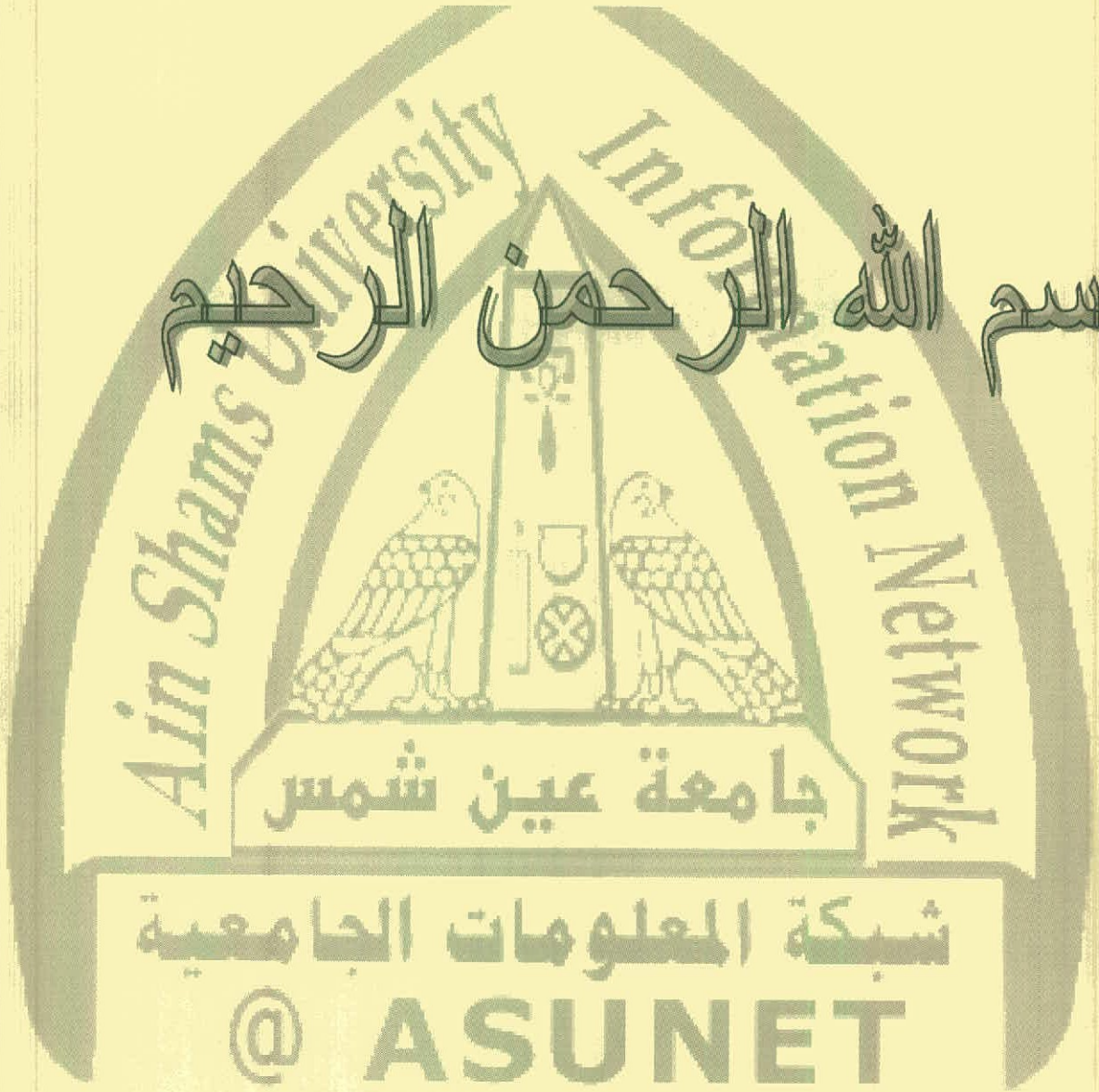
شبكة المعلومات الجامعية



بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
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بعض الوثائـــــــق الاصليـــــة تالفـــة

بالرسالة صفحات لم ترد بالاصل

Evaluation of the use of transdermal Nitroglycerin on the clinical parameters and the fetoplacental circulation in Primigravidae with mild pre-eclampsia.

Thesis

Submitted to the faculty of medicine

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INTRODUCTION

Hypertensive Disorders In Pregnancy

Pregnancy can induce hypertension in normotensive women, or aggravate already existing hypertension.[1] Compared with normotensive gravidas, patients with elevated blood pressure have significantly greater maternal and foetal morbidity and mortality.[2]

Terminology and classification:

More than one classification has been used to classify the different hypertensive disorders of pregnancy.[3] According to American College of Obstetricians and Gynecologists (ACOG, 1996), hypertensive disorders in pregnancy are classified into:[4]

1. Pregnancy induced hypertension (PIH):

Hypertension that develops as a consequence of pregnancy and regresses post-partum.

a) Gestational hypertension: Hypertension without proteinuria or pathological edema.

b) Pre-eclampsia: With proteinuria with or without pathological edema:

   i) Mild. ii) Severe.

c) Eclampsia with proteinuria with or without pathological edema along with convulsions.

2. Pregnancy aggravated hypertension (PAH):

Underlying hypertension worsened by pregnancy.

a) Superimposed pre-eclampsia.

b) Superimposed eclampsia.

3. Coincidental hypertension:

Chronic underlying cause that antecedes pregnancy or persists post-partum.

4. Transient hypertension:

Hypertension which develops after the mid trimester of pregnancy and is characterized by mild elevations of blood pressure that do not compromise pregnancy. It regresses after delivery but may return in subsequent gestations.

The committee on terminology of the American College of Obstetricians & Gynecologists suggested the following definitions [5]:

Hypertension:

Is defined as diastolic blood pressure of at least 90 mm Hg, or a systolic pressure of at least 140 mm Hg, or a rise in the former of at least 15 mm Hg, or the latter of at least 30 mm Hg. The blood pressure must be measured on at least two occasions, 6 hours or more apart, in both arms.[5]

Proteinuria:

Is defined as the presence of 300 mg or more of protein in urine, in a 24 hours urine collection, or a protein concentration of 1 gram / Liter or more in at least two random urine specimens collected 6 hours apart. (5)

Pre-eclampsia:

Is defined as hypertension associated with proteinuria greater than 0.3 g/L in a random sample; generalized edema, greater than +1 pitting edema after 12 hours of rest in bed, or a weight gain of 5 Libra or more in 1 week, or both after 20 weeks of gestation.(2)

Eclampsia:

Is the occurrence of convulsions, not caused by any coincidental neurological diseases (such as epilepsy) in a woman whose condition fulfills the criteria of pre-eclampsia. (5)

Superimposed pre-eclampsia or eclampsia:

Is the development of pre-eclampsia or eclampsia in a woman with chronic hypertensive vascular or renal disease. (5)

Chronic hypertension (coincidental):

Is the presence of hypertension (140/90 mm Hg or greater) antecedent to pregnancy, before 20 weeks of gestation (in absence of gestational trophoblastic diseases), or persistent beyond 6 weeks post-partum. (5)

Transient hypertension (Gestational hypertension):

The development of hypertension only during pregnancy or early peurpeurium in a previously normotensive woman, whose blood pressure normalizes within 10 days post-partum. [2]

Gestational edema:

Is the generalized accumulation of fluid of greater than +1 pitting edema after 12 hours of bed rest, or a weight gain of 5 pounds or more in a week. [4] The edema should be generalized and not just dependent.

Gestational proteinuria:

Is proteinuria 300 mg or more of urinary protein/24 hours or 100 mg/dl in 2 random urine samples at least 24 hours apart during pregnancy in the absence of hypertension, edema, renal infection, or known reno-vascular disease. The existence of such an entity is questionable. [5]

Incidence & predisposing factors of pregnancy-induced hypertension:

The incidence of pre-eclampsia is commonly cited to be about 5 %, although remarkable variations are reported. In Alexandria, the incidence was found to be 2%, as calculated from Shatby Maternity Hospital. [6]

Age has an important influence on the incidence of hypertensive disorders in pregnancy. Young Primigravidae and both Primigravidae and multigravidas over the age of 30 years are at higher risk. [7]

Familial and genetic factors seem to have strong influence on the occurrence of hypertension in pregnancy. [8] Multiple pregnancy is also associated with greater incidence of hypertension and proteinuria. This may be due to hyper-placentosis, increased placental hormone secretion, increased blood volume, cardiac output and other haemodynamic changes. [7]

The incidence of pre-eclampsia is higher in pregnancies with foetal hydrops (up to 50%) whether due to Rh-isoimmunization or otherwise. [10]

The incidence is also higher in cases of polyhydramnios with foetal malformation, diabetes mellitus,[10] Chronic hypertension, [10] and chronic renal disease. [10] Obesity, smoking and racial background are of importance.

Severity of PIH [1]***:***

The severity of PIH is assessed by the frequency and intensity of the abnormalities listed below:

Abnormality	*Mild*	*Severe*
• Diastolic BP	• ≥ 90 mm Hg and <110 mm Hg	• ≥ 110 mm Hg
• Proteinuria	• ≥ 0.3 g/L	• ≥ 2 g/L
• Headache	• Absent	• Present
• Visual disturbances	• Absent	• Present
• Epigastric and right Hypochondrial pain	• Absent	• Present
• Oliguria (≤ 25 ml/hr)	• Absent	• Present
• Serum creatinine	• Normal	• Elevated
• Thrombocytopenia	• Absent	• Present
• Hyperbilirubinaemia	• Absent	• Present
• Liver enzymes Elevation	• Minimal	• Marked
• Foetal growth retardation	• Absent	• Obvious
• Pulmonary edema	• Absent	• Present
• Serum uric acid	• Normal	• Elevated

Aetiology of pre-eclampsia:

The exact cause of pre-eclampsia is not yet known. A very long list of theories can be given to explain the aetiology of this disease.

Walker (1998) proposed the multimodular approach to the pathophysiology of pre-eclampsia, summarized in three main entities:[9]

1- Abnormalities of placentation, pre-eclampsia is associated with excessive placentation, as in twin and molar pregnancies. The stimulus would be produced either from the ischaemic, small, poorly implanted placenta of classic pre-eclampsia or from a large placenta found in a multiple pregnancy.

2- Abnormalities of platelet / vessel wall interaction, leading to increased vascular sensitivity and platelet consumption. The vascular activity also appears to antedate the clinical signs of pre-eclampsia. These changes may be mediated through a dysfunction of the vascular endothelial cell, which is part of a generalized cellular dysfunction. The degree of dysfunction and which structures are affected varies between women.

3- Maternal response to these changes which will modify the way that the disease is manifest, the severity of the signs and symptoms and the outcome for mother and child.