Amniotic fluid embolism, venous thromboembolism, and eclampsia

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Summary
A recent report, released by UNICEF in 2009, highlights the risks faced during pregnancy and childbirth. In this report, the overall maternal mortality is 1500 women per day, although the highest rates of maternal mortality and severe morbidity can be found in developing countries, where prepartal and intrapartal care are compromised by limited resources. Pulmonary embolism is the leading cause of maternal death in the developed world with current estimates of 1.1 to 1.5 per 100 000 deliveries. Together with amniotic fluid embolism, pulmonary thromboembolism accounts for nearly 20 percent of perinatal maternal mortality in the United States. Venous thromboembolism often is fatal due to a delayed diagnosis or inadequate prophylaxis and treatment. In the United Kingdom, venous thromboembolism accounts for one third of all maternal deaths. Another serious complication in pregnancy is eclampsia with a maternal mortality ranging from 0 to 14 percent. The purpose of this article is to provide a practical approach to the diagnosis, management, and prevention of these complications in pregnant patients.

Introduction
Albeit the recent progress in screening, diagnostics, and therapy of the known causes of maternal mortality, thromboembolism, eclampsia as well as amniotic fluid embolism remain severe complications of pregnancy. According to the WHO, hypertensive diseases during pregnancy represent the leading cause for maternal mortality worldwide and cause approximately 50000 deaths per year (Waterstone et al. 2001). Amniotic fluid embolism and thromboembolism are responsible for approximately 20% of overall perinatal maternal mortality (Chang et al. 2003).

Thromboembolism

Incidence
In the western industrialized countries, thromboembolic complications are among the most frequent causes of mortality during pregnancy. The incidence of venous thromboembolism is 0.06% to 1.8% (Gherman et al. 1999). In pregnant women with predisposing factors (e.g. immobilization, HELLP syndrome or autoimmune diseases such as antiphospholipid syndrome or lupus erythematosus), the risk is sixfold higher than in nonpregnant women. Before the 20th week of gestation, deep venous thrombosis is more frequent, while 40% to 60% of pulmonary embolism cases occur during puerperium (Marik and Plante 2008).

Pathophysiology
During pregnancy, various thrombogenic mechanisms accumulate. The hormones cause dilatation of the venous vessels and – aggravated by the pressure of the growing uterus –this causes a reduction in the blood flow velocity of the pelvic veins. Simultaneously, pregnancy causes changes in coagulation which predispose to thrombosis (Virchow's triad during pregnancy, see Table 1)

Table 1: Virchow's triad during pregnancy

I. Dilatation of the venous vessels by reduced venous tonus (progesterone) and heavy pressure load
II. Reduced blood flow (70% in the third trimester)
III. Hypercoagulability by progressive increase in the procoagulant factors fibrinogen, factor VII, factor VIII, factor X; marked reduction in activity inhibitors of coagulation, e.g. free protein S and antithrombin

The purpose of these changes is the enhanced hemostasis after the delivery in order to prevent substantial maternal hemorrhage during placental expulsion and in order to prevent birth traumata. Thus, in combination with additional predisposing risk factors, thrombosis or embolism can occur (Barbour 2001). On the other hand, pregnancy also activates fibrinolysis.

Karl K. Amniotic fluid ... Gynakol Geburtsmed Gynakol Endokrinol 2009;5(3):194–204 published 31.11.09 www.akademos.de/gyn © akademos Wissenschaftsverlag 2009 ISSN 1614-8533

Classifying the risk for venous thromboembolism into low, intermediate and high has proven successful for the implementation of adequate preventive or therapeutic interventions (Bauersachs et al. 2007):

- low risk: thrombophilia or previous thrombosis (not pregnancy-related), antiphospholipid syndrome
- intermediate risk: thrombophilia and previous thrombosis, previous thrombosis during pregnancy or with oral contraceptives, antithrombin deficiency, two abortions with known antiphospholipid syndrome
- high risk: antithrombin deficiency or antiphospholipid syndrome and previous thrombosis, long-term anticoagulation, acute thrombosis during ongoing pregnancy

Additional risk factors such as long-term immobilization (e.g. fractures, preterm labor), a positive family history, additional thrombophilia (factor V Leiden, protein C deficiency, prothrombin mutation), increased maternal age (>35 years) as well as adiposity can further worsen the prognosis. Smokers have a highly increased risk for deep venous thrombosis (odds ratio [OR]: 1.3), stroke (OR: 1.7) or pulmonary embolism (OR: 2.5) (Roelands et al. 2009).

The risk for thromboembolism is fivefold higher during a cesarean section, and threefold higher in preeclampsia during puerperium.

The congenital thrombophilias known at present vary in their prevalences and bear differing risks for thrombosis. This has to be considered when consulting a patient (Table 2).

Table 2: Congenital thrombophilias and relative risk for thrombosis

Thrombophilia	Prevalence (%)	Risk (-fold)
APC resistance	2-15	5-10 (HEZ)
(factor-V-Leiden)		50-100 (HOZ)
Prothrombin gene mutation	1-2.5	2-3 (HEZ)
		50 (HOZ)
Protein C deficiency	0.2	10
Protein S deficiency	–	1.7-2
Antithrombin deficiency	0.02	> 10
Lupus anticoagulant	3.6	> 10
Hyperhomocysteinemia	5-10	2.5

hez: heterozygous; hoz: homozygous

Prophylaxis

Thrombosis or embolism prophylaxis should be implemented if a patient presents with several of the above-mentioned risk factors. The recommendations differ depending on the three mentioned risk groups (Bauersachs et al. 2007):

- low risk: clinical surveillance and non-pharmacological prophylaxis (compression stockings, sufficient fluid intake, prophylaxis before long flights, etc.), Dalteparin 50-100 IU/kg/day in the first two weeks after delivery or if additional risk factors are present
- intermediate risk: Dalteparin 50-100 IU/kg/day until six weeks after delivery
- high risk: Dalteparin 100-150 IU/kg/day until six weeks after delivery or until optimal dose adjustments of vitamin K antagonists

It is likely that during pregnancy, the dose will have to be adjusted to the weight. The effect of low-molecular-weight heparin (LMWH) is best assessed by factor Xa activity, as the partial thromboplastin time (PTT) is not influenced by low-molecular-weight heparin.

Clinic

When developing deep venous thrombosis (DVT) during pregnancy, development of edema with a change in diameter of both legs can become prominent. DVT affects the left leg in 70% to 90% of cases (Marik and Plante 2008). In addition, the affected patients often feel painful tension and unspecific paresthesia. Apart from cyanosis and increased phlebectasia, the typical clinical signs of DVT (e. g. Homan, Payr etc.) have to be excluded. Patients with pulmonary embolism may present with sudden onset of dyspnea and thoracic pain. Further symptoms such as hemoptysis, tachypnea and tachycardia will require further investigations (Chan et al. 2002).

Diagnostics

Deep venous thrombosis

When suspecting deep venous thrombosis during pregnancy, compression sonography should be performed immediately. If this method is not available, it is justifiable and advisable to immediately apply heparin in a therapeutic dose and delay the sonography.

In case of normal sonography, D-dimer testing is oftentimes recommended. However, it has to be taken into account that the D-dimer levels increase during pregnancy and thus carry no negative predictive value (Eichinger 2009).

In case of a positive result, magnetic resonance imaging (MRI), computed tomography (CT), both without contrast agents, or duplex sonography should be performed in order to exclude pelvic venous thrombosis (Marik and Plante 2008). Due to the radiation exposure, phlebography and CT should only scarcely be used during pregnancy. MR phlebography with direct detection of the thrombus by Met-Hb detection is not available ubiquitously. Scintigraphy has a relatively low radiation exposure, but results are often ambiguous. In a multisplice spiral CT, however, radiation exposure is acceptable and – for a mother with vital indication – is indicated during pregnancy.

The diagnostic algorithm when suspecting a deep venous thrombosis is depicted in Figure 1 (Marik and Plante 2008).

Pulmonary embolism

Compression sonography of the leg veins should also be performed when suspecting pulmonary embolism. In most cases, a chest X-ray in addition to the blood gas analysis (ASTRUP) will be necessary in order to verify pulmonary embolism. Another parameter for pulmonary embolism is the increase in the N-terminal pro B type natriuretic peptide (NT-proBNP), which indicates right heart failure following pulmonary embolism and thus is known as the late-onset marker for pulmonary embolism (Franz et al. 2008). Apart from an electrocardiogram as well as an echocardiography in order to exclude right heart failure or a heart defect, cardiotocography as well as doppler sonography should be performed when suspecting pulmonary embolism in order to monitor the fetus. The diagnostic algorithm when suspecting pulmonary embolism is depicted in Figure 2 (Marik and Plante 2008)

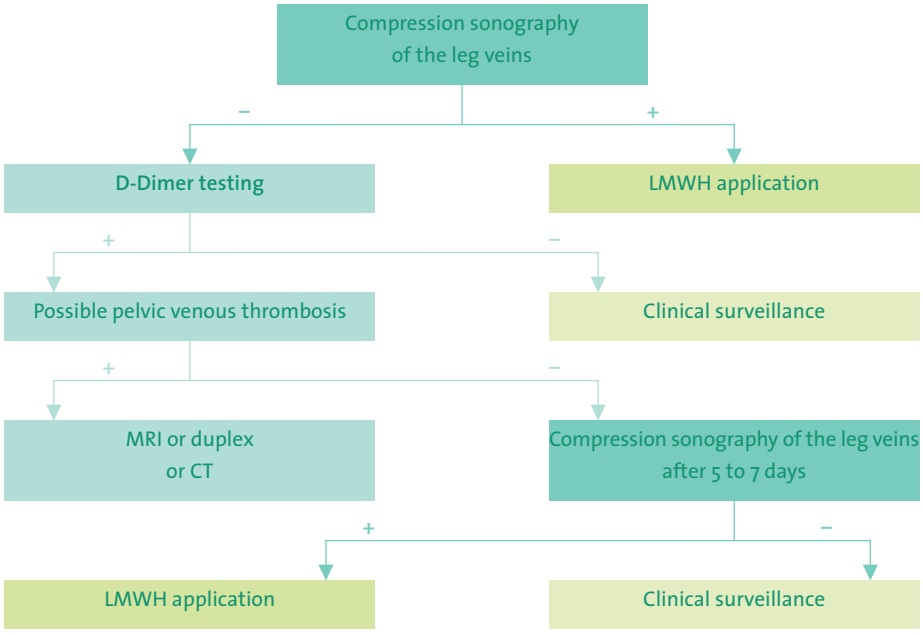

Figure 1: Algorithm when suspecting deep venous thrombosis (taken and modified from Marik 2008)

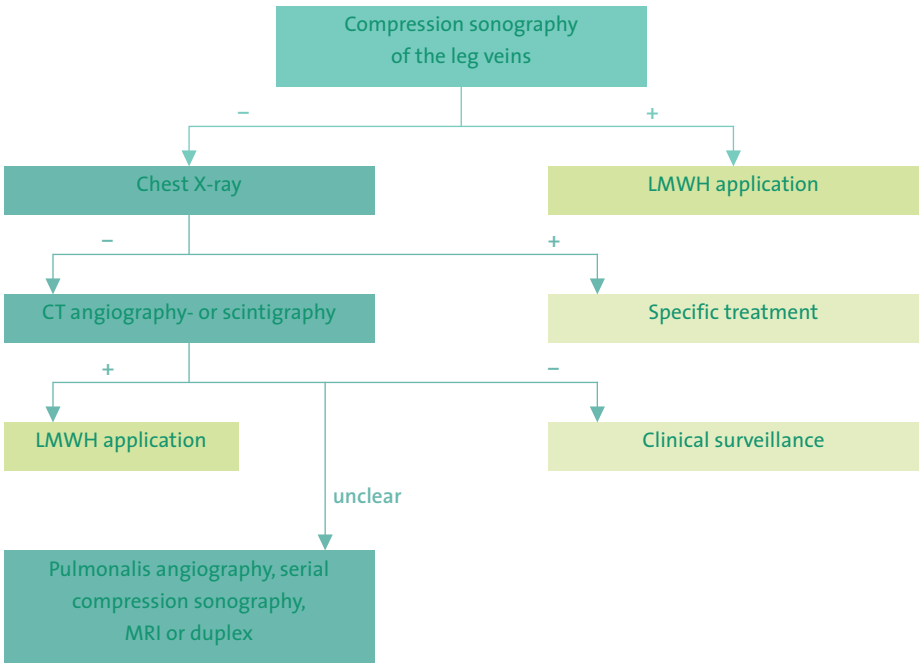

Figure 2: Algorithm when suspecting pulmonary embolism (taken and modified from Marik 2008)

Therapy

The therapy of deep venous thrombosis corresponds to that of nonpregnant women. Anticoagulant treatment can be implemented with the common substances, also during pregnancy. While cumarin derivatives are contraindicated in pregnancy due to their teratogenicity and their long half-life, heparin is safe for the fetus, as it cannot cross the placenta (first choice). Also low molecular weight heparin can be used, with the advantage of a longer half-life with the same effectiveness compared to unfractionated heparin. Thus, LMWH only have to be administered once a day, depending on the indication (prophylaxis or therapy). Scientific data clearly show a lower rate of the known heparin side effects (osteoporosis, heparin induced thrombocytopenia, hemorrhage). A disadvantage of LMWH is the accumulation in kidney failure, which requires careful dose adjustment with an anti-activated factor Xa test. The last option may be selective lysis by catheter technique, which can also be initiated during pregnancy. Alongside this, thrombectomy procedures have been proposed as a successful method during pregnancy but also in the peri- and postpartal phase. It is necessary to completely reconstruct the deep veins for the elimination of an embolism source and in order to avoid postthrombotic syndrome (Pillny et al. 2003).

The aim of the therapy of pulmonary embolism is the elimination of hypoxia, hemodynamic stabilization and the prevention of recurrent embolism. Pulmonary embolism is mortal for 15% of all pregnant women. With adequate therapy, this rate has been reduced to below 1%. Thus, also in the clinical suspicion of pulmonary embolism, all necessary therapeutic interventions have to immediately be implemented and documented together with the anesthesiologist or intensivist.

Peripartal management is of critical importance; unfractionated heparin is the adequate therapy due to its shorter half-life. After the administration of a low dose, an epidural or spinal anesthesia may be started after four hours. If LMWH is used in prophylactic doses, the 12-hour-limit applies, while in therapeutic doses, the 24-hour-limit is valid.

Eclampsia

Incidence

Due to improved diagnostic and therapeutic measures, the prevalence of eclampsia in industrial countries is declining. In Great Britain, the prevalence has dropped from 8 per 1000 to 0.5 per 1000 births (Douglas and Redman 1994).

While in the industrial countries the incidence of eclampsia is 0.3 to 0.5 in 1000 deliveries, the incidence varies from 6 to 100 per 10000 deliveries in the developing countries (1988).

Known risk factors are preexisting high blood pressure and nulliparity, multiple pregnancies, preeclampsia during previous pregnancies, adiposity as well as diabetes mellitus (Hernandez-Diaz et al. 2009; Odegard et al. 2000).

Pathophysiology

Similar to preeclampsia, the etiology of eclampsia is yet unknown. Two hypotheses are currently being discussed (Morriss et al. 1997):

- High blood pressure is thought to cause spasms and cerebral ischemia by microthrombus formation in the small intracranial blood vessels
- By the loss of autoregulation of cerebral perfusion, cerebral edema as well as intracerebral bleeding develop.

Prediction und prevention

In order to evaluate the risk for the development of preeclampsia, anamnestic information and duplex sonography performed in the second trimester can be helpful. Apart from a positive familial history, diabetes mellitus type 1, chronic hypertension or kidney diseases, previous preeclampsia is known to be a great risk factor (Hernandez-Diaz et al. 2009).

If, in between week 22 and 24 of gestation, the doppler sonography of the uterine arteries is bilaterally abnormal, preeclampsia is to be expected in more than 60% of patients in the course of pregnancy (Yu et al. 2005).

For the prevention of preeclampsia, the administration of acetylsalicylic acid (100 mg/d) is an established measure in early pregnancy in Germany. Prevention is especially advantageous for women with severe preeclampsia. It has reduced the risk for preeclampsia by 19% and reduced perinatal mortality by 16% (Knight et al. 2000).

Clinics and diagnostics

Hypertensive diseases during pregnancy are classified into:

- gestational hypertension (blood pressure ≥ 140/90 mmHg without proteinuria in pregnant women that were normotensive before)
- HELLP syndrome (triad of hemolysis, pathologically increased liver parameters and decreased thrombocyte count <100000 thrombocytes/µl),
- preeclampsia and eclampsia.

Gestational hypertension has to be differentiated from chronic hypertension, which is already diagnosed before the 20th week of gestation. Preeclampsia is defined by the presence of hypertension, proteinuria ≥ 300 mg/24 hours with or without edema after the completed 20th week of gestation. Eclampsia is characterized by the appearance of tonic-clonic seizures in patients who had developed preeclampsia and in whom the seizures cannot be assigned to another cause.

Thus, eclampsia is a clinical diagnosis. It is based on the existence of one or more generalized seizures or coma in women with previous preeclampsia who have no additional neurological symptoms. Eclampsia is directly connected to gestational hypertension. However, it does not have to be the consequence of an especially severe hypertension. The obligatory symptoms of preeclampsia, i.e. hypertension and proteinuria, may be less pronounced (Douglas and Redman 1994). Possible prodromal symptoms such as frontal and occipital headaches, photophobia, right-sided pain in the upper abdomen, and an altered mental state may be absent. In eclampsia, generalized tonic-clonic seizures occur in addition to preeclampsia symptoms. Other organic causes such as epilepsy or intracerebral space-occupying lesions have to be excluded, however, this is often difficult in an acute seizure and is usually time consuming.

Eclampsia-related seizures are usually self-limited and seldom remain for more than three to four minutes. Especially dreaded are life-threatening complications such as maternal intracerebral bleeding, abruptio placentae and intrauterine death (Sibai 1990).

Eclampsia cannot only occur during pregnancy or delivery but also during puerperium. The prevalence is 5% to 39% within the first 48 hours after delivery and 5% to 17% after more than 48 hours (Sibai 2005; Tuffnell et al. 2005).

Therapy

Immediate induction of therapy in eclampsia includes the prevention of maternal hypoxia and traumata, antihypertensive therapy, the avoidance of recurrent seizures and the immediate induction of delivery in pregnant patients.

Even if data from randomized studies on the necessity of acute blood pressure reduction are not available, the AMWF guidelines ("Diagnostik und Therapie hypertensiver Schwangerschaftserkrankungen") recommend antihypertensive therapy on maternal indications if the blood pressure reaches values of above 170/110 mmHg (Brown et al. 2000). Possibilities of pharmaceutical therapy (see Table 3) include the administration of an initial dose of Nifedipin 5 mg p.o., if necessary repeated after 20 minutes, or the administration of an intial i.v. bolus of Urapidil 6.25-12.5 mg, followed by a dose of 3-24 mg/h in the perfusor. The DGGG guidelines for the pharmaceutical acute therapy recommend i.v. dihydralazin 5 mg i.v. every 20 minutes or 5 mg i.v. as a bolus with a subsequent dose of 2-20 mg/h in the perfusor.

Table 3: Acute therapeutic intervention in severe hypertensive pregnancy diseases: Substances and dosages (DGGG guidelines of the "Arbeitsgemeinschaft Schwangerschaftshochdruck/Gestose")

Antihypertensive therapy	Nifedipin	Initial dose 5 mg p.o., if necessary repeated administration after 20 minutes
	Or Urapidil	Initial dose 6.25-12.5 mg as i.v.bolus injection for 2 minutes, then 3-24 mg/h (perfusor)
	alternatively: Dihydralazin	5 mg i.v. every 20 min or 5 mg i.v. as bolus und afterwards 2-20 mg/h (perfusor)
Anticonvulsive therapy	Magnesiumsulphate	4-6 g i.v. for 15-20 min maintenance dose 1-2 g/h until 24-48 h post partum
	Second choice: Phenytoin	250 mg i.v.
In pulmonary edema/ Cardiac insufficiency	Furosemide	110-20 mg i.v., if necessary repeated with an increased dose

Magnesium is known to be the first choice in prevention as well as therapy of eclampsia. In a Cochrane analysis, magnesium administration was superior to the administration of Diazepam (5-10 mg i.v.) (Duleyand Henderson-Smart 2003). In animal experiments, the systemic application of magnesium counteracted noradrenalin-induced vasoconstriction and promoted brain perfusion without influencing blood pressure (Perales et al. 1997). Vasorelaxation caused by magnesium sulphate is especially important during pregnancy.

The initial dose of magnesium sulphate is an intravenous bolus injection of 4 to 6 g for 15 to 20 minutes. As a sustaining dose, 1-2 g per hour administered by a perfusor, are sufficient. Due to the central side effects in an overdose, especially the tendon reflexes and respiratory frequency have to be strictly monitored. Paralysis of the skeletal muscles can occur from a plasma concentration of 5.5 mmol/l onwards and is the consequence of a reduced release of acetylcholine into the synaptic cleft. Magnesium sulphate can also cause kidney dysfunction. It may be necessary to reduce magnesium intake or transiently terminate the intake if severe oliguria develops. In an overdose, cardiac arrest may occur. Here, calcium gluconate serves as an antidote (1 g, slowly administered intravenously).

During or immediately after an eclamptic seizure, fetal bradycardia may develop. Instead of an emergency cesarean section in this situation, the stabilization of the mother with anticonvulsives, oxygen administration and, if necessary, antihypertensive drugs, is recommended, as this can lead to the intrauterine calming of the fetus. Transient fetal tachycardia, also with decelerations, has been described after such maternal seizures (Paul et al. 1978). If, within the first 10 to 15 minutes, no improvement of the fetal situation can be achieved, immediate delivery by cesarean section has to be initiated (Sibai 2005). An established therapeutic aim is the delivery in the seizure-free interval, i.e. after hemodynamic stabilization of the pregnant woman. However, especially at early gestational age, observational behavior in order to await fetal maturation and lung maturation is justifiable in an eclamptic seizure.

Amniotic fluid embolism ("anaphylactoid syndrome of pregnancy")

Incidence

The exact incidence of amniotic fluid embolism is unknown but estimated to be between 1 in 8000 and 1 in 80000 births (Clark et al. 1995). Associations with quick deliveries, advanced maternal age, high multiparity, operative or instrumental delivery, placenta praevia, abortions, cervix injuries, and eclampsia have been observed (Abenhaim et al. 2008; Turner et al. 2002).

Although the risk for amniotic fluid embolism is highest during delivery, cases in the second trimester have been published (Clark et al. 1995). A British analysis showed that in 70% of cases, amniotic fluid embolism develops during delivery. The majority occurs within the first five minutes after the second stage of labor, while 19% occur during a cesarean section (Tuffnell and Johnson 2000). Maternal mortality is stated to be 60-80% in reviews and collective statistics. Perinatal mortality is <80% with a high morbidity but depends on when the disease becomes manifest. Only 15% of all survivors have no chronic neurological complications (Clark et al. 1995).

Pathophysiology

As amniotic fluid embolism is more a combination of an anaphylactic and a septic shock than an embolic process, the term "anaphylactoid syndrome of pregnancy" has been proposed to replace the term amniotic fluid embolism. Due to the rarity of amniotic fluid embolism, the knowledge of its pathophysiology relies mainly on experiments with animal models. The disease seems to proceed in two phases (Clark 1990; Reis et al. 1969):

- Phase 1: Vasospasm of the pulmonary artery causes pulmonary hypertension with right ventricular pressure increase. The resulting hypoxia causes myocardial and pulmonary capillary damage which can lead to left heart failure and acute respiratory distress syndrome. Only later does lung edema develop, either in the context of left heart failure or capillary leak syndrome.
- Phase 2: If the patient survives phase 1, the hemorrhagic phase 2 with uterine atony and disseminated intravascular coagulation begins. Consumption coagulopathy is sometimes referred to as the first symptom.

Clinical symptoms

The main symptoms are dyspnea with respiratory arrest, acute hypotension and coagulopathy, followed by tonic-clonic seizures and cardiac arrest. Early symptoms are initial restlessness as well as confusion combined with dyspnea and a decrease in oxygen saturation (Clark et al. 1995). After severe bleeding with consumption coagulopathy, the symptoms can become fatal within an hour.

Diagnostics

The diagnosis of amniotic fluid embolism is only clinical and is an exclusion procedure, as, until today, no secure diagnostic measures exist. Also the pathological detection of fetal components in the pulmonary vessels, which can only be observed in an autopsy, can merely support the diagnosis, but not prove it (Tuffnell and Johnson 2000). The most important differential diagnoses are peripartal bleeding, severe preeclampsia, anaphylactic shock and pulmonary embolism.

Therapy

The prevention of amniotic fluid embolism is not possible. The clinical picture requires immediate intensive care measures with immediate intubation in order to provide adequate oxygenation with ventilation and hemodynamic stabilization by transfusion or cardiopulmonary reanimation. Disseminated intravascular coagulation has, in certain cases, successfully been treated by the administration of recombinant factor VIIa (rFVIIa, NovoSeven) (Prosper et al. 2007). However, a specific pharmacological therapy is yet unknown (Tuffnell and Johnson 2000).

From a forensic point of view, when suspecting amniotic fluid embolism, therapy should immediately be initiated with the consultation of an anesthesiologist or intensivists and documented. In addition, immediate transfer to a center with maximum care should be initiated. After unsuccessful reanimation of the pregnant patient, delivery has to be initiated immediately.

CME PraktFortbildGynakolGeburtsmedGynakolEndokrinol 2009; 5(3): 194–204

Keywords
Amniotic fluid embolism, venous thromboembolism, eclampsia, maternal mortality

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Conflict of interest
The author declares that there is no conflict of interest as defined by the guidelines of the International Committee of Medical Journal Editors (ICMJE; www.icmje.org).

Manuscript information
Submitted on: 22.04.2009
Accepted on: 30.07.2009

CME

Amniotic fluid embolism, venous thromboembolism, and eclampsia

Question 1
Amniotic fluid embolism is characterized by the following aspect:
a. It can easily be diagnosed with typical laboratory parameters.
b. Fetal components in the pulmonary vessels can always be detected in an autopsy.
c. The first symptom may be coagulopathy.
d. Amniotic fluid embolism is rather comparable to an embolic than to an anaphylactic process.
e. In most cases, amniotic fluid embolism is not a life-threatening complication.

Question 2
Which of the following is correct concerning amniotic fluid embolism?
a. Dyspnea is a rare phenomenon.
b. The patients usually stay calm and indifferent.
c. The concomitant hypoxia is, in particular, caused by pulmonary embolism.
d. Among others, important differential diagnoses are pre-eclampsia and pulmonary embolism.
e. Especially the primipara is endangered to develop amniotic fluid embolism.

Question 3
The following statement concerning deep venous thrombosis of the leg is correct:
a. Deep venous thrombosis of the leg affects the left leg in up to 90% of women.
b. Deep venous thrombosis of the leg is more frequent in developing countries than in industrial countries.
c. Deep venous thrombosis of the leg especially occurs within the first trimester.
d. Deep venous thrombosis of the leg is twofold more often in pregnant women compared to nonpregnant women.
e. Deep venous thrombosis of the leg always occurs with the typical symptoms such as tension feeling and cyanosis.

Question 4
The following statement on pulmonary embolism is incorrect:
a. Pulmonary embolism is one of the main reasons for maternal mortality.
b. 40% to 60% of all pulmonary embolisms occur during puerperium.
c. Immobilization and adiposity are two main risk factors.
d. Pulmonary embolism during pregnancy requires fibrinolytic therapy.
e. By heparin therapy, mortality can be reduced to below 1%.

Question 5
The following statement concerning therapy of thromboembolism is correct:
a. Unfractionated heparin crosses the placenta.
b. Low-molecular-weight heparin has a longer half-life.
c. Low molecular-weight heparin is preferentially used peripartally.
d. Fibrinolytic therapy during pregnancy is contraindicated.
e. During puerperium, the dosage of heparins should be halved.

Question 6
The following statement concerning eclampsia is incorrect:
a. The diagnosis is based on clinical symptoms.
b. Prodromal symptoms may be missing.
c. An important differential diagnosis is intracerebral bleeding.
d. Focal seizures are feared.
e. Intrauterine death may occur during an eclamptic seizure.

Question 7
The following statement on eclampsia is correct:
a. The incidence in industrial countries has increased over the last decades.
b. In industrial countries, the incidence is 3-5 in 10000 births.
c. The incidence in developing countries is similar to the incidence in Europe.
d. Eclampsia always occurs as a consequence of severe hypertension.
e. It is always accompanied by pronounced proteinuria.

Question 8
The frequency of eclampsia in puerperium is:
a. 0 %
b. 1-2%
c. 5-17%
d. 50%
e. > 70%

Question 9
For the therapy of eclampsia, the following statement is incorrect:
a. Antihypertensive acute therapy is recommended in diastolic blood pressures of 105 to 110 mmHg.
b. Magnesium is the first choice medication in the therapy and prevention of eclampsia.
c. Magnesiumsulphate is contraindicated in normotensive patients due to its blood pressure reducing effects.
d. A maintenance dose of 1-2 mg magnesium sulphate i.v. per hour is sufficient.
e. Antihypertensive acute therapy is recommended if the systolic blood pressure is above 160 mmHg.

Question 10
A 36 year-old woman is pregnant with her second child (GA 39+5) and suffering from sudden cardiac arrest at a cervical dilation of 8 cm. Which of the following diseases is most likely?
a. An epileptic seizure,
b. the cava syndrome,
c. tetanus of pulmonary origin,
d. amniotic fluid embolism,
e. eclampsia.