

ALGORITHM OF DIAGNOSTICS AND FIRST AID AT ECLAMPSIA

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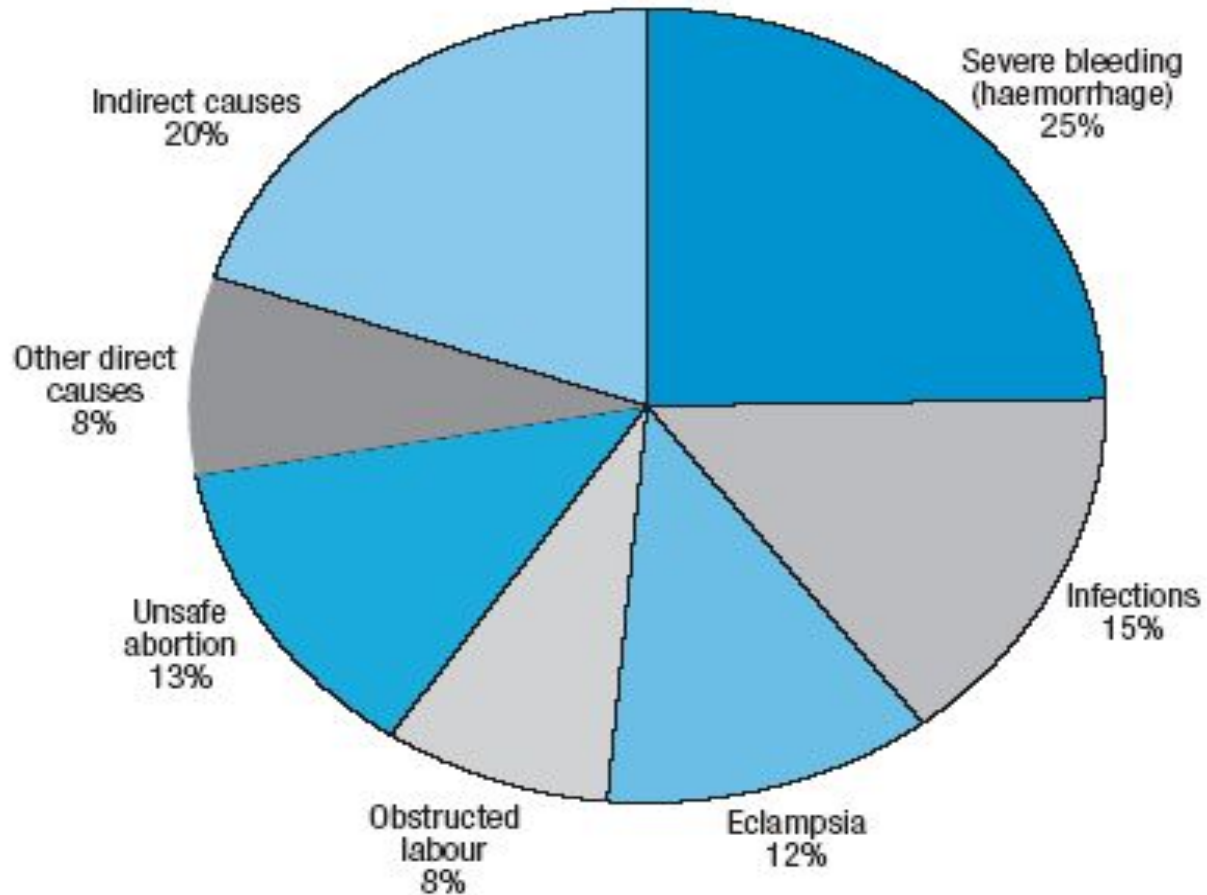


DEFINITION

- Eclampsia is an acute and life-threatening complication of pregnancy, characterized by the appearance of tonic-clonic seizures, usually in a patient who has developed pre-eclampsia

- Eclampsia includes seizures and coma that happen during pregnancy but are not due to pre-existing or organic brain disorders. Approximately 63,000 pregnant women die every year because of eclampsia and severe pre-eclampsia, which are also associated with a higher risk of newborn deaths. That is nearly 200 women every day.

Causes of maternal mortality



^a Total is more than 100% due to rounding.

SIGNS AND SYMPTOMS

- Typically patients show signs of pregnancy-induced hypertension and proteinuria before the onset of the hallmark of eclampsia, the eclamptic convulsion. Other cerebral signs may precede the convulsion such as nausea, vomiting, headaches, and cortical blindness.

- In addition, with the advancement of the pathophysiological process, other organ symptoms may be present including abdominal pain, liver failure, signs of the HELLP syndrome, pulmonary edema, and oliguria. The fetus may already have been compromised by intrauterine growth retardation, and with the toxemic changes during eclampsia may suffer fetal distress. Placental bleeding and placental abruption may occur.

SEIZURES

Chesley[2] distinguishes these four stages of an eclamptic event:

1. in the "stage of invasion" facial twitching can be observed around the mouth.
2. in the "stage of contraction" tonic contractions, or sustained muscular contractions without intervals of relaxation, render the body rigid; this stage may last about 15 to 20 seconds. (Tonic contractions are also known as tetanic contractions).

3. the next stage is the "stage of convulsion" when involuntary and forceful muscular movements occur, the tongue may be bitten, foam appears at the mouth. The patient stops breathing and becomes cyanotic; this stage lasts about one minute.
4. the final stage is a more or less prolonged "coma". When the patient awakens, she is unlikely to remember the event.

In some rare cases there are no convulsions and the patient falls directly into a coma. Some patients may experience temporary blindness upon waking from the coma.

During a seizure, the fetus may experience bradycardia.

RISK FACTORS

- Eclampsia, like pre-eclampsia, tends to occur more commonly in first pregnancies and young mothers where it is thought that novel exposure to paternal antigens is involved. Furthermore, women with pre-existing vascular diseases (hypertension, diabetes, and nephropathy) or thrombophilic diseases such as the antiphospholipid syndrome are at higher risk to develop pre-eclampsia and eclampsia.

- Having a large placenta (multiple gestation, hydatidiform mole) also predisposes women to eclampsia. In addition, there is a genetic component: patients whose mother or sister had the condition are at higher risk.[4] Patients who have experienced eclampsia are at increased risk for preeclampsia/eclampsia in a later pregnancy. Pulmonary edema is a rather common complication of severe eclampsia affecting approximately 3% of patients. Most cases result of aggressive use of crystalloid solutions for intravascular volume expansion.

PATHOPHYSIOLOGY

- While multiple theories have been proposed to explain preeclampsia and eclampsia, it occurs only in the presence of a placenta and is resolved by its removal. Placental hypoperfusion is a key feature of the process. It is accompanied by increased sensitivity of the maternal vasculature to pressor agents leading to vasospasm and hypoperfusion of multiple organs. Further, an activation of the coagulation cascade leads to microthrombi formation and aggravates the perfusion problem. Loss of plasma from the vascular tree with the resulting edema additionally compromises the situation. These events lead to signs and symptoms of toxemia including hypertension, renal, pulmonary, and hepatic dysfunction, and—in eclampsia, specifically—cerebral dysfunction. Preclinical markers of the disease process are signs of increased platelet and endothelial activation.

- Placental hypoperfusion is linked to abnormal modelling of the fetal–maternal interface that may be immunologically mediated. The invasion of the trophoblast appears to be incomplete. Adrenomedullin, a potent vasodilator, is produced in diminished quantities by the placenta in pre-eclampsia (and thus eclampsia). Other vasoactive agents are at play including prostacyclin, thromboxane A₂, nitric oxide, and endothelins leading to vasoconstriction. Many studies have suggested the importance of a woman's immunological tolerance to her baby's father, whose genes are present in the young fetus and its placenta and which may pose a challenge to her immune system

- Eclampsia is seen as a form of hypertensive encephalopathy in the context of those pathological events that lead to pre-eclampsia. It is thought that cerebral vascular resistance is reduced, leading to increased blood flow to the brain. In addition to abnormal function of the endothelium, this leads to cerebral oedema. Typically an eclamptic seizure will not lead to lasting brain damage; however, intracranial haemorrhage may occur.

DIAGNOSIS

- Seizures during pregnancy that are unrelated to pre-eclampsia need to be distinguished from eclampsia. Such disorders include seizure disorders as well as brain tumor, aneurysm of the brain, and medication- or drug-related seizures. Usually the presence of the signs of severe pre-eclampsia precede and accompany eclampsia, facilitating the diagnosis.
- Investigations: CBC, Renal function test (RFT), Liver function test (LFT), coagulation screen, plasma rate concentration, 24 hour urine analysis, ultrasound.

PREVENTION

- Detection and management of pre-eclampsia is critical to reduce the risk of eclampsia. Appropriate management of patients with pre-eclampsia generally involves the use of magnesium sulphate as an agent to prevent convulsions, and thus preventing eclampsia.

CONVULSIONS

- Prevention of seizure convulsion is usually done using magnesium sulphate.[11] The idea to use Mg^{2+} for the management of eclamptogenic toxemia dates from before 1955 when it was tested and published—the serum Mg^{2+} therapeutic range for the prevention of the eclamptic uterine contractions is still considered 4.0–7.0 mEq/L

As per Lu and Nightingale, serum Mg^{2+} concentrations associated with maternal toxicity (also neonate depression or hypotonia and low Apgar scores) are:

- 7.0–10.0 mEq/L: loss of patellar reflex
- 10.0–13.0 mEq/L: respiratory depression
- 15.0–25.0 mEq/L: altered atrioventricular conduction and (further) complete heart block
- >25.0 mEq/L: cardiac arrest

- Even with therapeutic serum Mg^{2+} concentrations, recurrent convulsions and seizures may occur—patients should receive additional $MgSO_4$ but with close monitoring for respiratory, cardiac, and neurological depression. The treat dosage is 4–6 g loading dose in 100 mL IV fluid over 15–20 min then 2 g/h as a continuous infusion.[3] If high Mg^{2+} concentrations fail to take effect, IV anticonvulsants will ease patient intubation and mechanical ventilation as adjuvants against the eclamptic convulsions (plus the hypermagnesemic thoracic muscleparalysis). The international MAGPIE study, of 2004, evaluated the long-term implications of the magnesium sulphate therapies.

TREATMENT

- The treatment of eclampsia requires prompt intervention and aims to prevent further convulsions, control the elevated blood pressure, and immediately deliver the baby if possible.
- Antihypertensive management
- Antihypertensive management at this stage in pregnancy may consist of hydralazine or labetalol

DELIVERY

- If the baby has not yet been delivered, steps need to be taken to stabilize the patient and deliver her speedily. This needs to be done even if the baby is immature, as the eclamptic condition is unsafe for both baby and mother. As eclampsia is a manifestation of a multiorgan failure, other organs (liver, kidney, lungs, cardiovascular system, and coagulation system) need to be assessed in preparation for a delivery (often a caesarean section), unless the patient is already in advanced labor. Regional anesthesia for cesarean section is contraindicated when a coagulopathy has developed.

Invasive haemodynamic monitoring

- Invasive haemodynamic monitoring may be useful in eclamptic patients with severe cardiac disease, renal disease, refractory hypertension, pulmonary oedema, and oliguria.

Management (carried out by a team)

1. Turn the patient on her side
2. Ensure clear airway (suction, mouth gag)
3. Maintain iv access
4. Stop fits (mag.sul, diazepam)
5. Control BP (hydralazine, labetalol)
6. Intake & output chart
7. Investigations (urine, FBC, RFT, LFT, clotting profile, cross match)
8. Monitor patient and her fetus
9. After stabilization (BP controlled, no convulsions, hypoxia controlled) deliver

THANK YOU FOR THE KIND
ATTENTION