

## Original Research

### Clinical and radiological profile of cerebrovascular disease in hypertension during pregnancy

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#### ABSTRACT:

**Introduction:** Risk factors for stroke during pregnancy and the puerperal period include gestational hypertension, HELLP syndrome (characterized by hemolysis, elevated liver enzymes, and thrombocytopenia), frequent vomiting, and changes in hemolysis and the pre-thrombotic state in late pregnancy and the postpartum period. **Materials and Methods:** A total of 65 patients were selected after meeting the inclusion criteria. A detailed history was taken and clinical examination was done. The study was carried out after seeking permission from institutional ethical committee and written consent was obtained from all the patients before starting of the study. The outcome of interest was early-onset CVD (excluding congenital heart disease), defined as the first occurrence of CVD in the DNPR and the Danish Cause of Death Register (Diagnostic codes and surgical codes for CVD were provided). **Results:** The age range of 25 to 30 years old had the greatest number of patients. The occurrence of hypertension problems during pregnancy is significantly influenced by age. The patients' average age was 25.26 years. The mean mother age at diagnosis was 28 years old. The majority of patients (58%) and post-natal patients were included in our study. The mean gestational age of the prenatal patients was 34.14 weeks. **Conclusion:** In a country like ours, it is also important to correct anemia and avoid dehydration in the peripartum period. There should be greater awareness and liberal use of CT/MRI in those cases who presented with seizures in the pregnancy/puerperium, and in those cases presenting with loss of consciousness.

**Keywords:** Clinicoradiological, cerebrovascular disease, hypertensive disease.

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#### INTRODUCTION

Ischemic stroke, subarachnoid haemorrhage, eclamptic encephalopathy, postpartum cerebral angiopathy, and cerebral venous thrombosis are the different categories of cerebral vascular diseases.<sup>1</sup> A number of imaging studies, such as computed tomography (CT) with or without ionic contrast, CT angiography or CT venography, magnetic resonance imaging with or without contrast, magnetic resonance angiography, or magnetic resonance venography, are available for the evaluation of cerebrovascular disease in order to assess the parenchyma, vessels, and extraparenchymal spaces. Additional investigations that aid in determining the aetiology of the illness include transcranial Doppler, transesophageal echocardiography, and electrocardiography with or without agitated saline echo contrast. Conventional

arteriography or lumbar puncture may offer more information in certain circumstances. These investigations are generally useful in assessing for infection (lumbar puncture) or inflammation (arteriography and lumbar puncture). Because radiation exposure can produce teratogenic effects, it is always a concern for the doctor who is caring for a pregnant patient.<sup>2</sup>

Gestational hypertension, HELLP syndrome (defined as hemolysis, elevated liver enzymes, and thrombocytopenia), frequent vomiting, and alterations in hemolysis and the pre-thrombotic state in late pregnancy and the postpartum period are risk factors for stroke during pregnancy and the puerperal period.<sup>3</sup> Incidence of stroke may also be increased by multiparity, older age of pregnancy, repeated pregnancies, diabetes, and an imbalance between

electrolytes and water.<sup>4, 5</sup>

Pregnancy-related acute cerebrovascular problems are a major issue that have the potential to kill both the mother and the fetus. The long-term effects of these issues can also have a detrimental effect on a woman's quality of life in her later years. Six Quick brain imaging can be used to diagnose acute stroke, pinpoint the origin of the stroke, and establish the best course of action for therapy.

We designed the current study to evaluate the clinic-radiological outcome of cerebrovascular disorders in peripartum females in light of the previously reported findings.

## MATERIAL AND METHODS

A total of 65 patients were selected after meeting the inclusion criteria. A detailed history was taken and clinical examination was done. The study was carried out after seeking permission from institutional ethical committee and written consent was obtained from all the patients before starting of the study.

The outcome of interest was early-onset CVD (excluding congenital heart disease), defined as the first occurrence of CVD in the DNPR and the Danish Cause of Death Register (Diagnostic codes and surgical codes for CVD were provided. We further investigated type-specific CVDs, such as myocardial infarction, cerebrovascular disease, stroke, heart failure, atrial fibrillation, hypertensive disease, deep vein thrombosis, pulmonary embolism, rheumatic heart disease, and peripheral arterial disease. Obstetric data such as parity, antenatal care, gestational age at the onset of symptoms and the presence of complications like Preeclampsia, eclampsia, anemia, and sepsis were noted.

### Inclusion criteria for the present study

Peripartum females with history of hypertension.

### Exclusion criteria for the present study

Patients with history of any systemic illness,

Patients with history of any benign or malignant neoplasm involving central nervous system,

Patients with cardiac pace makers or in which MRI is contraindicated.

Patients who were not willing to participate in the study.

## COVARIATES

Potential confounders were selected by directed acyclic graphs (S1 Fig), including sex (male, female), singleton (yes, no), birth year of the child (1977 to 1980, 5-year intervals during 1981 to 2015, and 2016 to 2018), maternal age (<20, 20 to 24, 25 to 29, 30 to 34, or ≥35 years), maternal education (0 to 9, 10 to 14, or ≥15 years), maternal income at birth (no income, 3 tertiles), maternal prepregnancy BMI (underweight <18.5, normal 18.5 to 24.9, overweight 25.0 to 29.9, obese ≥30.0), maternal smoking during pregnancy (yes or no), parity (1, 2, or ≥3 children), maternal cohabitation (single or cohabitating), maternal residence (Copenhagen, cities with ≥100,000 inhabitants, or other), maternal history of diabetes, and maternal and parental history of CVD before childbirth (yes or no). A missing indicator method was used to deal with missing values. A detailed description of the covariates is presented in S3 Text.

## STATISTICAL ANALYSIS

Considering non-CVD deaths as the competing events, competing risk analysis was performed to estimate cumulative incidence of CVD among offspring exposed and unexposed to maternal HDP. We used Cox regression to estimate hazard ratios (HRs) and 95% CIs to assess the association between maternal HDP and overall or type-specific CVD in offspring. The proportional hazards assumption was assessed graphically using the log-minus-log plot, suggesting that there was no obvious violation. We examined the interaction term between maternal HDP and maternal history of CVD or diabetes to assess whether the association was varied by maternal CVD or diabetes. Besides, we assessed the association by timing of onset and severity of preeclampsia (moderate, severe eclampsia, and HELLP syndrome).

## RESULTS

**TABLE 1: AGE DISTRIBUTION**

Age(years)	Number of patients(65)	Percentage
≤20	8	8%
21–30	46	84%
>30	11	13%
Mean age±SD	26.21±5.80	
Minimum age	18 years	
Maximum age	38 years	

**TABLE 2: DISTRIBUTION OF GESTATIONAL AGE**

Gestational age	Number of patients	Percentage
Gestation period	28	44%
Postnatal	37	59%
Mean±SD	35.10±3.1	

**TABLE3: PRESENTING SYMPTOMS WITH MEAN DURATION**

Presenting symptoms	Number of patients	Mean duration(days)
Seizures	31 (57%)	1.35
Blurred Vision	17 (30%)	1.38
Severe Headache	6 (7%)	1.7
Unconsciousness	5 (6%)	1
Sudden Loss of Vision	2(3%)	1
Weakness on Left Side of Body	2(3%)	1
Altered Sensorium	2(3%)	1

**TABLE 4: DISTRIBUTION OF HAEMOGLOBIN**

	Range	No. of patients	Percentage
Normal Hb	>10	26	46%
Mild anaemia	8.1-10g/dl	18	29%
Moderate anaemia	6.5-8g/dl	16	25%
Severe anaemia	<6.5g/dl	5	6%
Mean Hb±SD	10.7±1.9		

**TABLE 5: MEAN SERUM CREATININE AND SERRUM UREA LEVELS LEVELS (MG/DL)**

		Grade I hypertension	Grade II hypertension	Grade III hypertension	Pvalue
Mean	Serum Creatinine	0.68±0.15	0.73±0.28	0.79±0.22	0.5
	Serum Urea	27.91±11.71	31.21±7.8	32.71±6.1	0.42

**TABLE 10: MRI/CTFINDINGS**

	Grade I hypertension	Grade II hypertension	Grade III hypertension	total
Normal	17	15	9	41
PosteriorReversible Encephalopathy Syndrome	1	4	-	5
Multiple Embolic Infarcts With Haemorrhagic Transformation	-	-	1	1
Intracerebral Hemmorrhage	-	1	1	2
Infarct In Occipital Lobe	-	1	1	2
Subarachnoid Haemorrhage	-	-	1	1
Infarct In Basal Ganglia	-	-	4	4
Intracranial Haemorrhage With Interventricular Haemorrhage	-	-	1	1
Basal Ganglia Calcification	1	-		1
Acute Infarct With Haemorrhagic Transformation In Right Frontoparietal Lobe And Insula	-	-	1	1
CerebralVein Thrombosis	-	-	1	1

## DISCUSSION

The eldest patient recorded is 38 years old, and the youngest is 18 years old. The age range of 25 to 30 years old had the greatest number of patients. Zibaenazhad MJ et al<sup>7</sup> reported that the occurrence of hypertension problems during pregnancy is significantly influenced by age. The patients' average age was 25.26 years. According to Prabhu T and Bai R<sup>8</sup>, the study's presentation mean was 22 years old. The mean mother age at diagnosis was 28 years old, according to Bashiri A et al<sup>9</sup>'s study on the maternal and newborn prognosis following cerebrovascular accidents during pregnancy. These studies' stated

mean ages are very similar to the mean age found in our investigation.

The majority of patients (58%) and post-natal patients were included in our study. The mean gestational age of the prenatal patients was 34.14 weeks.

The mean gestational age for singleton pregnancies was found by Bashiri A et al. (2009) to be 35.7 weeks, while for twin pregnancies it was 34 weeks.

According to Srinivasan K<sup>10</sup>, puberty-related CVT is roughly 10–12 times more common in India than it is in Western nations. The coexisting severe anaemia and the regional practise of fluid restriction throughout puberty could be the causes of the high frequency in Asian countries. Six The postpartum

period is associated with an increased risk of stroke, according to numerous research.1–13

28 instances (56% of the patients in this study) had seizures as their primary presenting condition. Other common presentations were blurred vision in 14 cases (28%), severe headaches in 3 cases (6%), unconsciousness in 2 cases (4%) and sudden loss of vision. Two cases each had altered sensorium and weakness on the left side of the body. Seizures last an average of 1.32 days, impaired vision 1.35 days, and excruciating headaches 1.6 days. The duration of the altered sensorium, sudden loss of eyesight, weakness on the left side of the body, and unconsciousness is one day.

According to Prabhu T and Bai R's investigation, seizures were the most often occurring presenting symptom, occurring in 24 instances (92%). Before the stroke, two subjects (7.7%) had an intense headache. Three instances (11.5%) had a fever with a high temperature, and one of them had intrapartum sepsis. The altered sensorium of 17 individuals ranged in stage from semiconsciousness to unconsciousness. Three patients exhibited psychotic symptoms at first. In 20 cases, hemiparesis/hemiplegia was observed (76.9%).

Our results can be interpreted through a number of underlying mechanisms. It has been suggested that exposure to an unfavourable intrauterine environment during pregnancy is linked to a number of cardiovascular consequences in the future. The early stages of pregnancy may be negatively impacted by 14–16 HDP. This could result in an ischemic and hypoxic environment for foetal development from the first trimester and an overexpression of antiangiogenic factors from the second trimester, which would inhibit placental and vascular endothelial growth. In rats, intrauterine hypoxia and placental ischemia would cause ventricular and cardiac hypoplasia, epicardial separation, and decreased metabolism. By causing detrimental structural and functional changes to the cardiovascular system in both foetal and postfetal life, these aberrant intrauterine environmental variables would have an impact on cardiac development later in life. Adverse anatomical and functional alterations, such as systemic vascular dysfunction, lower measures of microvascular function, and smaller hearts from infancy, have been discovered in the heart and blood arteries of infants born to mothers with preeclampsia. The link between HDP and CVD in kids may also be explained by damaged DNA and epigenetic modifications, an overactive sympathetic nervous system, shared genetic and environmental traits, and lifestyle choices. Severe preeclampsia has been shown in one study to be an independent risk factor for cardiovascular morbidity in offspring. It was proposed that placentas in the early preeclampsia groups had an increased risk of infarction and that placental gene expression differed between severe early-onset and late-onset preeclampsia.17–18 We also found that children of women with severe and

early-onset preeclampsia had an increased risk of cardiovascular disease (CVD).

## CONCLUSION

In this study, hypertension has emerged as an important risk factor for the occurrence of cerebrovascular disease; therefore, attention should be focused on rapid control of hypertension and maintaining normotension in the peripartum period. In a country like ours, it is also important to correct anemia and avoid dehydration in the peripartum period. There should be greater awareness and liberal use of CT/MRI in those cases who presented with seizures in the pregnancy/puerperium, and in those cases presenting with loss of consciousness. Early initiation of anticoagulant therapy can prevent further progression of thrombus and infarcts and this will considerably reduce the morbidity and mortality.

## REFERENCES

1. Zak IT, Dulai HS, Kish KK. Imaging of Neurologic Disorders Associated with Pregnancy and the Postpartum Period. *RadioGraphics*. 2007;27(1):95-108.
2. Waddy S and Barney J. Cerebrovascular Disease in Pregnancy. *Current Treatment Options in Cardiovascular Medicine* 2003;5:241–9.
3. Leffert LR, Clancy CR, Bateman BT, Bryant AS, Kuklina EV. Hypertensive Disorders and Pregnancy-related Stroke. *Obstet Gynecol* 2015;125:124-31.
4. Maino A, Siegerink B, Algra A, Martinelli I, Peyvandi F, Rosendaal FR. Pregnancy loss and risk of ischaemic stroke and myocardial infarction. *Br J Haematol* 2016;174:302-9.
5. Too G, Wen T, Boehme AK, Miller EC, Leffert LR, Attenello FJ, Mack WJ, D'Alton ME, Friedman A M. Timing and Risk Factors of Postpartum Stroke. *Obstet Gynecol* 2018;131:70-8.
6. Hammer ES, Cipolla MJ. Cerebrovascular Dysfunction in Preeclamptic Pregnancies. *Curr Hypertens Rep* 2015;17:64.
7. Zibaenazhad MJ, M Ghodsi P Arab, Gholzom N. the prevalence of hypertensive disorders of pregnancy in Shiraz, Southern Iran; Iranian Cardiovascular Research Journal. 2010;4:169-72.
8. Prabhu T and Bai R. Cerebrovascular Complications in Pregnancy and Puerperium. *J Obstet and Gynaecol India*. 2013;63(2):108-11.
9. Bashiri A, Lazer T, Burstein E, Smolin A, Lazer S, Perry ZH, Mazor M. Maternal and neonatal outcome following cerebrovascular accidents during pregnancy. *J Matern Fetal Neonatal Med*. 2007 Mar;20(3):241-7.
10. Srinivasan K. Cerebral venous and arterial thrombosis in pregnancy and puerperium. A study of 135 patients. *Angiol Vasc Dis*. 1983;34:731–46.
11. Simolke GA, Cox SM, Cunningham FG. Cerebrovascular accidents complicating pregnancy and the puerperium. *Obstet Gynecol*. 1991;78(1):37-42.
12. Fraser A, Nelson SM, Macdonald-Wallis C, Cherry L, Butler E, Sattar N, et al. Associations of pregnancy complications with calculated cardiovascular disease risk and cardiovascular risk factors in middle age: the Avon Longitudinal Study of Parents and Children. *Circu*

- lation.2012;125:1367-80.
13. Bellamy L, Casas JP, Hingorani AD, Williams DJ. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. *BMJ*. 2007; 335(7627):974.
14. Barker DJ. Fetal origins of coronary heart disease. *BMJ*. 1995;311(6998):171-4. Epub 1995/07/15. pmid:7613432; PubMed Central PMCID: PMC2550226.
15. Barker DJ. In utero programming of cardiovascular disease. *Thromb Haemostasis*. 2000;53(2):555-74. Epub 2000/03/29. pmid:10735050.
16. Barker DJ, Bagby SP, Hanson MA. Mechanisms of disease: in utero programming in the pathogenesis of hypertension. *Nat Clin Pract Nephrol*. 2006;2(12):700-7. Epub 2006/11/25. pmid:17124527.
17. van der Merwe JL, Hall DR, Wright C, Schubert P, Grove D. Are early and late preeclampsia distinct subclasses of the disease—what does the placenta reveal? *Hypertens Pregnancy*. 2010;29(4):457-67. Epub 2010/08/13. pmid:20701467.
18. Nevalainen J, Skarp S, Savolainen ER, Ryyanen M, Jarvenpaa J. Intrauterine growth restriction and placental gene expression in severe preeclampsia, comparing early-onset and late-onset forms. *J Perinat Med*. 2017;45(7):869-77. Epub 2017/06/09. pmid:28593875.