



Severe Preeclampsia

Click the next button to continue...



Copyright © 2020 Shelly Betancourt and Michelle Becher

All rights reserved. No part of this publication may be reproduced, distributed, or transmitted in any form or by any means, including photocopying, recording, or other electronic or mechanical methods, without the prior written permission of the publisher, except in the case of brief quotations embodied in critical reviews and certain other noncommercial uses permitted by copyright law. For permission requests, write to the publisher at the address below.

Maternal 911 Education Systems, LLC
475 West Center St.
Ithaca, MI 48847
www.maternal911.com

Course Description:

Hypertensive disorders in pregnancy remains the leading cause of maternal death. The Maternal 911 Severe Preeclampsia module will give you a basis of knowledge to better recognize and treat preeclampsia. This knowledge base will help with communication to the patient and her family. The goal would be to increase the maternal safety for the unit where she will undergo care and delivery.

Approximate Time to Complete: 120 minutes



Click here to download a print version of this course.

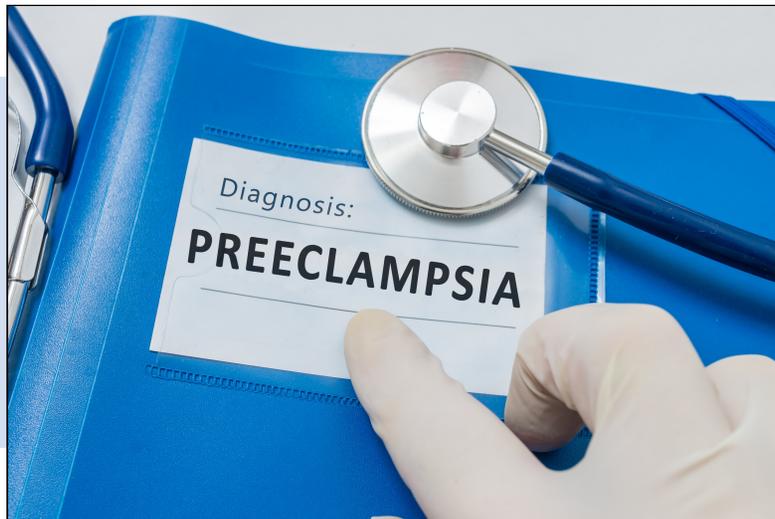


The purpose of this module is for the participant to:

- Explain criteria for preeclampsia, severe preeclampsia, and eclampsia.
- Identify risks associated with causing preeclampsia.
- Recognize the signs and symptoms of worsening preeclampsia so prompt health care delivery can be implemented.
- Describe the pathogenesis of preeclampsia.
- Identify clinical features and pathophysiology by organ system.
- Describe the medications used for resuscitation and how they may affect the woman and expected outcomes.

- ☰ Preeclampsia
 - ☰ Criteria for Preeclampsia
 - ☰ Urine Protein
 - ☰ Eclampsia
 - ☰ Occurrence Rates
 - ☑ Pathophysiology
 - ☰ Pathophysiology
 - ☰ Pathophysiology Cont'd
 - ☰ Signs and Symptoms
 - ☑ Clinical Features and Pathophysiology by Organ System
 - ☰ Clinical Features and Pathophysiology by Organ System
 - ☰ Reducing Mortality
 - ☑ Risk Factors
 - ☰ Risk Factors
 - ☰ Second or Third Trimester Risk Factors
 - ☑ Planning and Prevention
 - ☰ Planning and Prevention
 - ☰ Planning - Health History
 - ☰ Planning - Post Diagnosis Testing
 - ☰ Planning - Additional Tests
 - ☑ Management and Treatment
 - ☰ Management and Treatment
 - ☑ Recommendations of National and International Societies
 - ☰ Recommendations of National and International Societies
 - ☰ Common Order Set for Severe Hypertension





Preeclampsia involves multi-system progressive disorder characterized by the new onset of hypertension and proteinuria, or hypertension and end-organ dysfunction with or without proteinuria, in the last half of pregnancy or postpartum, often in a previously normotensive woman.

Criteria for Diagnosing Preeclampsia

Preeclampsia is defined as systolic BP > 140 mmHg or diastolic BP > 90 mmHg on two occasions at least four hours apart after 20 weeks of gestation in previously normotensive woman with Proteinuria < 0.3 grams in a 24-hour urine specimen or protein(mg/dL)/creatinine (mg/dL) ratio < 0.3 .

Severe preeclampsia is diagnosed when systolic BP is > 160 mmHg or diastolic BP is > 110 mmHg, confirmation within minutes is sufficient **AND** Proteinuria > 0.3 grams in a 24-hour urine specimen **or** protein(mg/dL)/creatinine (mg/dL) ratio > 0.3 .

Protein dipstick 1+ if a quantitative measurement is unavailable.



It is important to note the collection of 24 hour urine should not delay treatment.

The protein creatinine ratio is calculated with this formula:
(urine protein x 0.88) + (urine creatinine)

An online calculator can be found at:

<https://www.easycalculation.com/medical/urinaryprotein.php>

Eclampsia is diagnosed when grand mal seizures have occurred in a woman with no history of neurological conditions.

Seizures can occur before, during, or after delivery of the fetus.



Preeclampsia

- Is estimated to occur in 4.6 percent of pregnancies worldwide [1].
- The prevalence of preeclampsia in the United States (U.S.) is about 3.4 percent, but 1.5-fold to 2-fold higher in first pregnancies [2].
- In one population-based study, onset of preeclampsia ≥ 34 weeks is more prevalent than early onset, < 34 weeks [3].



Slide 1 of 3



Click the above arrows to see more information.





Both maternal and fetal/placental factors are involved in the pathophysiology of preeclampsia with both affecting the severity of the disease.



Critical components in the pathogenesis of preeclampsia include:

- Hypoperfusion
- Hypoxia
- Ischemia

The release of factors into the maternal blood stream alters the maternal endothelial cell function leading to characteristic systemic signs and symptoms of preeclampsia elaborating the hypoperfusion, hypoxemia and ischemia. [33-39].

The pathogenesis of preeclampsia has critical components of hypoperfusion, hypoxemia and ischemia leading to a variety of factors being released into the maternal blood stream altering maternal endothelial function and leading to characteristic systemic signs and symptoms of preeclampsia [33-39]

Hypoperfusion becomes more pronounced as pregnancy progresses since the abnormal uterine vasculature is unable to accommodate the normal rise in blood flow to the fetus/placenta with increasing gestational age [25-27]



It is unknown why the normal sequence of events, in development of the uteroplacental circulation, does not occur in some pregnancies.

The following are suspected to play a role:

- Vascular
- Environmental
- Immunological
- Genetic factors [40]





It is imperative to the health of the mother and fetus to communicate worsening signs and symptoms of worsening or severe preeclampsia to the provider if they are present:

- headache
- increasing blood pressure
- altered consciousness - restless, agitation, hallucinations, lethargy, confusion
- visual disturbances - floaters, blurred vision, spots, blind spots
- upper abdominal pain
- urine output < 30mL/hour
- shortness of breath
- complaints of chest pain
- SaO₂ <95 %
- cough
- tachypnea > 26 breaths/min
- tachycardia > 100 bpm
- adventitious breath sounds
- eclamptic seizure
- magnesium toxicity [163]



Cardiopulmonary

Hypertension may be the earliest clinical finding of preeclampsia and is the most common clinical indication to the presence of the disease.

Some women may develop hypertension rapidly or before 34 weeks of gestation or in the postpartum period.



The blood pressure usually rises gradually to $\geq 140/90$ mmHg.

- Often in the third trimester and after the 37th week of gestation [33].

A systolic blood pressure of ≥ 160 mm Hg or diastolic blood pressure of ≥ 110 mm Hg on two occasions at least four hours apart is a feature of severe disease [4].

Intravascular volume

A reduced volume is suspected to result from vasoconstriction due to enhanced responses of vasocative substances.

Intravascular volume may be reduced when severe features of preeclampsia are present.

The reduction in intravascular volumes has never been fully understood to date.

Edema

May be due to capillary leaking or represent "overflow" edema.

Edema itself does not indicate developing preeclampsia; many pregnant women have edema.

Further evaluation for preeclampsia is needed when the pregnant woman develops sudden, rapid weight gain of more than five pounds per week with facial edema.





CARDIAC FUNCTION

The myocardium is not directly affected, but the heart responds to physiologic changes caused by preeclampsia.

- Left ventricular ejection fraction usual remains within normal limits (WNL) [43].
- Left ventricular longitudinal, circumferential, and radial systolic strain have been observed [44].
- The reduction in left ventricular performance is a physiologic response to increased afterload [43-45].



Click through all four slides before moving on to the next page.

Slide 1 of 4



Pulmonary Edema

May be the presenting feature with severe preeclampsia

- The etiology is multifactorial [54-57]
- Pulmonary vascular hydrostatic pressure is elevated compared with plasma oncotic pressure that may cause edema
- Edema is present more in the postpartum period
- Not all preeclamptic women with pulmonary edema demonstrate these features

Other causes may include capillary leak, left sided heart failure, and unknown volume overload.



RENAL FUNCTION

Glomerular filtration rate (GFR) decreases by 30 to 40 percent in preeclampsia compared with pregnant normotensive women.

Renal plasma flow decreases but to a lesser degree.



Click through all six slides before moving on to the next page.

Slide 1 of 6



Hematologic

The most common coagulation abnormality in preeclampsia is thrombocytopenia.

Microrangiopathic endothelial injury lead to the formation of platelet and fibrin thrombi in the microvasculature.

Thrombocytopenia occurs due to accelerated platelet consumption, however, immune mechanisms may also play a role [74].

A platelet count less than 100,000/microl upstages the preeclampsia to severe preeclampsia.

The PT, PTT and fibrinogen concentrations are not affected unless additional complications occur such as placental abruption or severe liver dysfunction [75].



When hemolysis and reduced plasma volume are both present the hematocrit may be normal.

White blood cell (WBC) count may be slightly elevated due to neutrophilia.

The accelerated consumption of platelets leads to thrombocytopenia

- Immune mechanisms are thought to also play a role [74]

A platelet count of $< 100,000/\mu\text{mol}$ moves the patient to severe preeclampsia.

Concentrations of the following are not affected unless abruptio placenta or severe liver dysfunction is also present:

- Prothrombin time (PT)
- Partial thromboplastin time (PTT)
- Fibrinogen[75]



HEPATIC

Histologic findings observed in the livers of preeclamptic women [76,77]

- Periportal fibrin deposits
- Sinusoidal fibrin deposits
- Microvesicular fat deposits

Reduced hepatic blood flow can lead to

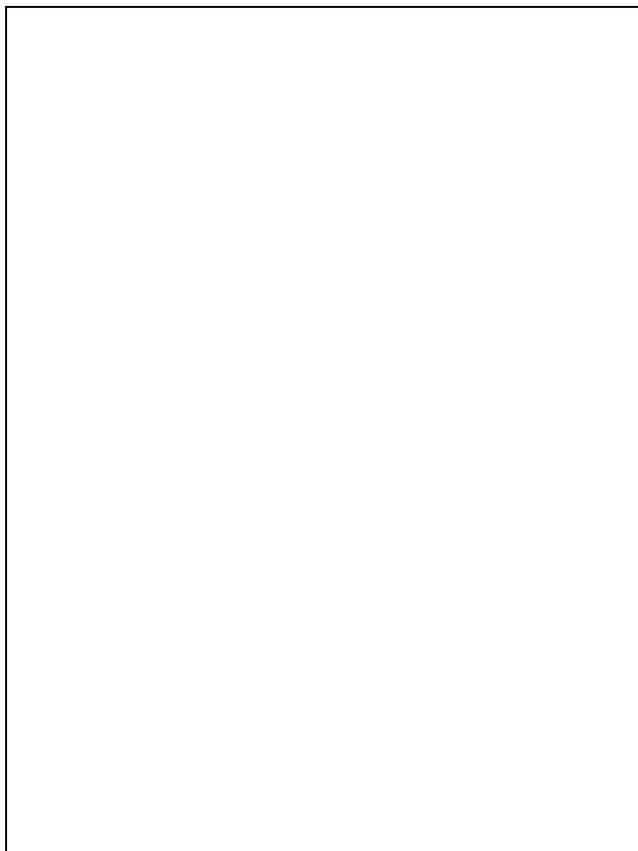
- Ischemia
- Periportal hemorrhage



Click through all five slides before moving on to the next page.

Slide 1 of 5





Central nervous system (CNS)

Central nervous system manifestations of preeclampsia include:

- Headache
- Visual symptoms
- Generalized hyperreflexia
- Sustained ankle clonus may be present

CNS

Headache may be

- Temporal
- Frontal
- Occipital
- Diffuse [79, 80]

Pain described as

- Throbbing or pounding
- Piercing

The headache is not relieved with over-the-counter (OTC) analgesics and worsens.

CNS - Eye

Visual symptoms

- Are caused by retinal arteriolar spasm [81]

Symptoms include:

- Blurred vision
- Flashing lights or sparks (photopsia)
- Scotomata (dark area or gaps in the visual field [82-84])
- Diplopia (blindness in one eye)
- Cortical blindness is rare and typically transient [85]

CNS - Eye

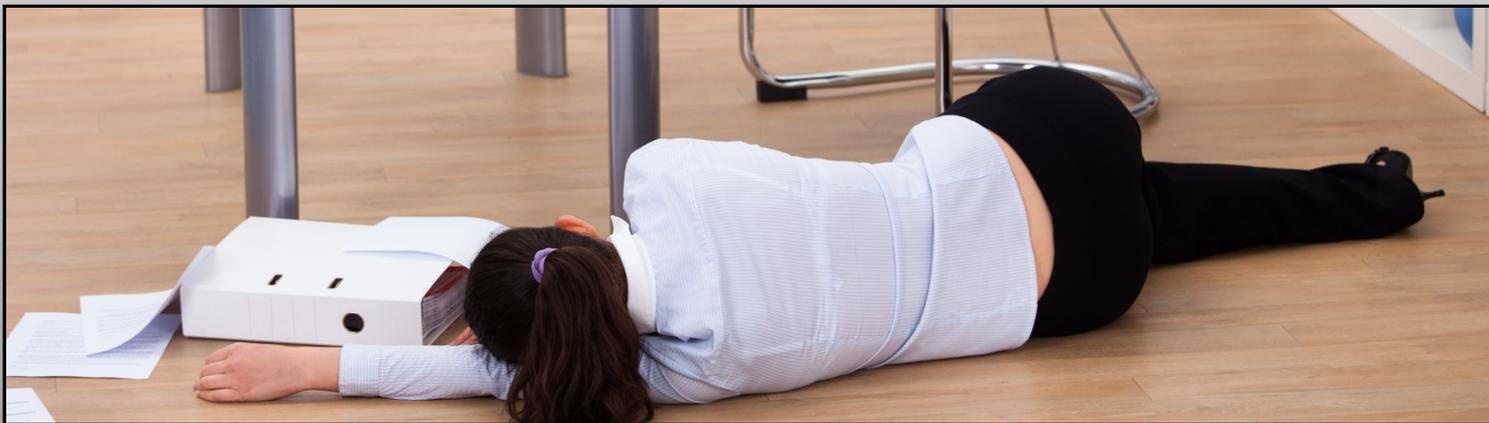
Blindness

- Blindness due to the following pathology may be permanent [86]
- Retinal artery occlusion
- Retinal vein occlusion
- Retinal detachment
- Optic nerve damage
- Retinal artery spasm
- Retinal ischemia

CNS - Seizures

When a seizure occurs in a woman with preeclampsia it signifies worsening of the condition. She is given the diagnosis of Eclampsia.

- 1 in 400 women with preeclamptic without severe features develop eclamptic seizures [4].
- 1 in 50 severely preeclamptic women will develop eclamptic seizures [6-12].



CNS – Cerebrovascular Manifestations

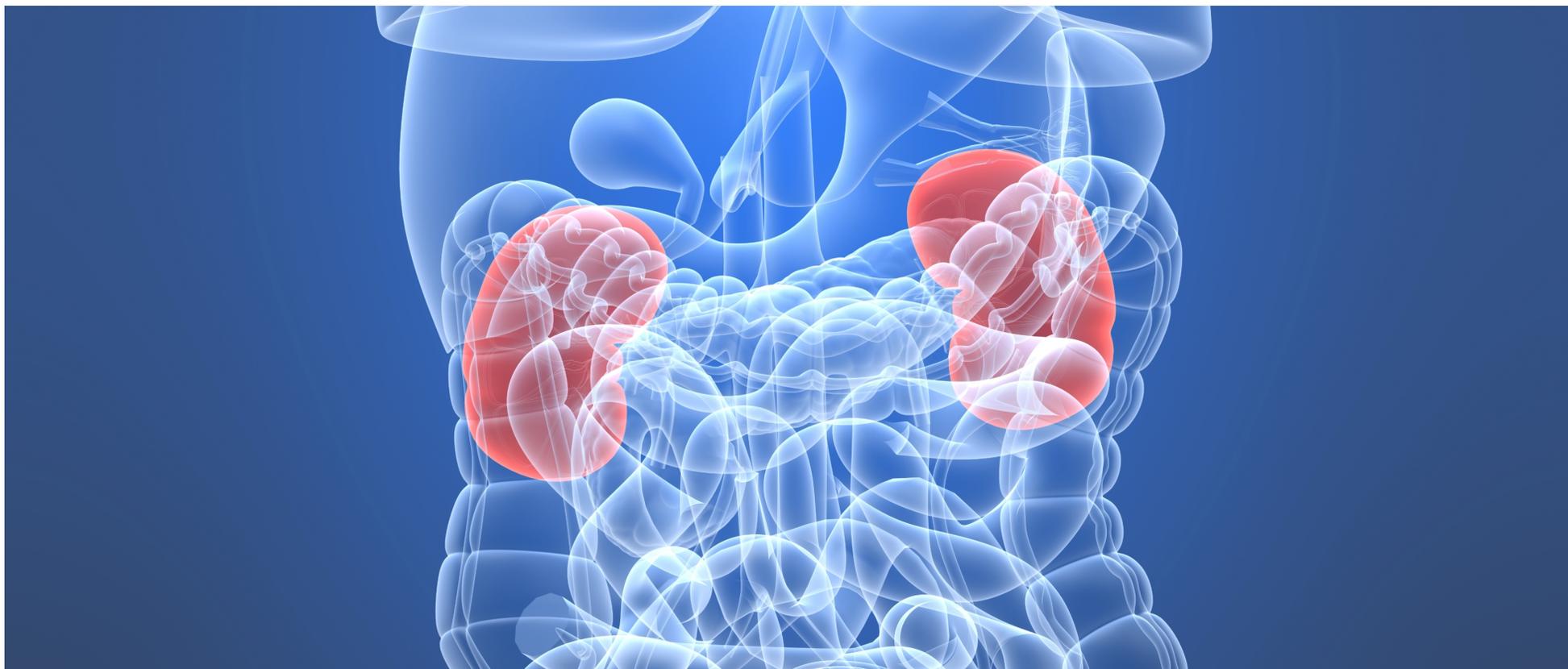
Are poorly understood.

Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) findings may include:

- Cerebral edema
- Cerebral ischemia
- Hemorrhagic changes [88, 89]

CT or MRI findings

- Identify generalized endothelial cell dysfunction
- May result from loss of cerebrovascular autoregulation
- Posterior reversible leukoencephalopathy syndrome (PRES) [90, 91]
- PRES is associated with severe hypertension but can progress quickly in a woman who has endothelial damage [92]



Click here to see more information.





MATERNAL 911
