VALLEYCARE OLIVE VIEW-UCLA MEDICAL CENTER/HEALTH CENTERS INPATIENT OBSTETRICS POLICY & PROCEDURE

NUMBER: 4816 VERSION: 1

SUBJECT/TITLE: HYPERTENSIVE DISORDERS OF PREGNANCY, MANAGEMENT OF:

MD ORDER: YES [X] NO []

POLICY: PATIENT CARE PROTOCOL/PROCEDURE

PURPOSE: To provide a guideline for the management of the antepartum, intrapartum and postpartum patient with Preeclampsia or Eclampsia. To provide a guideline for the administration of antihypertensive agents used to treat Preeclampsia or Eclampsia.

EXPECTED To prevent or minimize injury to mother and fetus.

DEPARTMENTS: Anesthesiology, Medicine, Nursing 3D- PostPartum/Nursery, Respiratory Care

DEFINITIONS:

OUTCOME:

PROCEDURE:

CARE DIRECTIVES

- I. Classification of Hypertensive Disorders of Pregnancy
 - A. Preeclampsia-Eclampsia
 - 1. Preeclampsia
 - a. Hypertension: $BP \ge 140/90 \ge 2$ six hours apart
 - b. Proteinuria: $\geq 1+$ on urine dip or $\geq 300 \text{ mg/}24 \text{ hrs}$
 - c. Onset usually after 20 weeks, resolves les than six weeks postpartum
 - 2. Eclampsia: New onset convulsions in a woman with preeclampsia without evidence of a neurologic disease
 - B. Chronic hypertension: Hypertension onset prior to pregnancy or before 20 weeks gestation and/or beyond 12 weeks postpartum
 - C. Preeclampsia or eclampsia superimposed on chronic hypertension
 - D. Gestational hypertension
 - 1. If in doubt whether a patient has preeclampsia versus

gestational hypertension; treat as if preeclampsia

- 2. Gestational hypertension can only be diagnosed with certainty retrospectively after the postpartum exam
- 3. Hypertension without proteinuria developing after 20 weeks and resolves less than 6 weeks postpartum

II. Significance

- A. Hypertensive disorders complicate about 12-22% of pregnancies
- B. Hypertension remains a leading cause of maternal and perinatal morbidity and mortality
- C. Preeclampsia is a multiorgan disease process with considerable clinical variation
- D. Maternal complications
 - 1. Neurologic sequalae
 - a. Cerebral edema
 - b. Brainstem herniation
 - c. Cerebral hemorrhage
 - d. Cerebral ischemia
 - 2. Hepatic hemorrhage/rupture
 - 3. Abruptio placenta
 - 4. Renal failure
 - 5. Disseminated intravascular coagulation (DIC)
 - 6. Pulmonary edema
- III. Etiology
 - A. The underlying cause of preeclampsia remains unknown
 - B. Proposed mechanisms include:
 - 1. Abnormal trophoblast invasion and remodeling of the spiral arteries
 - a. Normal pregnancy is associated with vascular changes in the blood vessels supplying the placenta such that the vessels become pliable leading to an increased perfusion to the placental site
 - b. These physiologic vascular changes do not occur in pregnancies complicated by preeclampsia resulting in a decreased blood flow to the placenta with poor perfusion of the fetal placental unit.
 - 2. Endothelial dysfunction
 - a. Pregnancy is normally associated with a decrease in vascular reactivity
 - b. Vascular reactivity is abnormal in preeclampsia, i.e. more prone to vasoconstriction
 - c. Preeclampsia is associated with platelet aggregation, release of vasoactive substances and intravascular coagulation all of which are signs of endothelial

damage

- 3. Prostaglandin imbalance: There is a relative excess of vasoconstrictive substancessuch as thromboxane and endothelins relative to the vasodilatory prostacyclin
- IV. Incidence

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- A. Overall affects 6 to 8% of pregnancies
- B. Risk factors include:
 - 1. Primigravida
 - 2. Extremes of age (< 18 years or > 35 years)
 - 3. New biological father
 - 4. Multiple gestation
 - 5. Preexisting hypertension (of any etiology)
 - 6. Renal disease
 - 7. Diabetes mellitus (preexisting or gestational)
 - 8. Prior preeclampsia
 - 9. Family history
 - 10. Antiphospholipid syndrome
 - 11. African American race
 - 12. Angiotensinogen gene mutation
 - 13. Obesity (increased body mass index)

V. Diagnostic Criteria

- A. Elevated Blood Pressure (BP)
 - 1. Systolic BP \geq 140mmHg and/or diastolic BP \geq 90mmHg on two occasions, six or more hours apart
 - 2. In some patients, the diagnosis of preeclampsia should or can be made in less than six hours
 - a. Intrapartum patient who has sustained systolic \geq 140mmHg or diastolic \geq 90mmHg
 - b. Those patients who have an eclamptic seizure or deliver in less than six hours
 - c. In cases where symptoms or signs suggest severe preeclampsia
- B. Significant proteinuria
 - 1. Suggested by urine dipstick
 - 2. A spot urine protein/creatinine ratio can be used as a screening tool for the quantitation of proteinuria with a protein/creatinine clearance ratio of 0.6 being a good predictor of 300mg or more proteinuria
 - 3. May consider 24 hour urine collection in select cases: \geq 300mg/24 hr
- C. Criteria for severe preeclampsia- is diagnosed in the setting of any of the following findings:
 - 1. Persistent systolic BP \geq 160mmHg and/or diastolic \geq

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- 110mmHg
- 2. Severe proteinuria: \geq 5 gms in 24 hour urine collection
- 3. Oliguria: \leq 30cc/hr or \leq 500 cc/24 hr
- 4. Pulmonary edema or cyanosis
- 5. HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets)
- 6. Eclampsia
- 7. Symptoms of headache, epigastric pain, visual disturbances
- 8. Intrauterine fetal growth restriction
- 9. Cerebrovascular accident

VI. General Management Strategies for Preeclampsia

- A. Preeclampsia is cured only by delivery. Any attempt at delaying delivery must be justified by clear benefits to the fetus/neonate without excessive risks to the mother.
 Anticonvulsants and antihypertensive therapies are utilized to prevent seizures and cerebrovascular accidents. Delivery is usually considered in cases of severe preeclampsia at or near term. Severe preeclampsia remote from term requires close daily maternal and fetal surveillance.
- B. Management issues to consider:
 - 1. Gestational age
 - 2. Administration of Antenatal Steroids
 - 3. Mild versus severe preeclampsia
 - 4. Outpatient versus inpatient management
 - 5. Timing of delivery
 - 6. Route of delivery
 - 7. Cervical ripeness
 - 8. Fetal presentation
 - 9. Sub-specialty consultations
- VII. Specific Management Strategies for preeclampsia
 - A. Perform a complete evaluation
 - 1. History and review of symptoms
 - 2. Physical exam
 - 3. Laboratory
 - a. Complete blood count (CBC), platelets
 - b. Electrolytes, serum creatinine, uric acid, AST, ALT
 - c. Coagulation studies: prothrombin (PT), partial thromboplastin times (PTT), fibrinogen if evidence of severe preeclampsia or platelets $\leq 100,000$
 - d. 24 hour urine collection for protein and creatinine clearance; may not be needed in those cases when delivery is likely within the next 24 hours
 - 4. Fetal assessment including ultrasound for fetal growth,

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antepartum testing for fetal well-being

- 5. Magnesium sulfate therapy for seizure prophylaxis. Give 4 grams IV over 20 minutes followed by continuous infusion of 2 grams/hr. Adjust rate based on symptoms, signs or serum magnesium levels, if ordered. Strict I's and O's during magnesium infusion.
 - a. Antepartum period while the patient is being evaluated
 - b. Intrapartum period
 - c. Postpartum period (first 24 hours)
- B. Determination should be made as to whether "mild" versus "severe" preeclampsia is present. Management should be determined based on the judgment of whether or not delaying delivery outweighs the risks of prematurity.
- C. Mild preeclampsia at term: Mild preeclampsia at or beyond 37 weeks gestation is generally best managed by delivery
 - 1. If the cervix is unfavorable, cervical ripening may be undertaken, followed by oxytocin augmentation as indicated
 - 2. Although arbitrary time limits are difficult to set, prolonged induction should be avoided, since the patient with preeclampsia may deteriorate quickly.
- D. Mild preeclampsia- preterm: Mild preeclampsia prior to 37 weeks should be managed expectantly. This approach includes:
 - 1. Modified bedrest in the lateral decubitus position to augment uteroplacental blood flow
 - 2. Daily maternal weight
 - 3. Daily assessment of urinary protein
 - 4. Blood pressure monitoring
 - 5. Periodic laboratory evaluations (every one to three days)
 - 6. Fetal surveillance twice weekly with NST, AFI or equivalent tests
 - 7. Close clinical observation for any signs or symptoms of worsening disease
 - 8. Neither antihypertensive agents nor diuretics have any role in the expectant antepartum management of mild preeclampsia
 - 9. Outpatient management may be considered in select cases, i.e., in mild preeclampsia where patient compliance and follow up can be assured. When in doubt, hospitalization is advisable
 - 10. Timing of delivery also can be individualized
 - a. If clinical findings remain consistent with mild disease, delivery can be delayed until or beyond 37 weeks gestation

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- b. Delivery prior to 37 weeks gestation should be undertaken when there is confirmed fetal lung maturity or evidence of disease progression to severe preeclampsia
- E. Severe preeclampsia at term (37 weeks gestation or greater): Delivery should be undertaken without delay
 - 1. Vaginal delivery is the preferred route
 - 2. Approach to cervical ripening and labor induction is similar to that described for mild preeclampsia at term
 - 3. Initial management plan should be achieved within 24 hours. In selected cases of rapidly progressing disease, clinical judgment may suggest that cesarean delivery is advisable to avoid the possible delay associated with a trial of labor
- F. Severe preeclampsia- preterm: The majority of such patients will require delivery because the risks to both mother and fetus outweigh the risks of premature delivery
 - 1. Recommend consultation with a Maternal-Fetal Medicine specialist
 - 2. In the extremely premature gestation (< 28-30 weeks), expectant management with meticulous in-hospital observation of maternal-fetal condition may be an option in select cases. Expectant management must be:
 - a. Individualized
 - b. Dependent on the criteria that determined severe preeclampsia
 - c. Determined by the patient's initial response to bedrest or treatment.
 - 3. It is also critical to assess the condition of the fetus by ultrasound and antepartum testing
 - 4. In cases where delivery is being delayed, therapeutic considerations include betamethasone to facilitate fetal lung maturity
 - 5. Sustained antihypertensive agents are not generally used in this management because they may mask further deterioration of the disease process. Continued or an increase in the dosage of anti-hypertensive medications may be an indication for delivery
 - 6. In those cases where preterm delivery is planned, labor induction remains an option, but proceeding to a cesarean section directly may be appropriate in cases where maternal and/or fetal condition suggests that further delay could result in increased morbidity and/or mortality. Neonatology consult is recommended
- G. Intrapartum management
 - 1. Encourage labor in lateral decubitus position (left or right)

- 2. Frequent monitoring of blood pressure
- 3. Strict measurement and recording of intake and output
- Consider placement of foley catheter if preeclampsia is 4. severe
- 5. Continuous fetal heart rate monitoring
- Magnesium sulfate therapy 6.
- 7. Laboratory evaluation
 - Obtain baseline studies described above a.
 - Repeat every 4-6 hours if preeclampsia is severe b. and every 12-24 hours if preeclampsia is mild
- 8. Labor induction is an option for both mild and severe preeclampsia. The time allowed to enter active labor and achieve delivery will vary depending on the acuity of the case, but usually should be limited to 24 hours. As noted, a prolonged induction is not advisable unless disease remains mild, with no signs of deterioration and labor progress is evident
- 9. Prevention of an eclamptic seizure
 - Eclampsia is an infrequent but serious complication a. of preeclampsia and is associated with significant morbidity
 - Etiology of eclampsia may be due to focal cerebral b. ischemia, cerebral hemorrhage, or cerebral edema
 - Large randomized controlled trials have c. demonstrated that Magnesium sulfate is the optimal agent for prevention of eclamptic seizures
- H. Postpartum care
 - Magnesium sulfate is usually continued for 24 hours 1. postpartum to prevent an eclamptic seizure
 - 2. Continual observation of the patient for symptoms and signs of worsening disease is necessary since complications can develop after delivery
 - a. Assess urinary output. Notify physician if < 30 cc/hr
 - DTRs hourly b.
 - BP and respiratory rate hourly c.
 - d. Check for proteinuria
- Management Strategies for the Complications of Preeclampsia: VIII. These complications occur infrequently but may be associated with major maternal and fetal morbidity and mortality. Consultation with a maternal-fetal specialist is recommended if any of the following complications occur:
 - A. Severe Hypertension
 - Goal of treatment is to prevent maternal cerebrovascular 1. event
 - Stroke a.
 - Eclamptic seizure b.

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- 2. Indications
 - Systolic BP \geq 160mmHg **OR** a.
- B. Diastolic BP \geq 110mmHg
 - 3. **Drug** Therapies
 - Hydralazine a.
 - i. Potent direct vasodilator
 - ii. Administered in IV boluses (by physician)
 - Does not require intra-arterial blood pressure iii. monitoring
 - Initial dose of 5mg IV over 1-2 minutes then iv. 5mg to 10mg IV every 20-30 minutes
 - Monitor BP every 5 minutes V.
 - vi. Maximal effect variable; occurs in 20 to 40 minutes
 - Maximum cumulative dose: 40mg vii.
 - Side effects: Hypotension, headache, viii. tachycardia, fluid retention, neonatal thrombocytopenia
 - b. Labetolol
 - i. Beta and alpha adrenergic blocker
 - 20mg IV bolus (by physician) ii.
 - Maximal effect in 10 minutes iii.
 - Check BP every 5 minutes iv.
 - If initial dose ineffective, repeat with V. increasing doses 20mg to 80mg every ten minutes
 - Maximum cumulative dose: 300mg vi.
 - Side effects: postural hypotension, nausea, vii. dizziness, dyspnea
 - Contraindications: Asthma, cardiac viii. compromise/failure
 - Nifedipine (not for acute management of c. hypertension)
 - Calcium channel blocker i.
 - ii. 5mg to 10mg PO, repeat in 30 minutes to a total of 60mg
 - Onset of action in 10 minutes and peaks in iii. 10 to 20 minutes
 - Side effects: headache, dizziness, iv. constipation, and tachycardia
 - 4. Hypertension unresponsive to first-line drugs
 - Consult with high-risk specialist including: a. Maternal-fetal medicine, intensive care, anesthesia or equivalent
 - Consider treatment with IV nitroprusside, b.

nitroglycerin or another effective drug for hypertensive emergencies (be cautious for cyanide toxicity)

c. If postpartum, transfer to intensive care unit or alternative setting where equivalent monitoring and care can be performed

C. Eclampsia

- 1. General management
 - a. Ensure airway and prevent aspiration; administer oxygen as needed
 - b. Protect from injury
 - c. Establish intravenous access
 - d. Monitor maternal vital signs: pulse, BP, respirations
 - e. Maternal pulse oximetry
 - f. Fetal assessment
 - i. Transient fetal heart rate (FHR) decelerations or bradycardia are common during an eclamptic seizure and usually resolve spontaneously when maternal stabilization is assured with an anticonvulsant and oxygen for in utero resuscitation
 - An emergent cesarean section is rarely needed and should only be undertaken in the setting of persistent FHR bradycardia and if maternal status allows
 - g. Eclampsia in the antepartum period warrants delivery, regardless of gestational age
- 2. Pharmacological Management of Eclampsia
 - a. Initiate or continue Magnesium sulfate therapy
 - b. If seizure occurs in a patient already on Magnesium sulfate:
 - i. Obtain serum magnesium level stat
 - ii. If seizure is self-limited, continue on Magnesium sulfate without additional therapy. Evaluate the patient for higher doses of Magnesium sulfate
 - iii. If seizure activity continues for 10 minutes consider treatment with one of the following drugs
 - 1. Lorazepam 2mg to 4mg slow IV push
 - 2. Diazepam 5mg IV
 - 3. Pentobarbital 125mg IV
- 3. Postpartum care
 - a. Initial close observation of maternal vital signs and urine output. Puerperal diuresis is the most accurate

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clinical indicator of resolution of preeclampsia/eclampsia. Late Postpartum Eclampsia or Posterior Reversible Encephalopathy Syndrome (PRES) are two distinct clinical entities associated with eclamptic seizures that occur 3 or 4 days to 4 weeks postpartum

- i. Clinical presentation of recent seizure, persistent headache, hypertension and nausea
- ii. Radiologic findings of vasogenic edema in the cerebral white matter, particularly in the parieto-occipital regions
- iii. Management includes anticonvulsant, antihypertensive and close observation

D. Persistent oliguria

- 1. Possible etiologies
 - a. Kinked or blocked foley catheter
 - b. Intravascular volume depletion
 - c. Cardiac compromise with decrease in cardiac output
 - d. Isolated renal artery vasospasm
 - e. Ureteral obstruction
- 2. Assess fluid balance
- 3. If delivery is imminent, careful observation without treatment is acceptable
- 4. If persistent, consider limited IV fluid challenge such as 500-1000cc Lactated Ringers or normal saline over 30-60 minutes
- 5. If urine output does not improve after fluid challenge, withhold further fluid boluses or give additional boluses very carefully to avoid precipitating fluid overload and possible pulmonary edema
- Persistent oliguria following fluid challenge warrants consideration for central venous catheter placement. Further fluid therapy can then be based upon hemodynamic findings.

E. Pulmonary Edema

- 1. The etiology of pulmonary edema can be multifactorial
 - a. Reduced colloid oncotic pressure
 - b. Increased vascular permeability
 - c. Increased intravascular hydrostatic pressure (cardiogenic pulmonary edema or fluid overload)
- 2. Diagnosis is based on clinical findings with confirmation on radiologic studies
 - a. Dyspnea, chest pain

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- b. Tachypnea, tachycardia, pulmonary rales
- c. Hypoxia
- 3. Management
 - a. 100% oxygen administration with mask
 - b. Pulse oximetry
 - c. Fluid restriction
 - d. IV furosemide 15mg to 20mg IV initially over one to two minutes. Larger doses of 40mg to 100mg IV may be required to promote adequate venodilation and diuresis
 - e. Central hemodynamic monitoring may be indicated to guide further management if initial steps fail to improve status
- F. HELLP Syndrome
 - 1. Occurs in 20% of cases with severe preeclampsia
 - 2. Clinical signs and symptoms are due to maternal hepatic vasospasm
 - a. Malaise, nausea, vomiting, epigastric pain
 - b. Hemolysis, elevated liver enzyme, low platelets
 - c. Complications
 - i. Intrapartum and postpartum hemorrhage
 - ii. Hepatic hematoma and/or rupture
 - iii. Placental abruption is infrequent
 - 3. Management
 - a. Delivery is indicated except in very rare cases with an extremely premature fetus and minimal laboratory abnormalities
 - b. Serial laboratory assessments in labor and postpartum to monitor platelet count, coagulation status and liver function
 - c. Rapid deterioration in terms of symptoms and laboratory findings may necessitate abandoning attempt at vaginal birth and proceeding to cesarean section
 - d. Platelet transfusion indicated infrequently but should be available
 - i. Platelet count <50,000 if cesarean delivery planned
 - ii. Platelet count <20,000 if normal vaginal delivery anticipated

EDUCATION

Instruct patient/family to report any of the following warning signs of

Preeclampsia:

- 1. Headache that does not go away after resting
- 2. Swelling of the face of hands
- 3. Pain in the upper stomach
- 4. Vision changes, such as seeing spots or blurred vision
- 5. Weight gain of three pounds in one week
- 6. Dizziness, shortness of breath, weakness, especially if on Magnesium Sulfate

DOCUMENTATION

Document the following:

- 1. All pertinent maternal and fetal assessments (including vital signs, FHT, UCs, etc.)
- 2. Initiations of all protocols used in patient care
- 3. All nursing and medical interventions and patient's response
- 4. Physician notification including indication and response
- 5. Any seizure activity and subsequent fetal and maternal response

APPENDIX A

Magnesium Sulfate Administration

Recommendation that this medication:

- Only be hung after two RNs have checked correct dosage and concentration
- Use of a Buretrol as additional safety measure
- Prepackaged, premixed solutions limiting concentrations
- Ideally 20 grams and 500 ml of fluid

Loading Dose: 4 grams of 10% solution IV slowly over 20 minutes

Maintenance Dose:

<u>IV Route</u> 2-3 grams per hour 10% solution <u>IM Route</u> 10 grams solution IM (5 grams per buttock with loading dose) 5 grams solution IM every 4 hours

Decrease or omit dose if: Reflexes are absent Urine output is less than 30cc/hr Respirations are less than 12/minute

PRECAUTIONS:

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Intravenous route (preferred route of administration)

- An infusion pump must be used to prevent fluctuations in rate of administration
- Reflexes, respirations and urine output should be checked every hour to assess effects of Magnesium sulfate
- Close observation is necessary. Blood values of Magnesium sulfate should be obtained if any signs or symptoms of Magnesium toxicity develop
- Rapid intravenous injection causes an uncomfortable sensation of warmth due to vasodilation
- Use only 10% solution for IV administration

Intramuscular route (associated with significant pain; rarely used)

- Reflexes, urine output and respirations should be checked before administering each IM injection
- A local anesthetic should be added to Magnesium sulfate to minimize pain of injection (procaine 1%, 0.5cc per injection)
- Use Z technique, deep into muscle of upper outer quadrant of buttock using long needle
- Pull plunger back each time to check for blood
- Knead buttock after injection to disperse solution. Alternate sides.

Antidote: Should be readily available

Calcium Gluconate 10%: 10cc of a 10% solution which provides a total dose of 1 gram of Calcium Gluconate

1.5-2.5 mEq/liter
4.0-7.5 mEq/liter
10 mEq/liter
15 mEq/liter
25 mEq/liter

Absence of tendon reflexes may indicate levels above the therapeutic range and if allowed to rise, may lead to respiratory and cardiac arrest.

References:		
PAC/LAC Prenatal and Intrapartum Guidelines of Care, 2009		
ACOG Practice Bulletin #33, January 2002 (reaffirmed 2010)		
Approved by: Marci Hamilton (Clinical Nurse Director)	Date: 01/30/2012	
Review Date: 01/30/2015	Revision Date:	
Distribution: Anesthesiology, Medicine, Nursing 3D- PostPartum/Nursery, Respiratory Care		
Original Date: 01/30/2012		

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