

# Clinical Presentations in Preeclamptic Women Attending Elnihoud Teaching Hospital

Mubarak Adam Suleiman Ameen<sup>1</sup>, Hafiz Ahmed Hobiel Ahmed<sup>2,\*</sup>

<sup>1</sup>Department of Obstetric and Gynecology, Faculty of Medicine and Health Sciences, West Kordufan University, Elnihoud City, Sudan

<sup>2</sup>Department of Biochemistry, Faculty of Medicine and Health Sciences, West Kordufan University, Elnihoud City, Sudan

## Email address

Mubarak amin7@gmail.com (M. A. S. Ameen), hebriel78@yahoo.com (H. A. H. Ahmed)

\*Corresponding author

## To cite this article

Mubarak Adam Suleiman Ameen, Hafiz Ahmed Hobiel Ahmed. Clinical Presentations In Preeclamptic Women Attending Elnihoud Teaching Hospital. *Open Science Journal of Clinical Medicine*. Vol. 7, No. 2, 2019, pp. 71-75.

Received: May 13, 2019; Accepted: July 5, 2019; Published: July 15, 2019

## Abstract

Preeclampsia is the most common hypertensive disorders of pregnancy which is an idiopathic multisystem disorder that contributes greatly to the maternal morbidity and mortality. To evaluate the clinical presentations in preeclamptic women attending Elnihoud Teaching Hospital and to compare the findings between severe and mild cases. This study was descriptive cross sectional study; carried out in Elnihoud Teaching Hospital. A total of forty tow pregnant women were included in this study. They were selected from the Wards of the Hospital at admission before starting treatment. Clinical examination was done after taking of permission from every participants. The study revealed significant elevation in the clinical presentation of headache ( $p$ -value =  $< 0.0001$ ), blurring vision ( $p$ -value =  $< 0.0001$ ), epigastric pain ( $p$ -value =  $< 0.0001$ ), RHP ( $p$ -value =  $0.006$ ), breathlessness ( $p$ -value =  $0.015$ ) abdominal pain ( $p$ -value =  $0.002$ ), tachycardia ( $p$ -value =  $0.010$ ), less than date fundal level ( $p$ -value =  $0.005$ ), hyper reflexes ( $p$ -value =  $0.007$ ), lower limbs edema ( $p$ -value =  $0.009$ ), and papillodema ( $p$ -value =  $0.002$ ) among sever preeclamptic patients compared to mild cases. Elevation in clinical presentations associated with preeclamptic women might reflect the severity of the disease.

## Keywords

Preeclampsia, Hypertension, Breathlessness, Papillodema

## 1. Introduction

Pregnancy is characterized by significant metabolic and hemodynamic changes that begin early in the gestational period. Major hemodynamic changes include an increase in the cardiac output during the first trimester, sodium and water retention leading to plasma volume expansion with a peak around week 30, and reductions in the systemic vascular resistance and systemic blood pressure [1]. Hypertension is the most prevalent maternal complication worldwide (several studies estimate that it affects 7–10% of all pregnancies) [2], and it is associated with a significant morbidity and mortality of the mother and fetus. In fact, hypertension is the second largest cause of direct maternal death worldwide (14% of the total) [3], and it is estimated that 192 people die every day because of hypertensive disorders in pregnancy [4].

Preeclampsia and eclampsia are two hypertensive disorders of pregnancy, considered as major causes of maternal and perinatal morbidity and mortality [2].

Preeclampsia is a multisystemic disease characterized by the development of hypertension after 20 weeks of gestation in a previously normotensive woman, with the presence of proteinuria or, in its absence, of signs or symptoms indicative of target organ injury [5]. The clinical signs involve multiple organs, including the liver, kidneys, heart, lungs, brain, and pancreas. These complications can result in maternal and fetal adverse outcomes that can lead to intrauterine growth restriction, placental hypoperfusion, premature placental disruption or, in most serious situations, termination of pregnancy and fetal and maternal death [6].

Women with severe preeclampsia may present headaches, visual disturbances (including blindness), epigastric pain, nausea and vomits, hepatic and renal insufficiency, and

pulmonary edema [7].

The incidence of preeclampsia is also explained by several risk factors that include maternal age under 20 years old or over 40 years old, history of preeclampsia, previous hypertension, autoimmune diseases, and obesity [8]. A woman is at moderate risk for preeclampsia if she has no more than one risk factor; a woman is at high risk for pre-eclampsia if she has two or more risk factors for the disease [9].

Although it is a well-studied disease, the pathophysiology of preeclampsia remains uncertain. Several key features are thought to have a role in the development of pre-eclampsia, which is mainly considered as a vascular disorder. The most probable causes for this disease are a failure of trophoblast invasion leading to a failed transformation of the uterine spiral arteries, and an incorrect deep placentation [10].

Trophoblasts are the first cells that differentiate from the fertilized egg, they form the outer membrane of the placenta, and are responsible for the nutrients and oxygen exchange between the mother and the fetus [11]. Also, decidual natural killer (NK) cells can regulate trophoblast invasion and vascular growth, two essential processes in placental development [12].

An abnormal expression of NK cell surface antigens and a failure in the regulation of NK cell cytotoxicity and cytokines or angiogenic factors may be some of the causes of preeclampsia [13], resulting in a high-flow and high-pressure state [14]. Consequently, there is a high risk for ischemia-reperfusion injury of the placenta because of the vasoconstriction of the maternal arteries, which will lead to the formation of reactive oxygen radicals and further endothelial dysfunction [15]. Thus, preeclampsia can be related with the excessive release of some mediators by the injured endothelial cells.

This study aimed to evaluate headache, blurring of vision, epigastric pain, right hypochondrial pain (RHP), breathlessness, abdominal pain, fetal heart rate (FHR), fundal level, reflexes, lower limbs edema and papilloedema as clinical presentations of preeclampsia for preeclamptic patients attending Elnihoud Teaching Hospital and to compare the findings between mild and severe cases.

## 2. Material and Methods

This study was descriptive cross sectional study; carried out in Elnihoud Teaching Hospital, Elnihoud Locality, West Kordufan State, Sudan. From January 2018 to December 2018. A total of forty tow pregnant women were included in this study. They were selected from the Wards of the Hospital at admission before starting treatment. Institutional research and ethics approval was obtained before commencement of the study. All participants spoke sufficient Arabic to provide informed consent.

### 2.1. Inclusion Criteria

Preeclamptic women with blood pressure  $\geq 140/90$ , and proteinuria  $\geq 300\text{mg}/24\text{hrs}$  urine collection were included.

Preeclamptic patients with blood pressure  $\geq 160/110$  or/and

proteinuria  $\geq 1\text{g}/24$  hours urine collection or/and presence of papilloedema were taken as severe preeclamptic cases, while preeclamptic patients with blood pressure  $159/109 - 140/90$ , proteinuria  $0.3$  to  $1\text{g}/24$  hours urine collection and absence of papilloedema taken as mild preeclampsia.

### 2.2. Exclusion Criteria

Pregnant women with pre-gestational cardiac, hepatic or renal disorders, diabetes mellitus, primary or secondary lipid disorders, severe anemia, those suffer from any other hematological or endocrine disorders were excluded.

Clinical examination was done and questionnaires were filled for every participant. Data were analyzed by SPSS program version 20.

## 3. Results

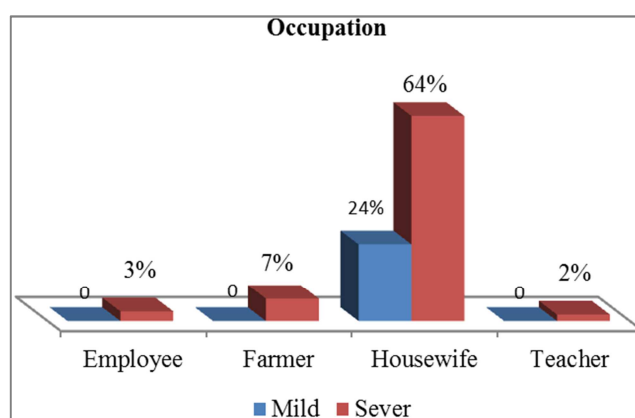


Figure 1. Occupations of the study group.

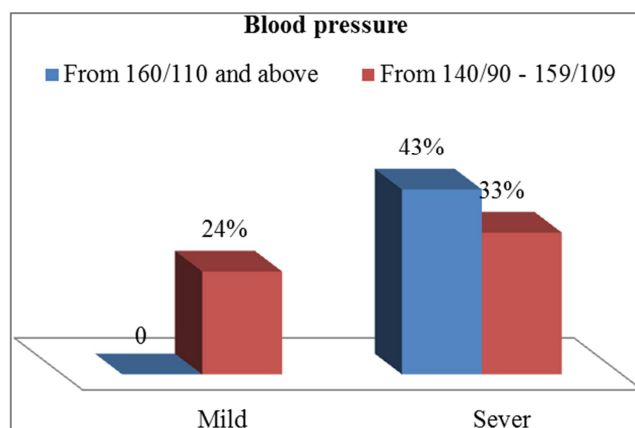


Figure 2. Blood pressure of the study group.

Figure 1 shows the occupation of the study group, in which (88%) were house wife, (7%) farmer, (3%) employee and (2%) teacher. Figure 2 shows the Blood pressure of the study group. From the entire participants, (43%) their blood pressure  $\geq 1160/110$  (sever preeclampsia) and (57%) their blood pressure from  $140/90 - 159/109$  (33% with sever preeclampsia for other causes and 24% with mild preeclampsia). Figure 3 presents the preeclampsia status of the study group, (76%) of the study group have sever

preeclampsia and (24%) have mild preeclampsia. Figure 4 revealed the causes of sever preeclampsia for the study group. The most common cause of the sever preeclampsia was blood pressure (36%) followed by proteinuria (28%) then (13%) both blood pressure and papilloedema, (13%) blood pressure, proteinuria and papilloedema, (7%) both blood pressure and proteinuria, (3%) papilloedema.

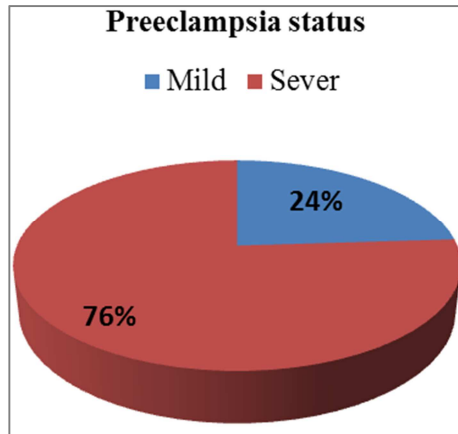


Figure 3. Preeclampsia status of the study group.

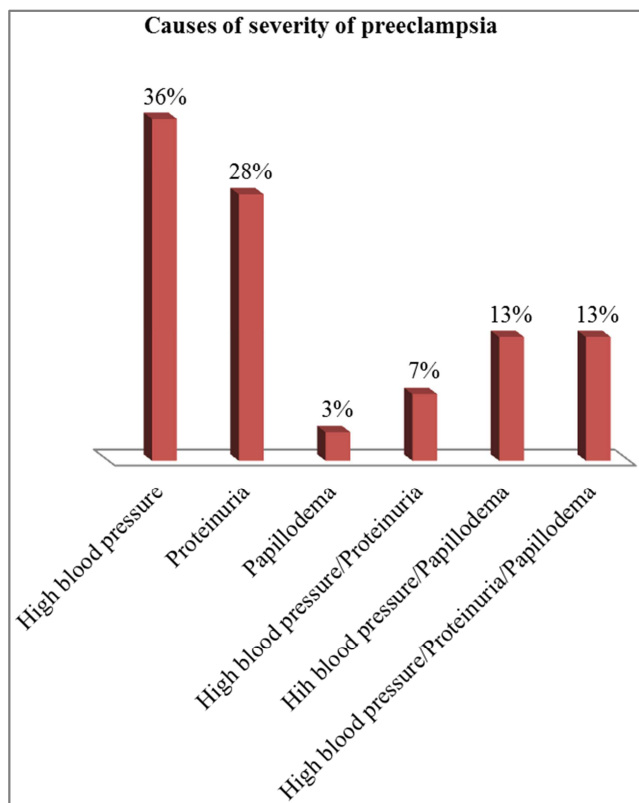


Figure 4. Causes of sever preeclampsia for the study group.

### 3.1. Characteristics and Description of the Study Group

Table 1 shows the Characteristics and description of the study group. The participants ages per year were 14 – 20 (28.6%), 21 – 25 (21.4%), 26 – 30 (31%) and > 30 (19%). The study group ages at time of marriage per year were 14 – 20

(73.8%), 21 – 25 (14.3%), 26 – 30 (7.1%) and > 30 (4.8%) years old. For gestational ages at time of diagnosis/week (onset of preeclampsia) they were 20 – 24 (9.5%), 24 + 1 – 28 (7.1%), 28+1 – 32 (19%), 32 + 1 – 36 (40.5%) and > 36 (23.9%). The parity for the participants were primiparous (45.2%), multiparous (35.7%) and grand multiparous (19.1%).

Table 1. Characteristics and description of the study group.

Character	Preeclampsia status		Total	p-value
	Mild	Sever		
Age/year				
14 -20	3 (30%)	9 (28.1%)	12 (28.6%)	1.000
21 -25	2 (20%)	7 (21.9%)	9 (21.4%)	
26 – 30	3 (30%)	10 (31.2%)	13 (31%)	
> 30	2 (20%)	6 (18.8%)	8 (19%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Age at time of marriage/year				
14 – 20	7 (70%)	24 (75%)	31 (73.8%)	0.746
21 – 25	2 (20%)	4 (12.5%)	6 (14.3%)	
26 – 30	0	3 (9.4%)	3 (7.1%)	
> 30	1 (10%)	1 (3.1%)	2 (4.8%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Gestational age at time of diagnosis/week				
20 – 24	2 (20%)	2 (6.3%)	4 (9.5%)	0.394
24 + 1 – 28	0	3 (9.3%)	3 (7%)	
28 + 1 – 32	2 (20%)	6 (18.8%)	8 (19%)	
32 + 1 – 36	5 (50%)	11 (34.4%)	17 (40.5%)	
> 36	1 (10%)	10 (31.2%)	10 (23.8%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Parity				
Primiparous	3 (30%)	16 (50%)	19 (45.2%)	0.012
Multiparous	5 (50%)	10 (31.2%)	15 (35.7%)	
Grand multiparous	2 (20%)	6 (18.8%)	8 (19.1%)	
Total	10 (100%)	32 (100%)	42 (100%)	

### 3.2. Clinical Presentation Findings of the Study Group

Table 2 represents the clinical presentation findings for the study group. Regarding headache presentation, it presents in (57.1%) of participants and absent in (42.9%), and it was significantly higher in sever preeclamptic than mild cases ( $p$ -value < 0.0001). For blurring vision, (33.3%) of the participants have blurring vision versus (66.7%) have no blurring vision, and it was highly significant in sever preeclamptic patients compared to mild preeclamptic cases ( $p$ -value < 0.0001). Concerning epigastric pain, (47.6%) of participants have epigastric pain versus (52.4%) with no epigastric pain, and it was significantly higher in sever preeclamptic than mild cases ( $p$ -value < 0.0001). For RHP, (26.2%) of the study group have RHP while (73.8%) have no RHP, and it was significant in severe cases compared to mild cases ( $p$ -value 0.006). Regarding breathlessness, (16.7%) of the participants have breathlessness versus (83.3%) with no breathlessness and it was significantly elevated in severe preeclamptic versus mild cases ( $p$ -value 0.015). From the all participants, (23.8%) have abdominal pain and (76.2%) have no abdominal pain with significant value in sever versus mild patients ( $p$ -value 0.002). Concerning FHR, from entire participants (88.1%) have normal FHR, (4.8%) have tachycardia and (7.1%) have bradycardia and there was

significant increase regarding tachycardia in severe cases compared to mild cases ( $p$ -value 0.010). The fundal level presentation for the study group, more than date (9.5%), less than date (21.4%) and equal date (69.1%), and there was significant increase in less than date fundal level among severe versus mild cases ( $p$ -value 0.005). From entire participants (76.2%) have normal reflexes, (21.4%) have hyper reflexes and (2.4%) have hypo reflexes with significant elevation in hyper reflexes among severe preeclampsia versus mild preeclampsia ( $p$ -value 0.007). For lower limbs edema, (76.2%) of the study group have lower limb edema and (23.8%) revealed no lower limb edema and it was significant in severe preeclampsia compared to mild preeclampsia ( $p$ -value 0.009). From all participants, (21.4%) present papilledema and (78.6%) revealed no papilledema and it was significant in severe preeclamptic in relation to mild preeclamptic cases ( $p$ -value 0.002).

**Table 2.** Clinical presentation findings of the study group.

Character	Preeclampsia status			$p$ -value
	Mild	Sever	Total	
Headache				
Present	1 (10%)	23 (71.9%)	24 (57.1%)	0.0001
Absent	9 (90%)	9 (28.1%)	18 (42.9%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Blurring vision				
Present	0	14 (43.8%)	14 (33.3%)	0.0001
Absent	10 (100%)	18 (56.2%)	28 (66.7%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Epigastric pain				
Present	1 (10%)	19 (59.4%)	20 (47.6%)	0.0001
Absent	9 (90%)	13 (40.6%)	22 (52.4%)	
Total	10 (100%)	32 (100%)	42 (100%)	
RHP				
Present	1 (10%)	10 (31.2%)	11 (26.2%)	0.006
Absent	9 (90%)	22 (68.8%)	31 (73.8%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Breathlessness				
Present	1 (10.3%)	6 (18.7%)	7 (16.7%)	0.015
Absent	9 (90%)	26 (81.3%)	35 (83.3%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Abdominal pain				
Present	0	10 (31.2%)	10 (23.8%)	0.002
Absent	10 (100%)	22 (68.8%)	32 (76.2%)	
Total	10 (100%)	32 (100%)	42 (100%)	
FHR				
Normal	10 (100%)	27 (84.4%)	37 (88.1%)	0.010
Tachycardia	0	2 (6.2%)	2 (4.8%)	
Bradycardia	0	3 (9.4%)	3 (7.1%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Fundal level				
> date	0	4 (12.5%)	4 (9.5%)	0.005
< date	1 (10%)	8 (25%)	9 (21.5%)	
Equal date	9 (90%)	20 (62.5%)	29 (69%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Reflexes				
Hyper	0	9 (28.1%)	9 (21.4%)	0.007
Hypo	0	1 (3.1%)	1 (2.4%)	
Normal	10 (100%)	22 (68.8%)	32 (76.2%)	
Total	10 (100%)	32 (100%)	42 (100%)	
Lower limbs edema				
Positive	9 (90%)	23 (71.9%)	32 (76.2%)	0.009
Negative	1 (10%)	9 (28.1%)	10 (23.8%)	
Total	10 (100%)	32 (100%)	42 (100%)	

Character	Preeclampsia status			$p$ -value
	Mild	Sever	Total	
Papilledema				
Positive	0	9 (28.1%)	9 (21.4%)	0.002
Negative	10 (100%)	23 (71.9%)	33 (78.6%)	
Total	10 (100%)	32 (100%)	42 (100%)	

## 4. Discussion

Although PE only affects approximately 2% – 8% of pregnancies worldwide it is associated with severe complications such as eclampsia, hemorrhagic stroke, hemolysis, elevated liver enzymes and low platelets (HELLP syndrome), renal failure and pulmonary edema in addition to other variable mode of clinical presentation and hematological and biochemical changes. Importantly, there is no “cure” for the disease except for early delivery of the baby and placenta [16]. Hypertension, proteinuria, excessive weight gain and edema are classic clinical manifestations of the preeclampsia [17].

The current study revealed that most of the participants were house wife (88%), marriage at age 14 – 20 years old (73.8%), primiparous (45.2%), their blood pressure from 160/110 and above (57%) with severe preeclampsia (76%) and the main cause of preeclampsia was high blood pressure (36%).

Headaches and visual symptoms, epigastric abdominal pain, nausea and vomiting have been consistently reported as important premonitory symptoms for preeclampsia [18].

The current study shows highly significant elevation in headache among the severe preeclamptic cases compared to mild cases. The headache might present in mild cases of preeclampsia but not common. Blurring vision significantly higher in severe preeclamptic patients and completely absent in mild cases of preeclampsia. The present study revealed highly significant increase in epigastric pain cases among severe preeclamptic patients, and it might be presented by mild cases but not common. RHP was highly significant in severe preeclamptic patients compared to mild cases and it might presented by some mild cases. These findings were in accordance with works of [19, 20] whom reported similar results. Severe preeclamptic patients present significant elevation in breathlessness compared to mild cases. Abdominal pain were highly significant among severe preeclampsia cases and it was completely absent among mild cases of preeclampsia. These findings in agreement with similar results reported by [21].

The current study revealed that tachycardia and bradycardia were completely absent among mild cases of preeclampsia and therefore they was significantly elevated tachycardia among severe cases of preeclampsia. There was significant elevation in the cases having less than date fundal level among severe preeclamptic patients compared to mild cases. No previous study supports or dissent these results.

Hyper reflexes and hypo reflexes were completely absent among mild preeclamptic patients. Hyper reflexes and hypo reflexes were significantly higher in severe cases versus mild cases. These results were in accordance with similar report

stated by [22] they found there was significant elevation in hyper reflexes among sever preeclamptic cases. Lower limbs edema was significantly higher among sever preeclamptic cases than mild cases. Papillodema was highly significant among sever preeclamptic patients compared to mild cases and it was completely absent in mild preeclamptic patients and there was no previous study supports or dissent these results. The results of clinical presentations revealed by this study might attributed to the neurological system impairment and vasospasm which associated with preeclampsia.

## 5. Conclusion

Clinical presentations associated with preeclamptic women particularly headache, blurring vision, epigastric pain, RHP, breathlessness, abdominal pain, tachycardia, less fundal level, hyper reflexes, lower limbs edema and papillodema might reflect the severity of the disease and therefore might be used as diagnostic tools for sever preeclampsia.

## 6. Recommendations

More studies with further parameters can provide guidance for the prevention, management and follow-up of women who experience PE.

## References

- [1] Gongora, M. C. and Wenger, N. K. (2015) Cardiovascular Complications of Pregnancy *Int J Mol Sci* Volume (10): 23905-23928.
- [2] Lindheimer, M. D., Taler, S. J. and Cunningham, F. G. (2010) Hypertension in pregnancy *J Am Soc Hypertens* Volume (2): 68-78.
- [3] Say, L., Chou, D., Gemmill, A., Tuncalp, O., Moller, A. B., Daniels, J., Gulmezoglu, A. M., Temmerman, M. and Alkema, L. (2014) Global causes of maternal death: a WHO systematic analysis *Lancet Glob Health* Volume (6): e323-333.
- [4] Folic, M., Folic, N., Varjacic, M., Jakovljevic, M. and Jankovic, S. (2008) Antihypertensive drug therapy for hypertensive disorders in pregnancy *Acta Med. Median* Volume (47): 65-72.
- [5] Moussa, H. N., Arian, S. E. and Sibai, B. M. (2014) Management of hypertensive disorders in pregnancy *Womens Health (Lond)* Volume (4): 385-404.
- [6] Chaiworapongsa, T., Chaemsaitong, P., Yeo, L. and Romero, R. (2014) Pre-eclampsia part 1: current understanding of its pathophysiology *Nat Rev Nephrol* Volume (8): 466-480.
- [7] Practice, A. C. o. O. (2002) ACOG practice bulletin. Diagnosis and management of preeclampsia and eclampsia. Number 33, January 2002. American College of Obstetricians and Gynecologists *Int J Gynaecol Obstet* Volume (1): 67-75.
- [8] Grand'Maison, S., Pilote, L., Okano, M., Landry, T. and Dayan, N. (2016) Markers of Vascular Dysfunction After Hypertensive Disorders of Pregnancy: A Systematic Review and Meta-Analysis *Hypertension* Volume (6): 1447-1458.
- [9] English, F. A., Kenny, L. C. and McCarthy, F. P. (2015) Risk factors and effective management of preeclampsia *Integr Blood Press Control* Volume 7-12.
- [10] Fisher, S. J. (2015) Why is placentation abnormal in preeclampsia? *Am J Obstet Gynecol* Volume (4 Suppl): S115-122.
- [11] Gathiram, P. and Moodley, J. (2016) Pre-eclampsia: its pathogenesis and pathophysiology *Cardiovasc J Afr* Volume (2): 71-78.
- [12] Hanna, J., Goldman-Wohl, D., Hamani, Y., Avraham, I., Greenfield, C., Natanson-Yaron, S., Prus, D., Cohen-Daniel, L., Arnon, T. I., Manaster, I., Gazit, R., Yutkin, V., Benharroch, D., Porgador, A., Keshet, E., Yagel, S. and Mandelboim, O. (2006) Decidual NK cells regulate key developmental processes at the human fetal-maternal interface *Nat Med* Volume (9): 1065-1074.
- [13] Fukui, A., Yokota, M., Funamizu, A., Nakamura, R., Fukuhara, R., Yamada, K., Kimura, H., Fukuyama, A., Kamoi, M., Tanaka, K. and Mizunuma, H. (2012) Changes of NK cells in preeclampsia *Am J Reprod Immunol* Volume (4): 278-286.
- [14] Tessier, D. R., Yockell-Lelievre, J. and Gruslin, A. (2015) Uterine Spiral Artery Remodeling: The Role of Uterine Natural Killer Cells and Extravillous Trophoblasts in Normal and High-Risk Human Pregnancies *Am J Reprod Immunol* Volume (1): 1-11.
- [15] Burton, G. J., Woods, A. W., Jauniaux, E. and Kingdom, J. C. (2009) Rheological and physiological consequences of conversion of the maternal spiral arteries for uteroplacental blood flow during human pregnancy *Placenta* Volume (6): 473-482.
- [16] Amaral, L. M., Cunningham, M. W., Jr., Cornelius, D. C. and LaMarca, B. (2015) Preeclampsia: long-term consequences for vascular health *Vasc Health Risk Manag* Volume 403-415.
- [17] Luft, F. C. (2004) Hypertensive nephrosclerosis: update *Curr Opin Nephrol Hypertens* Volume (2): 147-154.
- [18] Cooray, S. D., Edmonds, S. M., Tong, S., Samarasekera, S. P. and Whitehead, C. L. (2011) Characterization of symptoms immediately preceding eclampsia *Obstetrics & Gynecology* Volume 995 – 999.
- [19] Gustavo, V., Luis R, H., Jocelyn Leon, P., Moraima, L. and Pedro, A. (2016) Differences in clinical presentation and pregnancy outcomes in antepartum preeclampsia and newonset postpartum preeclampsia: Are these the same disorder? *Obstet Gynecol Sci* Volume (6): 434-443.
- [20] Zehra, K., Mertihan, K., Glucin, A. and Tekin, Y. (2011) Retinal Findings in Cases of Preeclampsia *Perinatal Journal* Volume (2): 60-63.
- [21] Erdem, D., Mustafa, V., Ozer, D., Fatma Merve, B., Ayse Ayca, S., Y., A., Ufuk, A., Ozlem, Y. and Mehmet Atila, A. (2017) Evaluation of fundus findings in preeclampsia *Medicine Science International Medical Journal* Volume (3): 406-409.
- [22] Lewinsky, R. M. and Riskin-Mashiah, S. (1998) Autonomic imbalance in preeclampsia: evidence for increased sympathetic tone in response to the supine-pressor test *Obstet Gynecol* Volume (6): 935-939.