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RESEARCH ARTICLE

MATERNAL CENTRAL HEAMODYNAMICS IN PRE-ECLAMPSIA, OBSERVATIONAL RETROSPECTIVE AND ANALYTIC STUDY

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ABSTRACT

Objective: The objective of the study is to document maternal central hemodynamic during clinical phase of pre-eclampsia.

Material and methods: This was retrospective observational, comparative study. The study and control groups consist of 40 cases. It was carried out in Al-Hakeem general hospital in Al –Najaf province for the period from April 2016 to April 2017. This was conducted in the department of obstetrics and gynecology in Al –Hakeem maternity department in Al-Najaf. The study group was collected in delivery room. The first group was women who had an elevated blood pressure at least 145/90 mmHg first detected after mid pregnancy with marked proteinurea at least (2+), and the second group was normotensive women with no protein in their urine.

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INTRODUCTION

Hypertensive disorders of pregnancy remain a major cause of morbidity and mortality worldwide. A Pregnant women with hypertension ,either newly diagnosed or pre-existing remain at risk for severe complication such as abruptio placentae, cerebrovascular accident ,end organ failure and disseminated intravascular coagulation with the fetus at risk for intrauterine growth retardation, prematurity and intrauterine death."4"

Preeclampsia: hypertension of at least 140/90 recorded on two separate occasions at least4Hrs apart and in presence of at least 300mg protein in a24 HR collection of urine arising de novo after the 20th week of gestation (sometimes earlier in pregnancy as in hydatiform mole) in a previously normotensive woman and resolving completely by sixth postpartum week."1"

Chronic Hypertention: With or without renal disease, existing prior to pregnancy is of a different etiology from preeclampsia although it can predispose to the later development of superimposed preeclampsia.

Gestational Hypertention: Hypertension arising for the first time in the second half of pregnancy in absence of protein urea.

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It should be clearly distinguished from preeclampsia ……

Eclampsia: In a serious and life threatening complication of preeclampsia. It is defined as convulsions occurring in a woman with established preeclampsia in absence of any other neurological or metabolic cause. It is an obstetric emergency."5" Terms used to describe hypertension in pregnancy are numerous such as preeclampsia, gestational hypertension, toxemia of pregnancy, proteinuric hypertension, preeclamptic toxemia, pregnancy induced hypertension, organic hypertension , true preeclampsia, latent essential hypertension , chronic hypertension and transient hypertension in pregnancy have been used Hypertension in pregnancy is common occurring in approximately one in five women after 20 weeks gestation, however only a small minority of these have serious disease associated with morbidity. Obstetric day units help reduce need for inpatient management, as the onset and cause of disease is unpredictable, frequent assessment of women with hypertension remain the mainstay of safe clinical practice. As delivery is the only cure for preeclampsia ,it is the commonest cause of iatrogenic prematurity accounting for 15% of all premature births and approximately one in five very low birth weight infant <1500 gm. Preeclampsia complicate 3%of pregnancies, but the incidence varies depending on exact definition used as the population studied. Eclampsia is rare in UK occurring 1 in 2000 pregnancies. *"2"*

Classificaion of Hypertensive Disorders in Pregnancy:"4"

A-Preexisting Hypertension

Hypertension that predates pregnancy or it diagnosed before 20 weeks gestation In most cases hypertension persist more than 42 days postpartum. It may associated with protein urea.

- Essential :- primary
- Seconary:- secondary to such conditions such as renal diseases, pheochromacytoma, and Cushing syndrome.

B-Gestational Hypertension

Hypertension develop after 20 weeks gestation in most cases it resolves in less than 42 days postpartum.

- 1-without protein urea: corresponds to previous terminology pregnancy induced hypertension , transient hypertension and non proteinuric hypertension, protein collection in 24hrs urine less than 0.3g/d
- 2-without adverse condition
- 2-with adverse conditions convulsions eclampsia very high diastolic pressure>110mmhg, thrombocytopenia(low platelets count less than 100,000x10 ^{9}/l) ,oliguria less than 500ml/day, pulmonary edema, elevated liver enzymes ,shortness of breath, chest pain,, HELLP syndrome , oligohydrominia.
- 2-with protein urea;- corresponds to term preeclampsia, preeclamptic toxemia protein excretion in 24 hr urine collection more than 0.3g/d

a-without adverse condition.
b-with adverse conditions: protein excretion in24 hr collection especially with hypo-albuminemia (albumin level less than 8g/l)

C-Preexisting Hypertension and superimposed gestatoinal hypertension with proteinurea:

Defined as further worsening of blood pressure. & protein excretions more than 3g/dl in 24hr urine collection after 20 week gestation corresponds to chronic hypertension with superimposed preeclampsia

D-unclassifable antinatally:

Hypertension with or without systemic manifestations of blood pressure. was first recorded after 20 weeks gestation reassessment is necessary at or after 42 day postpartum. If the ht has resolved by then the condition should be reclassified as gestational hypertension with or without proteinurea, if the hypertension has not resolved then, the condition should reclassified as chronic hypertension.

Aetiology:"6"

The preeclampsia syndrome is thought in some cases to be caused by shallowly implanted placenta which become hypoxic leading to an immune reaction characterized by secretion of up regulated inflammatory mediators from placenta, acting on vascular endothelium. The shallow implantation is thought to stem from maternal immune systems response to the placenta. this theory emphasizes the role of maternal immune system & refers to evidence suggesting a lack of established immunological tolerance to paternal antigen from fetus &its placenta. In some cases of preeclampsia , it is thought that the mother lacks the receptors for the proteins the placenta is using to down regulate the maternal immune system response to it. In many cases of preeclampsia ,the maternal response to the placenta appears to have allowed for normal implantation .it is possible that woman with higher baseline levels inflammation stemming from underlying conditions such as chronic hypertension or autoimmune disease may have less tolerance for inflammatory burden of pregnancy. So many theories have attempted to explained why preeclampsia arises &have linked the syndrome to presence of following:

- Endothelial cellular injury
- immune rejection of placenta
- compromised placental perfusion
- altered vascular reactivity
- imbalance between prostocyclin & thromboxane
- decreased glomerular filtration rate
- retention of salts and water.
- uterine muscle ischemia
- dietary factors e.g. vitamin A deficiency.

Pathophysiology

The clinical picture of preeclampsia is due to an inactivations 0r dysfunction of vascular endothelial cells. the concentration of cell surface markers for endothelial cell damage including (fibronectin adhesion molecules & vonwillbrand factor) is increased in plasma of women with preeclampsia. Increased central nervous system irritability characterized by peripheral vasodilatation resulting in a fall in peripheral resistance despite an increase in cardiac output& circulating volume. The peripheral vasodilatation is accomplished through as reduced vascular sensitivity to vasoconstrictor such as angiotensin &possibly by enhanced vasodilator mainly vascular endothelial cells. in preeclampsia the insensitivity to vasoconstrictor is lost & vessels in vivo &in vitro show reduced sensitivity to vasodilator &enhanced sensitivity to vasoconstrictors .Vasopressin &endothelial cell dysfunction with subsequent platelets activation & micro aggregate formation account for many of pathological features of preeclampsia seen in almost every major organ system. Loss of endothelial cell integrity result in an increase in vascular permeability contribute to formation of generalized edema which is often found in woman with preeclampsia."1" Dependant edema of feet is very common in healthy pregnant women. rapidly progressing edema of face &hands is suggestive of preeclampsia. In the kidney a highly characteristic lesion called glomeruloendothelliosis is seen. This consist of endothelial & mesengial cell swelling basement membrane inclusion, but little disruption of renal epithelial podocyte, this relatively specific for preeclampsia& is associated with development of proteinurea, reduced renal clearance of uric acid & oligurea, it not seen with hypertension due to other causes. In the liver sub endothelial fibrin deposition is associated with elevated liver enzymes, haemolysis & low platelet count due to platelet consumption& activation of coagulation system presence of these finding is called HELLP syndrome. Vasospasm and cerebral edema have been implicated in cerebral manifestation of preeclampsia & their progression to eclampsia. Retinal

haemorrhage, exadute & papilloedema are found in hypertensive encephalopathy. Within the vasculature of the placental bed, there is acute atherosis of the spiral arteries, with platelet micro aggregate & large thrombus. *"7"*

Diagnostic Evalaution

History"5"

Mild to moderate preeclampsia may be a symptomatic many cases are detected through routine prenatal screening.

Sever preeclampsia display end organ effect

- Central nervous system;
  - Headaache, Visual disturbances "blurred, scintillating scotomata
  - Altered mental status
  - Blindness may be cortical or retinal
- Dyspnea
- Epigastric or upper right quadrant pain
- Weakness or malaise this may evidence hemolytic anemia
- Excessive nausea &vomiting

Preeclampsia is most often seen in first time pregnancy &in pregnant teens &women over 40 other risk factors;

- History of high blood pressure prior to pregnancy
- History of preeclampsia
- Ahistory of preeclampsia in mother or sisters
- Obesity prior to pregnancy
- Carrying more than one fetus (multiple pregnancy)
- History of diabetes mellitus, kidney diseases.

PHYSICAL EXAMINATION:"7"

- increased blood pressure compared with patient baseline or greater than 140/90
- altered mental status
- impaired vision
- paplliedema
- epigastric or right upper quadrant abdominal tenderness
- Hyperrflexia or clonus
- Seizures
- Focal neurological deficit
- Diagnostic criteria for sever preeclampsia include at least One of following;
  - Systolic blood pressure greater than 160mmHg or diastolic blood pressure greater than 110mmHg on two occasions 6 hrs apart with the patient at bed rest.
  - Protein urea greater than 5000mg in 24 hr collection or more than (+++) on two random urine sample collected at least 4 hrs apart.
  - Oliguria with less than 500ml urine per 24hrs.
  - Persistant maternal headache or visual disturbanceas.
  - Pulmonary edema or cyanosis.
  - Concerning abdominal pain.
  - Impaired liver function test findings.
  - Thrombocytopenia
  - Oligohydrominias, decreased fetal growth, or placental abruption.

Labaratory.Studies:"8"

1. Complete blood count
   a. Microangiopathic hemolytic anemia.
   b. Thrombocytopenia.
   c. Hemoglobin concentration may increase in sever preeclampsia.
2. liver function test;transaminase levels are elevated from hepatocellular injury &in HELLP syndrome.
3. Serum creatinine level :levels are elevated due to decrease intravascular volume &decrease glomerular filtration rate (GFR).
4. Urinelysis
   a. Protienurea is one of diagnostic criteria for preeclampsia.
   b. Protienurea is defined as greater than or equal to(+protein)on urine dipstick, alternatively protein concentration of 300mg /l or more on urine dipstick.
   c. Protienurea is also defined as 300mg or more protein in 24hrs urine sample.
5. Elevated PT.PTT. fibrin split products or decreased fibrinogen.
   ●Disseminated intravascular cougulopathy testing.
6. Serum uric acid.
   ●Serial levels may be useful to indicate disease progression.
7. Imaging studies.
   ● Head CT scan:- this study is used to detect intracranial hemorrhage in selected patients with sudden sever headache, focal neurological deficit or seizures with a prolonged post- ictal state.
   ● Ultrasonography this is used for fetal assessment."7"

Risk Factors:"9"

Patient with medical disorders such as diabetes mellitus, vascular &connective tissue diseases has potential to develop hypertension in pregnancy other risk factors include;

1. History of preeclampsia. personal or family history of preeclampsia increase the risk of developing the condition
2. First pregnancy; the risk of developing preeclampsia is highest in first pregnancy or first pregnancy with new partner.
3. Age; it is higher in pregnant women younger than 20yrs or older than 35yrs.
4. Obesity. the risk higher if the patient obese.
5. Multiple pregnancy. It is higher in women carrying twins, triplets or other multiples.
6. Gestational diabetes; women who having gestational diabetes have higher risk for developing preeclampsia as pregnancy progresses.
7. History of other conditions increase the risk such as SLE chronic hypertension, kidney disease& diabetes.
8. Race; the risk is more in black than white women.
9. Pregnancy associated factors; include factors present in this pregnancy &it may increase risk for developing preeclampsia;
   ● Chromosomal abnormalitie of fetuss
   ● Hydatidiform mole
   ● Hydrops fetalis
   ● Multifetal pregnancy
   ● Oocyte donation or donor insemination
   ● Structural congenital anomalies
   ● Urinary tract infection of mother.

Complications of Preeclampsia"13"

1-maternal complications: Most women with preeclampsia deliver healthy babies. , it can lead to slow growth, low birth

weight, preterm birth &stillbirth. with hypertension occurring earlier in pregnancy the greater the risk to patient &her baby .complication of preeclampsia are

Lack of blood flow to the placenta: Preeclampsia affects the arteries carrying blood to the placenta. if placenta does not get enough blood , the baby may receive less.

placental abruption: Preeclampsia increases the risk of placental abruption, in which the placenta separates from inner surface of uterus before delivery. severe abruption may cause heavy bleeding which can be life –threatening for both mother &baby.

HELLP syndrome: Which stand for hemolysis (destruction of RBCS), elevated liver enzymes& low platelet count – syndrome can rapidly become life threatening for both mother &baby. symptoms of HELLP syndrome include nausea &vomiting .headache& upper right abdominal pain. it dangerous because it can occur before signs& symptoms of preeclampsia appear.

Eclampsia: When preeclampsia is not controlled, eclampsia which is preeclampsia plus seizures-can particularly develop .symptoms of eClampsia include upper right abdominal pain, sever headache, vision problems& change in mental status such as decreased alertness. eclampsia can permanently damage another s vital organs liver, kidney& brain. left untreated eclampsia can cause coma, brain damage &death for both mother & baby.

Fetal complication-"9, 10": Fetal outcome is strongly influenced by gestational age &the severity of hypertension as expressed by the need for antihypertensive treatment, irrespective of underlying syndrome. Sever preeclampsia associated with different degrees the fetal injury. The main impact on the fetus is under nutrition as result of utero-placental vascular insufficiency, which lead to growth retardation. there are short and long term effects. the immediate impact observed is altered fetal growth resulting in greater fetal liability for.fetal death as well as it ′'s weight are highly compromised, leading to various degrees of fetal morbidity, fetal damage may be such as to cause fetal death.

Long term follow up studies have demonstrated that babies who suffered intrauterine growth retardation are more likely to develop hypertension, coronary artery disease, &diabetes in adult life. There is growing evidence to suggest that patterns of rarely growth &other life course factors play an important role in the origins &development of cardiovascular, but understanding the processes which mediate these effects is limited Unusual complication involving anoxia or catecholamine release in the mother, fetus or new born may predispose the baby to the development of precocious coronary atherosclerosis later in life. Many fetuses have to adapt to a limited supply of nutrient. In doing so, they permanently change their structure & metabolism. These programmed changes may be the origin of a number of diseases in later life, including coronary heart disease & related disorders; stroke, diabetes & hypertension. "

Hemodynamic Alterations Associated With Pregnancy-Induced Hypertension, Preeclampsia, and Eclampsia

Pregnancy-induced hypertension is hypertension that develops as a consequence of pregnancy and regresses after delivery. Pregnancy-induced hypertension can be differentiated from chronic hypertension, which appears before 20 weeks' gestation or continues for a long period after delivery. Preeclampsia, which is a type of pregnancy-induced hypertension characterized by progressive hypertension and pathological edema, is clinically defined as a blood pressure greater than 140/90 mm Hg after 20 weeks' gestation plus Protienurea (300 mg/24 hours or greater than 1+ protein on a dipstick sample of urine collected at random). Eclampsia is the occurrence of convulsions or coma unrelated to other cerebral conditions with signs and symptoms of preeclampsia."11" Preeclampsia and eclampsia may be complicated by the onset of the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count). Patients with HELLP syndrome are a subset of those with severe pregnancy-induced hypertension who are at increased risk for multiple organ system dysfunction. Maternal complications associated with HELLP include a coagulopathy (specifically, microangiopathic hemolytic anemia) due to liver failure and thrombocytopenia, acute respiratory distress syndrome, and acute renal failure; all of these may with preeclampsia varies depending on the stage of the disease. During the clinical or latent phase of preeclampsia, the homodynamic profile is characterized as hyper dynamic, that is, increased cardiac output with normal vascular resistance. With the onset of preeclampsia, the homodynamic profile varies. In one longitudinal study, women in whom preeclampsia developed crossed over from a high-output state to a high-resistance state, with a dramatic decrease in cardiac output (ie, greater than 3 L/min) and an increase in vascular resistance. However, in other studies, many women had an unchanged profile (high-output or high-resistance states). These differences may be due to variability in treatment among different study populations or to the presence require homodynamic monitoring to guide therapy. The homodynamic profile of a patient of more than a single homodynamic profile for preeclampsia. These findings highlight the importance of individualizing the treatment of patients who have preeclampsia"12"

Aim of the study

To document maternal central hemodynamics during clinical phase of preeclampsia. Patient and method- 40 pregnant women were enrolled in this study, presented in third trimester of pregnancy attending al -Hakeem general hospital in AL-Najaf starting from April 2016 to April 2017. These women who participated in the study were divided into two groups:-

GROUP 1: (20) Women had normal blood pressure and no evidence of protein in their urine.

GROUP 2: (20) women had elevated blood pressure (more than 145/90mmHg) and protein urea (pre-eclampsia).

Specific exclusion criteria at the time of recruitment included a history of cardiac disease or suggestion of chronic hypertension, or long –term use of medication, and multiple pregnancy. In addition, all subjects had baseline diastolic blood pressure less than 90 mmHg; were considered normal on basis of physical examination, electrocardiography, and echocardiography and had normal hemoglobin at the time of recruitment. All measurement were made with the subject in a semi recumbent, left- lateral position after at least 10 minutes rest. Blood pressure was measured using a mercury

sphygmomanometer. Heart rate was recorded from simultaneous three-lead electrocardiogram. Echocardiography was performed when patients were hem dynamically stable.

Total peripheral resistance (TPR) was calculated according to the formula;

TPR=mean arterial pressure (mmHg) /cardiac output (L.min) 80dynes.sec.cm-5

⬇

Where mean arterial pressure =2× diastolicBP+ systolicBP/3

Urine for protein is also sent

RESULTS

- Healthy pregnant women
- Preeclamptic pregnant women

Statistical analyses: The SPSS 15 for Window was used for all analysis ,Pearson correlation coefficient test and Chi Square $\chi 2$, Data from the two groups of patients are presented as means& standard error and evaluated by use of T-tests ($p<0.05$) regarded as statistically significant. $\chi 2$ (Chi- square) was applied for categorical association at level of significance $\alpha = 0.05$.

The value of studied parameters (heart rate, systolic &diastolic blood pressure)where increased in preeclamptic women in comparison with normal women.

All parameters increased significantly at $\alpha \leq 0.0001$

Table 1. Comparison of clinical variables from the healthy women and preeclamptic women

Groups	Heart rate Mean+SE (range)	Systolic blood pressure. Mean+SE (range)	Diastolic blood pressure Mean+SE (range)
Healthy pregnant women	78±1.24 (70-90)	110.25±2.06 (95-130)	62±1.5977 (50-70)
Preeclamptic pregnant women	100.5±2.32 (88-125)	161.500±3.57 (145-190)	94.75±1.22 (90-110)
p-value	0.0001	0.0001	0.0001

(Significant Differences were assessed by T –test analysis)

2-Preeclampsia and clinical variables (age, hemoglobin concentration, and body mass index)
Age show no significant difference between the two groups
While Hb & variables BMI are significant at $\alpha \leq 0.05$

Table 2. Comparison of (Age, Hb, BMI) from healthy pregnant women and preeclamptic pregnant women

	Age Mean+SE (Range)	Hb Mean+SE (range)	BMI Mean+SE (range)
Healthy pregnant women	27.50±1.468 (18-43)	10.80±0.229 (8.5-13)	30.59±0.858 (24.4-35.9)
Preeclamptic pregnant women	30.9±1.174 (25-40)	9.42±0.176 (8-11)	33.52±0.419 (29.29-36.6)
p-value	0.078	0.000	0.004

(significant differences assessed by T-test analysis). Significant differences at $\alpha \leq 0.05$. 3-The values of studied parameters (CO, TPR) assessed by T-test , showed statistically significant difference in CO (p-value $\leq$ 0.05) but not significant in TPR (p-value = 0.078).

Table 3. Comparison of (CO&TPR) between healthy pregnant women & preeclamptic pregnant women

	Cardiac output Mean+SD range	Total peripheral resistance Mean+SD range
Healthy pregnant women	5.45±0.101 (4.5-6.3)	1154.24±28.41 (896.3-1359)
Preeclamptic pregnant w0men	7.691±0.347 (6.26-11.8)	1252.94±46.16 (857-1517.9)
p-value	0.0001	0.078

Table 4. Preeclampsia and the mode of delivery

	NVD number	NVD* percentage	*c-section number	c-section percentage
Healthy pregnant women	15	75%	5	25%
Preeclamptic pregnant women	5	25%	15	75%

The difference is highly significant by chi-square test . Chi-square $\chi 2 = 27.85$, p value ≤ 0.004. For normal & preeclamptic patients, there is a highly significant association between preeclampsia & mode of delivery

*NVD= normal vaginal delivery
*c-section= cesarean section.

DISCUSSION

- Pre-eclampsia is a major progressive disorder of pregnancy, that results from various micro vascular diseases, it can cause multi-organic involvement including the brain, Kidney, liver, and lung.
- The key signs of pre-eclampsia are hypertension and Protienurea & these are responses to end-organ damage and they are not always the most important nor fundamental aspects of the syndrome, but are used as they are easy to measure.
- As delivery is the only cure for preeclampsia ,it is the commonest cause of iatrogenic prematurity accounting for 15% of all premature births and approximately one in five very low birth weight infants (<1500g)
- Hypertention remain a major cause of morbidity and mortality, a pregnant women with pre-eclampsia remain at risk for sever complication such as abruptio placentae, end organ failure, and disseminated intravascular coagulation with the fetus at risk of intrauterine growth restriction, prematurity, and intrauterine death.
- preeclampsia occurs in approximately 5% of all pregnancies. The incidence of preeclampsia23.6 cases per 1,000deliveriesin the unitedstates, the global incidence of preeclampsia has been estimated at 5-14% of all pregnancies.
- In our study documentation of maternal central homodynamic during clinical phase of preeclampsia support the concept of a hyper dynamic disease model for preeclampsia

- Also in our study we have no patient with pulmonary edema, renal failure and HELLP syndrome because small number of patient.
- In our study we have two groups healthy and preeclamptic pregnant women, there was no significant difference in age between the two groups, body mass index at recruitment was higher in preeclamptic (p value<o.oo4) than in normotensive controls. In addition serum hemoglobin concentration show significant difference p value <o.ooo1.
- Regarding cardiac output, the study showed highly significant difference between two groups (p value<o.ooo1), while total peripheral resistance show increase in patients over healthy control but not significantly high p value =0.078.
- The mechanism by which cardiac output increase in preeclampsia remain un known. Possible explanations include an increased heart rate secondary to a chronic state of sympathetic overactivity, and the indirect effects of a relative vascular underfill. Both of those conditions could be secondary to defective baroreceptor resetting or deficient plasma volume expansion, the resulting state of vascular underfill could drive cardiac output, and the compensatory vasodilatation would further reduce the vasodilatory reserve. These condition would result in the increased sensitivity to angiotensin 2 associated with preeclampsia.
- Our study show most of preeclamptic patient to have increased cardiac output more than 6.3 l/min while normotensive patients cardiac outputs were <6.3 l/min.
- The hemodynamics of preeclampsia may or may not play a fundamental role in the pathophysiology of disease, but whether the condition characterized as a high output and low resistant state or vice versa could have clinical implication.
- Most preeclamptic pregnant women with high cardiac output (CO >6.3 l/min) delivered by caesarean section and this is highly significant. High percentage of caesarean section is due to fetal condition that is more common in this group and necessitate immediate delivery.

Conclusion

Our study supports the concept of a hyper dynamic disease model for preeclampsia.

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