence, there is a current trend towards restricting the administration of fluids in women with non-complicated preeclampsia. However, patients with severe preeclampsia may experience hemorrhagic shock, requiring volume resuscitation. In this case, hemodynamic monitoring is recommended to guide fluid therapy while avoiding complications. Noninvasive methods, such as transthoracic echocardiography and lung ultrasound, are preferred.

Future studies should focus on the influence of fluid management on patient outcomes. A randomized controlled trial could help to define the appropriate volume, as well as to describe which outcomes are significantly impacted by different volume management strategies. Additionally, basic sciences research could help to clarify the physiopathology of preeclampsia.

Conflicts of interest

The authors declare no conflicts of interest.

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