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Original Research Article

Preeclampsia and its Association with Pulmonary Edema: An Analytical Study

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Abstract:

Background: Although the reasons for pulmonary edema in preeclamptic females are mostly unclear, this condition can occur. Respiratory edema is among the most dangerous side effects. It is a sign that a pregnancy should be terminated immediately. This phenomenon is still poorly understood, despite the fact that several ideas, such as pulmonary capillary leakage, left ventricular failure, and hypervolaemia, have been put out to explain it. However following proper care, the prognosis is usually favourable, and the symptoms go away entirely in a few days following birth. The purpose of the current investigation is to determine if pre-eclampsia and pulmonary edema is related, and to provide an analysis based on the study's findings.

Methods: The hospital carried out a case-control study of preeclamptic women for a year, beginning from April 1, 2022, and ending on March 31, 2023. The patients (n = 28) were preeclamptic women who, while hospitalized for the index birth, experienced pulmonary edema on a plain X-ray or chest CT scan. The 64 patients in the control group had preeclampsia but were not diagnosed with heart failure or pulmonary edema during their index stay for delivery. Each woman's data was used to determine the research variables via electronic medical record and paper chart. A final set of significant predictors was selected via backward elimination and multivariable logistic regression.

Results: Roughly prior to birth, pulmonary edema occurred in 50% of all patients. The usage of magnesium sulphate and a fall of A platelet count of 10 × 109 /L or a rise in peak serum uric acid content of 10 μmol/L were substantially linked to pulmonary edema. Pulmonary edema incidence was shown to be decreased in cases of multiparity and with every increased intravenous crystalloid by 500 mL dosage.

Conclusion: The study found a number of early risk factors for pulmonary edema in preeclamptic women. To learn more about how these and other variables affect the likelihood that pulmonary edema may occur in preeclamptic women, more research is required.

Keywords: Preeclampsia, Pulmonary Edema, Preeclamptic Women, Maternal Hypertension During Pregnancy.

Introduction

A multi-systemic illness known as preeclampsia affects pregnant women and is typified by proteinuria and hypertension that appears after 20 weeks of gestation.[1] Although the exact cause of preeclampsia is unknown, aberrant placentation is considered to have a role.

In wealthy countries, preeclampsia-related deaths have significantly declined in recent decades, but rates of maternal and fetal morbidity nonetheless remain high.[2] Pulmonary edema is a frequent yet underappreciated consequence that affects women with preeclampsia. Although the cause of pulmonary edema in certain women is unclear, it has significant consequences for care and prognosis in these women. This susceptibility to pulmonary edema may be explained by circulating angiogenic agents, reduced malfunction of endothelial cells, elevated intravascular pressures accompanied by a greater cardiac afterload, or colloid oncotic pressure.[3] In this investigation, we looked at the relationship between preeclampsia and pulmonary edema in females.

Methods

Case-control research was carried out on women who were diagnosed with preeclampsia during a

birth index hospital stay at Muzaffarnagar Medical College and Hospital, Department of Obstetrics and Gynaecology, between April 1, 2022, and March 31, 2023. The department has a NICU, a mother ICU, and a coronary care unit for both high- and low-risk females births. We looked for all women who gave birth to a live or stillborn child after 20 weeks of pregnancy and had preeclampsia. Preeclampsia was defined as starting to experience elevated blood pressure while pregnancy (typically a blood pressure reading of > 140 mmHg at the systolic and/or 90 mmHg at the diastolic points) and > 300 mg in a 24-hour urine sample or ≥ 1+ on a dipstick for proteinuria.[4]

Prospective occurrences included all the women diagnosed with heart failure or pulmonary edema at the same stay. Out of all potential instances, women with pulmonary edema on a simple chest X-ray or a chest CT scan obtained during the first hospital stay for childbirth were the actual cases, according to radiological records for each occasion. Neither the control group nor any of the patients had ever experienced cardiac problems before. Whenever feasible, women at Muzaffarnagar Medical College and Hospital who were diagnosed with ICD-10 preeclampsia during the index delivery hospitalization and who were not diagnosed with heart failure or pulmonary edema were designated as the control subjects for each study.

Study characteristics were documented based on each woman's medical record and interactions with other people during her index hospitalization for childbirth. Demographics, Included were pre-pregnancy weight and height, previous medical history, and usage of an antihypertensive drug during the present pregnancy. There was also information on the number of foetuses, the method of birth, the gestational age at delivery, and if the mother had taken corticosteroids to mature the fetal lungs or magnesium sulphate to prevent premature fetal growth.

The preeclampsia episode was characterized by several details, including the gestational age when high blood pressure began, the highest systolic and diastolic blood pressure recorded during the hospital stay, the level of protein in the urine, and laboratory findings indicating the lowest levels of haemoglobin and platelets, as well as the highest levels of alanine aminotransferase, aspartate transaminase, and uric acid in the blood. The transthoracic echocardiogram recordings were examined to categorize the pumping activity of the left ventricle. A left ventricular ejection fraction below 50% was a sign of left ventricular failure. [5] We also endeavoured to evaluate the cumulative quantity of intravenous fluids supplied throughout the 48-hour period prior to birth, as well as the quantity of blood products received. For some of our research variables, there was some missing data, which might lead to bias. Therefore, missing values were multiplicatively imputed based on observed patient features. To arrive at Except for the delivery method, which was compelled into the final model, all variables not chosen via 50% or more of the imputed datasets had backward deletion, eliminated from the final set of predictors.

For each variable in the final multivariate logistic regression model, chances ratios and 95% confidence intervals were found for each imputed dataset to account for heterogeneity both between and within imputation. “Thereafter, the SAS MIANALZE function was used to merge them. Furthermore, a comprehensive case study of the final model (n = 76) was conducted. Rerunning the primary model, the comparison between the control participants and the cases was focused on whether the pulmonary edema began before to or following delivery. The tests were done with SAS version 9.3 from SAS Institute Inc. in Cary, NC. The study might be conducted with approval from the research ethics boards.”

Results

The research group comprised 92 preeclamptic women, of which 28 were cases with pulmonary edema and 64 were control subjects without pulmonary edema (Table 1). 10.7% of cases compared to 48.4% of control individuals said they had previously given birth. The rates of Caesarean section were 75.0% vs. 54.7% in the current pregnancy, and the Persistent hypertension rates were 17.9% as opposed to 21.9%. Compared to the control individuals, the cases gave birth to their kids on average more than five weeks earlier (Table 1). About half of the patients had pulmonary edema before giving delivery. The majority of patients (64.3%) with a chest X-ray showed evidence of pulmonary edema, with a left ventricular ejection fraction of less than 50%.

Using the backward elimination method, twelve of the original factors were picked out and added to the final set, along with the delivery method (Table 2). For pulmonary edema, the use of antihypertensive medications throughout the delivery index hospitalization did not become substantial. “Pulmonary edema was shown to be highly associated using magnesium sulfate to safeguard fetuses neurologically or to avoid eclampsia, as well as with reductions of the minimum platelet count by 10 × 109/L and increases of the peak level of uric acid in the serum by 10 μmol/L.

Pulmonary edema risk was shown to be lower in multiparous people. While obtaining 500 mL more receiving any blood product was not linked to the progression of lung edema, and intravenous crystalloids was also linked to a decreased chance

of doing so (Table 2). The lowest peak concentration of uric acid and platelet count were the only significant markers linked to the onset of pulmonary edema, after the patients were grouped depending on when the edema first emerged. [6]

Table 1: Features of control individuals (preeclamptic women who did not have pulmonary swelling) and cases (preeclamptic women who had peripartum pulmonary edema) Unless otherwise noted, data are displayed as a number (%)

Characteristic	Cases (n = 28)	Control subjects(n = 64)
Mean (SD) age, years	32.2 (6.1)	32.4 (6.3)
Mean (SD) body mass index, kg/m2	31.4 (7.4)	33.6 (8.1)
Religion		
Muslims	12 (42.9)	39 (60.9)
Hindus	7 (25.0)	22 (34.4)
Others	9 (32.1)	3 (4.7)
Smoked tobacco during pregnancy		
Yes	1 (3.6)	1 (1.6)
No	24 (85.7)	62 (96.9)
Unknown	3 (10.7)	1 (1.6)
Pre-pregnancy chronic hypertension	5 (17.9)	14 (21.9)
Hypertension in a previous pregnancy		
Present	1 (3.6)	16 (25.0)
Absent	7 (25.0)	26 (40.6)
Unknown	3 (10.7)	1 (1.6)
Not applicable	17 (60.7)	21 (32.8)
Multifetal pregnancy	4 (14.3)	7 (11.0)
Caesarean section	21 (75.0)	35 (54.7)
Mean (SD) gestational age at delivery, weeks	30.3 (5.6)	35.2 (4.5)
Antihypertensive medication use at index delivery admission	11 (39.3)	28 (43.8)
Mean (SD) peak systolic blood pressure within 24 hours of the indexdelivery admission, mmHg	167.8 (23.3)	161.5 (14.7)
Mean (SD) crystalloid intravenous fluids received, mL	1987 (971)	2418 (950)
Received any blood product transfusion	5 (17.9)	5 (7.8)
Median (IQR) peak serum alanine aminotransferase concentration, U/L	41.0 (84.5)	22.5 (35.0)
Median (IQR) peak serum aspartate transaminase concentration, U/L	46.0 (60.0)	29.0 (38.0)
Mean (SD) minimum blood hemoglobin concentration, g/L	85.3 (19.0)	103.2 (16.7)
Mean (SD) minimum blood platelet concentration, × 10^9/L	137 (69.2)	175.9 (53.1)
Mean (SD) peak serum uric acid concentration, µmol/L	496.5 (154.5)	396.9 (109.2)
Received intravenous magnesium sulphate	20 (71.4)	20 (31.3)
Mean (SD) urine output (mL/24 hours)	1894 (1094)	2040 (962)
Method(s) to confirm pulmonary edema		
Chest X-ray	27 (96.4)	Not applicable
CT scan	10 (35.7)	Not applicable
Left-ventricular dysfunction on echocardiography*		
Present	18 (64.3)	Not applicable
Absent	4 (14.3)	Not applicable
Not performed	6 (21.4)	Not applicable
*Defined as left ventricular ejection fraction < 50%.		

Table 2: Adjusted risk of pulmonary edema among women with preeclampsia, in relation to factors during theindex delivery hospitalization (28 cases and 64 control subjects)

Variable determined in the index delivery hospitalization	Unit change or comparison of the variable	aOR (95%CI)
Maternal age, years	5-year increase	1.96 (0.82 to 4.69)
Parity	≥ 1 vs. 0	0.03 (0.004 to 0.29)
Antihypertensive medication uses at the time of index delivery admission	Not using vs. using	11.06 (0.99 to 123.54)

Pre-pregnancy chronic hypertension	Present vs. absent	7.25 (0.40 to 131.68)
Mode of delivery	Caesarean section vs. vaginal delivery	2.24 (0.22 to 23.03)
Gestational age at preeclampsia onset	1-week earlier onset	1.07 (0.88 to 1.30)
Peak serum alanine aminotransferase concentration	10 U/L increase	0.98 (0.96 to 1.00)
Minimum blood hemoglobin concentration	10 g/L decrease	1.60 (0.87 to 2.94)
Minimum blood platelet count	10×10^9/L decrease	1.32 (1.06 to 1.65)
Peak serum uric acid concentration	10 µmol/L increase	1.19 (1.06 to 1.34)
Received intravenous magnesium sulphate	Received vs. did not receive	10.42 (1.39 to 78.22)
Received crystalloid intravenous fluids at the time of delivery	500 mL increase	0.60 (0.37 to 0.98)
Received any blood product transfusion	Received vs. did not receive	0.16 (0.01 to 4.02)
Odds ratios were derived using multivariable logistic regression analysis, including all listed variables.		

Discussion

We found a correlation between indicators of women who already have preeclampsia are more likely to get lung edema, have a lower platelet count, and have a higher blood uric acid level.

It has been shown that multiparity lowers the risk of pulmonary edema. Compared to those without pulmonary edema, those with it got a lower intravenous crystalloid volume during delivery. In around half of the cases, the pulmonary edema existed prior to delivery. Preeclampsia is almost three times more common in nulliparous women than in non-nullipendent women, with a 2.4 times higher risk of developing in the former.[7] Multiparity was linked to a reduced incidence of pulmonary edema in our research. The likelihood of getting pulmonary edema increased by more than ten times following intravenous magnesium sulfate administration. The usage of magnesium sulfate may be a sign of a more severe case of preeclampsia because to avoid eclamptic seizures, it is given to women with mild to severe preeclampsia convulsions. Even when magnesium sulfate solution is given constantly throughout the day, the total amount infused is not very large.[8] As a result, rather than contributing till the pulmonary edema started, the use of magnesium sulfate is most likely a marker of the degree and timing of preeclampsia. [10]

We also found that for every 500 mL increase in intravenous crystalloid dosage, the risk of preeclampsia fell by 0.60. This might be partly because most instances of pulmonary edema (almost 50%) happened before birth, thereby limiting the quantity of intravenous fluids that could be given. Despite our findings, clinical practice recommendations rationally advise limiting the rate of intravenous fluid infusion up to 75 mL per hour; in preeclamptic females to prevent increasing the risk of pulmonary edema.[9] This recommendation also includes refraining from treating oliguria with extra intravenous fluid. [11] The chances ratio of developing pulmonary edema was found to be 1.32 for every 10 × 109/L drop in the lowest number of platelets in the blood. This might be explained by the fact that microvascular dysfunction and the severity of preeclampsia are both indicated by low platelet counts.[11] Endothelial injury-induced Temporary heart failure can be brought on by arterial vasospasm in the cardiac circulation. Our discovery that higher uric acid concentrations are linked to an increased risk of pulmonary edema may suggest that the severity of preeclampsia is mirrored in higher uric acid levels, which are also in women with preterm preeclampsia, there was a negative correlation shown with the length of expectant care.[12] Given that the majority of our instances are beyond of our control, patients had echocardiograms, we were unable to accurately compare the two groups' cardiac function. Additionally, there were no left ventricular function measurements taken before or during the pregnancy.

Therefore, we hypothesize that women who have preeclampsia with an early start may be more vulnerable to pulmonary edema. One of the study's limitations is that we were unable to differentiate; while looking for common This study aims to identify the specific Things that put people with preeclampsia at risk for lung edema, with a special focus on distinguishing how cardiogenic and non-cardiogenic lung edema are different. Echocardiography detected a certain level of impairment in the left ventricle's role in 64% of the remaining patients. six instances did not receive cardiac imaging. To confirm the diagnosis of pulmonary edema, X-ray imaging was performed on all patients; however, control participants were not checked in the same manner. Some research variables were reported via charts, although data abstraction was done using a conventional procedure. However, some factors, like the amount of urine produced or IV fluids given, could not have been precisely recorded in all charts, which could have prevented the observation of a real correlation with the beginning of pulmonary edema. The study's statistical ability to draw firm conclusions is limited by the little number of instances, especially in the study divided into days when pulmonary edema occurred development.

Conclusion

The knowledge of preeclampsia and its connection to pulmonary edema is provided by this case-control research. The impact of blood uric acid levels and the use of magnesium sulfate given intravenously when pulmonary edema develops requires more investigation. Finding the relationships between these factors and other recognized Indicators indicating the seriousness of preeclampsia, such as the date it started and the severity of blood pressure is also essential.

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