# A review on the management and outcome of Preeclampsia in Kasr-el-Ainy Hospital

Thesis

Submitted for fulfillment of

Master Degree in Obstetric and Gynecology

By


## Robert Emile Daniel

M.B.B.Ch


Under the supervision of


## Prof. Dr Ayman Abdel Halim Marzouk

Professor of Obstetric and Gynecology

Faculty of Medicine

Cairo University

## Dr Yahia M. Elfaissal Elrefaey

Lecturer of obstetrics and gynecology

Faculty of Medecine

Cairo University


2009

# مراجعة لأساليب علاج ونتائج تسمم الحمل بمستشفى قصر العينى

رسالة
مقدمة توطئة للحصول على درجة الماجستير فى التوليد و أمراض النساء
من


## الطبيب/ روبير اميل دانيال


تحت اشراف


الأستاذ الدكتور
## أيمن عبدالحليم مرزوق
استاذ التوليد و أمراض النساء
جامعة القاهرة

دكتور
## يحيى محمد الفيصل الرفاعى
مدرس التوليد و أمراض النساء
جامعة القاهرة



جامعة القاهرة
٢٠٠٩

# A review on the management and outcome of Preeclampsia in Kasr-el-Ainy Hospital

Thesis

Submitted for fulfillment of

Master Degree in Obstetric and Gynecology

By


## Robert Emile Daniel

M.B.B.Ch


Under the supervision of


## Prof. Dr Ayman Abdel Halim Marzouk

Professor of Obstetric and Gynecology

Faculty of Medicine

Cairo University

## Dr Yahia M. Elfaissal Elrefaey

Lecturer of obstetrics and gynecology

Faculty of Medecine

Cairo University


2009

# مراجعة لأساليب علاج ونتائج تسمم الحمل بمستشفى قصر العينى

رسالة
مقدمة توطئة للحصول على درجة الماجستير فى التوليد و أمراض النساء
من


## الطبيب/ روبير اميل دانيال


تحت اشراف


الأستاذ الدكتور
## أيمن عبدالحليم مرزوق
استاذ التوليد و أمراض النساء
جامعة القاهرة

دكتور
## يحيى محمد الفيصل الرفاعى
مدرس التوليد و أمراض النساء
جامعة القاهرة



جامعة القاهرة
٢٠٠٩

# Acknowledgement

***I am greatly indebted to Prof. Dr. Ayman Abdel Halim Marzouk, Professor of Obstetrics and Gynecology, Faculty of Medicine, Cairo University, who suggested the subject and supervised the work all through its stages. To him I express my deepest gratitude.***

***I am deeply grateful to Dr. Yahia Elfaissal Elrefaey, Lecturer of obstetrics and gynecology, Faculty of Medicine, Cairo University, who supervised and revised the whole work, for his continuous encouragement, guidance, cooperation and great support.***

***Finally, my great appreciation is given to my family and to all those who shared either practically or morally in my creation of this thesis.***

# Abstract

Preeclampsia and Eclampsia are serious syndromes that can affect human pregnancy causing serious damage. Preeclampsia described as pregnancy-specific syndrome of reduced organ perfusion secondary to vasospasm and endothelial activation. Eclampsia is described as the onset of convulsions in a woman with preeclampsia that cannot be attributed to other causes. Among the etiologies of preeclampsia the most convincing theory is the effect of the chorionic villi on the pregnant women. the incidence of preeclampsia was 6.72 %, The incidence of maternal mortality was 0.2%, The incidence of abdominal delivery was 66.8%, Regarding the main therapeutic profile magnesium sulphate was administered to 90% of patients.

**Key words:** pregnancy, hypertension, convulsions, magnesium sulfate.

# List of tables

# INTRODUCTION

Hypertensive disorders complicating pregnancy are common and form one of the deadly triad, along with hemorrhage and infection, which contribute greatly to maternal morbidity and mortality. Kuklina et al performed a cross-sectional study using the 1998-2006 Nationwide Inpatient Sample of the Healthcare Cost and Utilization Project; they found the overall prevalence of hypertensive disorders among delivery hospitalizations increased significantly from 67.2 per 1,000 deliveries in 1998 to 81.4 per 1,000 deliveries in 2006. Compared with hospitalizations without any hypertensive disorders, the risk of severe obstetric complications ranged from 3.3 to 34.8 for hospitalizations with eclampsia/severe preeclampsia and from 1.4 to 2.2 for gestational hypertension. The prevalence of hospitalizations with eclampsia/severe preeclampsia increased moderately from 9.4 to 12.4 per 1,000 deliveries ($P$ for linear trend <0.001) during the period of study. However, these hospitalizations were associated with 38% of hospitalizations with acute renal failure and 19% or more of hospitalizations with ventilation, disseminated intravascular coagulation syndrome, pulmonary edema, puerperal cerebrovascular disorders, and respiratory distress syndrome. Overall, hospitalizations with hypertensive disorders were associated with 57% of hospitalizations with acute renal failure, 27% of hospitalizations with disseminated intravascular coagulation syndrome, and 30% or more of hospitalizations with ventilation, pulmonary edema, puerperal cerebrovascular disorders, and respiratory distress syndrome.

How pregnancy incites or aggravates hypertension remains unsolved despite decades of intensive research. Indeed, hypertensive disorders remain among the most significant and intriguing unsolved problems in obstetrics. To elucidate these, ongoing research is sponsored by the National Institutes of Child Health and Human Development (NICHD) and its Maternal-Fetal Medicine Units Network. Another important stimulus for research is the International Society for the Study of Hypertension in Pregnancy. The National Heart, Lung, and Blood Institute promote research and coordination through the National High Blood Pressure Education Program (NHBPEP) and its Working Group for High Blood Pressure in Pregnancy.

# TERMINOLOGY AND CLASSIFICATION

The term gestational hypertension is used now to describe any form of new-onset pregnancy-related hypertension. It was adopted by the Working Group of the NHBPEP (2000), which proposed a classification system based on clinical simplicity to guide management. The term was chosen to emphasize the cause-and-effect connection between pregnancy and its unique form of hypertension—preeclampsia and eclampsia. It is also meant to be a working term that is purposefully vague, but it should convey that the development of hypertension in a previously normotensive pregnant woman should and must be considered potentially dangerous to both herself and her fetus.

The classification of hypertensive disorders complicating pregnancy by the Working Group of the NHBPEP (2000) is shown in Table 1. There are five types of hypertensive disease:

1. Gestational hypertension (formerly pregnancy-induced hypertension that included transient hypertension).
2. Preeclampsia.
3. Eclampsia.
4. Preeclampsia superimposed on chronic hypertension.
5. Chronic hypertension.

An important consideration in this classification is differentiating hypertensive disorders that precede pregnancy from preeclampsia and eclampsia, which are potentially more ominous.

TABLE 1 Diagnosis of Hypertensive Disorders Complicating Pregnancy

| |
|---|
| **Gestational hypertension** |
| BP >, 140/90 mm Hg for first time during pregnancy |
| No proteinuria |
| BP returns to normal < 12 weeks' postpartum |
| Final diagnosis made only postpartum |
| May have other signs or symptoms of preeclampsia, for example, epigastric discomfort or thrombocytopenia |
| **Preeclampsia** |
| *Minimum criteria* |
| BP >or = 140/90 mm Hg after 20 weeks' gestation |
| Proteinuria >or= 300 mg/24 hours or >or= 1 + dipstick |
| *Increased certainty of preeclampsia* |
| BPs:160/110me»»»Hg |
| Proteinuria 2.0 g/24 hours or >. 2+ dipstick |
| Serum creatinine > 1.2 mg/dL unless known to be previously elevated |
| Platelets < 100,000/mm3 |
| Microangiopathic hemolysis (increased LDH) |
| Elevated ALT or AST |
| Persistent headache or other cerebral or visual disturbance |
| Persistent epigastric pain |
| **Eclampsia** |
| Seizures that cannot be attributed to other causes in a woman with preeclampsia. |
| **Superimposed Preeclampsia (on chronic hypertension)** |
| New-onset proteinuria >. 300 mg/24 hours in hypertensive women but no proteinuria before 20 weeks' gestation. |
| A sudden increase in proteinuria or blood pressure or platelet count < 100,000/mm3 in women with hypertension and proteinuria before 20 weeks' gestation |
| **Chronic Hypertension** |
| BP >: 140/90 mm Hg before pregnancy or diagnosed before 20 weeks' gestation not attributable to gestational trophoblastic disease |
| or |
| Hypertension first diagnosed after 20 weeks' gestation and persistent after 12 weeks' postpartum |

## DIAGNOSIS

Hypertension is diagnosed when the resting blood pressure is 140/90 mm Hg or greater; Korotkoff phase V is used to define diastolic pressure. In the past, it had been recommended that an incremental increase of 30 mm

Hg systolic or 15 mm Hg diastolic pressure be used as diagnostic criteria, even when absolute values were below 140/90 mm Hg. These criteria are no longer recommended because evidence shows that these women are not likely to suffer increased adverse pregnancy outcomes (Levine and co-workers, 2000; North and colleagues, 1999) that said women who have arise of 30 mm Hg systolic or 15 mm Hg diastolic warrant close observation. Edema has been abandoned a diagnostic criterion because it occurs in too many normal pregnant women to be discriminant.

## GESTATIONAL HYPERTENSION

As shown in Table 1, the diagnosis of gestational hypertension is made in women whose blood pressure reaches 140/90 mm Hg or greater for the first time during pregnancy but in whom proteinuria is not identified. Gestational hypertension is also called transient hypertension if preeclampsia does not develop and the blood pressure has returned to normal by 12 weeks' postpartum. In this classification, the final diagnosis that the woman does not have gestational hypertension is not made until several weeks after delivery. Thus, gestational hypertension is a diagnosis of exclusion. Importantly, some women with gestational hypertension may later develop other findings of preeclampsia, for example, symptoms such as headaches or epigastric pain, proteinuria, or thrombocytopenia, all of which influence management.

When blood pressure rises appreciably during the latter half of pregnancy, it is dangerous, especially to the fetus, not to act simply because proteinuria has not yet developed. As Chesley (1985) emphasized, 10 percent of eclamptic seizures develop before overt proteinuria is identified, thus, it is clear that when blood pressure begins to rise, both mother and fetus are at increased risk. Proteinuria is a sign of worsening hypertensive disease, specifically preeclampsia. Overt and persistent proteinuria further increases maternal and fetal risks.

## PREECLAMPSIA

This condition is best described as a pregnancy-specific syndrome of reduced organ perfusion secondary to vasospasm and endothelial activation. Proteinuria is an important sign of preeclampsia, and Chesley (1985) rightfully concluded that the diagnosis is questionable in its absence. Significant proteinuria is defined by 24-hour urinary protein exceeding 300 mg per 24 hours, or persistent 30mg/dL (1+ dipstick) in random urine samples. The degree of proteinuria may fluctuate widely over any 24-hour period, even in severe cases. Therefore, a single random sample may fail to

demonstrate significant proteinuria. In their extensive study of renal biopsy specimens obtained from hypertensive pregnant women, McCartney and co-workers (1971) invariably found proteinuria when the glomerular lesion considered to be characteristic of preeclampsia was evident. Importantly, both proteinuria and alterations of glomerular histology develop late in the course. It is apparent that preeclampsia becomes evident clinically only near the end of a covert pathophysiological process that may begin as early as implantation.

Thus, the minimum criteria for the diagnosis of preeclampsia are hypertension plus minimal proteinuria. The more severe the hypertension or proteinuria, the more certain is the diagnosis of preeclampsia. Similarly, abnormal laboratory findings in tests of renal, hepatic, and hematological function increase the certainty of preeclampsia. In addition, persistent premonitory symptoms of eclampsia, such as headache and epigastric pain, also increase the certainty.

The combination of proteinuria and hypertension during pregnancy markedly increases the risk of perinatal mortality and morbidity (Ferrazzani and associates, 1990). A widely quoted study by Friedman and Neff (1976) of more than 38,000 pregnancies was completed over three decades ago. It showed that diastolic hypertension of 95 mm Hg or greater was associated with a three fold increase in the fetal death rate. Worsening hypertension, especially if accompanied by proteinuria, was more ominous, but proteinuria without hypertension was rather benign. In a recent study, however, Newman and co-workers (2003) reported that worsening proteinuria resulted in increasing preterm delivery, but that neonatal survival was not significantly altered. In contrast, following their analysis of more than 9000 nulliparous women ascertained from the Collaborative Perinatal Project, a large cohort study conducted between 1959 and 1965, Zhang and co-workers (2001) concluded that neither blood pressure severity nor proteinuria were sensitive predictors of adverse outcome.

*Epigastric or right upper quadrant pain* is thought to result from hepatocellular necrosis, ischemia, and edema that `stretch the Glisson capsule. This characteristic pain is frequently accompanied by elevated serum hepatic transaminase levels and usually is a sign to terminate the pregnancy. The pain presages hepatic infarction and hemorrhage or catastrophic rupture of a subcapsular hematoma. Fortunately, hepatic rupture is rare.

*Thrombocytopenia* is characteristic of worsening preeclampsia, and it probably is caused by platelet activation and aggregation as well as microangiopathic hemolysis induced by severe vasospasm. Evidence of gross hemolysis such as hemoglobinemia, hemoglobinuria, or hyperbilirubinemia is indicative of severe disease.

TABLE 2 Indications of Severity of Hypertensive Disorders during Pregnancy

| **Abnormality** | **Mild** | **Severe** |
|---|---|---|
| Diastolic blood pressure | <100 mmHg | 110 mmHg or higher |
| Proteinuria | Trace to 1+ | Persistent 2+ |
| Headache | Absent | Present |
| Visual disturbances | Absent | Present |
| Upper abdominal pain | Absent | Present |
| Oliguria | Absent | Present |
| Convulsion (eclampsia) | Absent | Present |
| Serum creatinine | Normal | Elevated |
| Thrombocytopenia | Absent | Present |
| Liver enzyme elevation | Minimal | Marked |
| Fetal growth restriction | Absent | Obvious |
| Pulmonary edema | Absent | Present |

### *Severity of Preeclampsia*

The severity of preeclampsia is assessed by the frequency and intensity of the abnormalities listed in Table 2. The more profound this aberration, the more likely is the need for pregnancy termination. The differentiation between mild and severe preeclampsia can be misleading because apparently mild disease may progress rapidly to severe disease. Although hypertension is a requisite to diagnosing preeclampsia, absolute blood pressure alone is not always a dependable indicator of its severity. For example, young adolescent women may have 34- proteinuria and convulsions with a blood pressure of 135/85 mm Hg, whereas most women with blood pressures as high as 180/120 mm Hg do not have seizures. A rapid increase in blood pressure followed by convulsions is usually preceded

by an unrelenting severe headache or visual disturbances. For this reason, these symptoms are considered ominous.

## ECLAMPSIA

The onset of convulsions in a woman with preeclampsia that cannot be attributed to other causes is termed eclampsia. The seizures are generalized and may appear before, during, or after labor. In older studies, in about 10 percent of eclamptic women, especially nulliparas, seizures did not develop until after 48 hours postpartum (Brown and colleagues, 1987; Lubarsky and associates, 1994). As prenatal care improved, many antepartum and intrapartum cases are now prevented, and a more recent study, reported that a fourth of eclamptic seizures developed beyond 48 hours postpartum (Chamesand co-workers, 2002).

## PREECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION

All chronic hypertensive disorders, regardless of their cause, predispose to development of superimposed preeclampsia and eclampsia. These disorders can create difficult problems with diagnosis and management in women who are not seen until after midpregnancy. The diagnosis of chronic underlying hypertension is made when:

1. Hypertension (140/90 mm Hg or greater) is documented antecedent to pregnancy.

2. Hypertension (140/90 mm Hg or greater) is detected before 20 weeks, unless there is gestational trophoblastic disease.

3. Hypertension persists long after delivery (see Table 1).

Additional historical factors that help support the diagnosis are multiparity and hypertension complicating a previous pregnancy other than the first. There frequently is also a family history of essential hypertension. The diagnosis of chronic hypertension may be difficult to make if the woman is not seen until the latter half of pregnancy, because blood pressure decreases during the second and early third trimesters in both normotensive and chronically hypertensive women. Thus a woman with chronic vascular disease, who is seen for the first time at 20 weeks, frequently has blood pressure within the normal accepted range. During the third trimester, however, if blood pressure returns to its former hypertensive level, it

presents a diagnostic problem as to whether the hypertension is chronic or induced by pregnancy. In these situations, a search for evidence of end-organ damage from chronic hypertension may help elucidate the underlying cause of hypertension. Examples include left ventricular hypertrophy or retinal changes such as arteriolar narrowing, exudates, or cotton-wool spots.

Some of the many causes of underlying hypertension that are encountered during pregnancy are listed in Table 3.

TABLE 3 Underlying Causes of Chronic Hypertensive Disorders

| |
|---|
| — Essential familial hypertension (hypertensive vascular disease) |
| — Obesity |
| — Arterial abnormalities<br>Renovascular hypertension<br>Coarctation of the aorta |
| — Endocrine disorders<br>Diabetes mellitus<br>Gushing syndrome<br>Primary aldosteronism<br>Pheochromocytoma<br>Thyrotoxicosis |
| — Glomerulonephritis (acute and chronic) |
| — Renoprival hypertension<br>Chronic glomerulonephritis<br>Chronic renal insufficiency<br>Diabetic nephropathy |
| — Connective tissue diseases<br>Lupus erythematosus<br>Systemic sclerosis<br>Periarteritis nodosa |
| — Polycystic kidney disease |
| — Acute renal failure |