



FLUID MANAGEMENT IN PREECLAMPSIA

Tom Archer, MD, MBA, University of California, San Diego Holly Champagne, MSN, RNC, CNS, Kaiser Permanente, Roseville

BACKGROUND

Fluid management in preeclampsia is often difficult because of a leakage of water, electrolytes, and plasma from the intravascular space, due to underlying endolethial damage. This leakage can produce significant fluid shifts into the interstitial space resulting in peripheral and/or central (pulmonary and central nervous system, CNS) edema. As fluid shifts out of the intravascular space, there is also the potential for hypovolemia. Therefore, fluid administration must be assessed in the context of preserving organ perfusion, while limiting or preventing pulmonary edema. Renal endothelial damage appears to be particularly sensitive to these fluid changes resulting in proteinuria and oliguria. Assessment of renal function (serum creatinine) should be assessed to determine the degree of renal dysfunction. One hallmark of intravascular depletion is hemoconcentration.¹⁻⁴ Since pulmonary edema is more common and permanent renal damage due to preeclampsia is rare, fluids are normally restricted (see section related to Magnesium administration, pg. 50).⁵

If oliquria occurs, a trial of intravenous (IV) fluid bolus of 250-500 ml isotonic fluid (normal saline or Lactated Ringer's) can be given. If after a total infusion of 1000 ml of crystalloid IV fluids oliguria is not resolved, consideration is usually given to other modalities for enhancing urine output. If hypovolemia is still suspected then a trial of colloid IV fluid administration or blood may be used. Oxygen saturation monitoring is essential in these clinical settings due to the risk of pulmonary edema with excessive fluid administration in preeclamptic patients. If the patient is determined to be adequately hydrated, a trial pharmacological diuresis (furosemide, 5-10 mg IV) may be attempted. Oliguria, particularly when combined with excessive IV fluid administration, significantly increases the risk for pulmonary edema. Oliquria will also reduce the renal clearance of magnesium and the maintenance dose of magnesium will need to be adjusted to reduce the risk of magnesium toxicity. In the setting of oliguria and reduced O2 saturation (i.e. below 95%) diuresis is indicated. In rare instances, placement of a pulmonary artery catheter and measurement of pulmonary capillary wedge pressure may be needed. If these steps become necessary, the patient should be transferred to the intensive care unit for hemodynamic monitoring





KEY LEARNING POINTS

- 1. Patients with severe preeclampsia should have strict fluid intake and output monitoring assessments.
- 2. Total fluid intake (oral and intravenous) should be limited in both preeclampsia without severe features (mild) and severe preeclampsia. Many recommend that the sum of oral and all IV fluid should be ≤ 125 ml/hr (range 60 to ≤ 125 ml/hr) unless there are other clinical circumstances that dictate a different management plan.
- 3. Serum creatinine should be assessed in all patients with gestational hypertension, preeclampsia, or chronic hypertension with superimposed preeclampsia.
- 4. A Foley catheter with urometer is useful for monitoring urine output and is essential in the setting of oliguria or pulmonary edema.
- 5. An oliguric patient (less than 30 ml per hour for two hours, or less than 500 ml in 24 hours), should be given a limited trial of IV fluid boluses (normal saline or Lactated Ringer's), usually starting with 250-500 ml.
- 6. After a total of 1000 ml of crystalloid IV fluid is administered without resolution of the oliguria, and hypovolemia is still suspected, consideration may be given to the administration of colloid (e.g., albumin) or blood to enhance renal perfusion and urine output.
- 7. After adequate hydration, consideration should be given to the use of pharmacological diuresis (furosemide).
- 8. In the setting of oliguria and reduced O2 saturation (i.e., below 95%), pulmonary edema should be strongly suspected and diuresis is indicated.
- 9. Anesthesiologists should consider omitting or reducing the fluid bolus prior to epidural analgesia and ensure proper lateral positioning to avoid hypotension. Early treatment of hypertension has consistently been found to reduce the incidence of hypertensive crisis and will decrease the risk or prevent intracranial hemorrhage.^{2-4,6}
- 10. Cardiac dysfunction should be strongly considered in the presence of persistently low O2 saturations, the development of respiratory distress, persistent pulmonary edema, or unanticipated low BP's. In this setting, measurement of maternal Brain Natriuretic Peptide (BNP) and/or maternal echocardiography is strongly recommended to evaluate cardiac function and detect dilated cardiomyopathy.

EVIDENCE GRADING Level of Evidence: IIIC





REFERENCES

- **1.** Aukes A, Wessel I, Dubois A, Aarnoudse J, Zeeman G. Self-reported cognitive functioning in formerly eclamptic women. *Am J Obstet Gynecol*. 2007;197:365.e361--365.e366.
- 2. Postma I, Wessel I, Aarnoudse JG, Zeeman G. Neurocognitive functioning in women with a history of eclampsia: executive functioning and sustained attention. *American Journal of Perinatology.* 2010;27(9):685-690.
- **3.** Wiegman M, de Groot J, Jansonius N, et al. Long-Term Visual Functioning After Eclampsia. *Obstet & Gynecology.* 2012;119(5):959-966.
- **4.** Zeeman G, McIntire D, Twickler D. Maternal and fetal artery Doppler findings in women with chronic hypertension who subsequently develop superimposed preeclampsia. *J Matern Fetal Neonatal Med.* 2003;Nov(14(5)):318-323.
- **5.** Creasy R, Resnik R, Iams J, Lockwood C, Moore T. *Creasy & Resnik's Maternal Fetal-Medicine: Principles and Practice.* Vol Sixth Edition, Chapter 35, pg. 673. 2008.
- **6.** Creasy R, Resnik R, Iams J, Lockwood C, T M. *Creasy & Resnick's Maternal Fetal-Medicine: Principles and Practice.* Vol Sixth Edition, Chapter 35, pg 673-674. 2008.