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Original Research

Assessment of risk factors for cardiopulmonary dysfunction in early-onset severe pre-eclampsia

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ABSTRACT:

Background: Pre-eclampsia is a pregnancy complication characterized by high blood pressure and signs of damage to another organ system, often the kidneys. The present study was conducted to assess risk factors for cardiopulmonary dysfunction in early-onset severe pre-eclampsia. **Materials & Methods:**78 patients with severe pre-eclampsia diagnosed at less than 34 weeks of pregnancywho received expectant management to prolong theirpregnancywere divided into 2 groups based on cardiopulmonaryfunction. Group I was patients with normal cardiopulmonary function and group II was patients with decompensatory cardiopulmonary function. The clinical characteristics of patients in the two groups were compared. **Results:** Fetal growth restriction was seen in 16 in group I and 22 in group II. 12 patients in group I and 15 in group II required intravenous antihypertensivemedications. The mean alanine aminotransferase level was 25.2IU/L in group I and 30.4IU/L in group II. The mean creatinine level was 57.3µmol/L in group I and 78.1µmol/L. Platelet count was 170.2cells/µL in group I and 165.2cells/µL. The mean proteinuria was 5.2g/24 h in group I and 8.4g/24 h in group II. The mean plasma albumin was 27.4g/L in group I and 25.3g/L in group II. The difference was significant (P< 0.05). The serumcreatinine (P-0.04), increased proteinuria(P-0.05), and ascites (P- 0.01) were factors that demonstrated associations with decompensation of cardiopulmonary function. **Conclusion:** Ascites and renal insufficiency were linked to cardiopulmonary dysfunction. More medical attention should be given to ascites while managing early-onset severe pre-eclampsia.

Keywords: Ascites, renal insufficiency, cardiopulmonary dysfunction

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INTRODUCTION

Pre-eclampsia is a pregnancy complication characterized by high blood pressure and signs of damage to another organ system, often the kidneys. This condition usually develops after 20 weeks of pregnancy and can lead to serious complications for both the mother and the baby if left untreated.[1]The exact cause of pre-eclampsia is still not fully understood, but it is believed to involve problems with the placenta, the organ that nourishes the fetus during pregnancy. Risk factors for developing pre-eclampsia include having a history of pre-eclampsia, being pregnant with multiples, being obese, being a teenager or over 40, having certain medical conditions like diabetes or hypertension, and having a first pregnancy.[2]

Pre-eclampsia can lead to serious complications such as eclampsia, which involves seizures, as well as HELLP syndrome, which involves liver and blood clotting problems. The only way to cure pre-eclampsia is to deliver the baby, which may be done early if the condition is severe enough. Otherwise, treatment focuses on managing symptoms and monitoring both the mother and the baby closely until delivery can be safely achieved.[3]When compared to late-onset pre-eclampsia, cardiovascular and pulmonary problems are more likely to cause unfavorable maternal and perinatal outcomes and are also more severe in early-onset pre-eclampsia.[4] Moreover, there is a correlation between stage B heart failure (asymptomatic left ventricular dysfunction/hypertrophy) and preeclampsia. According to clinical research, pre-eclampsia is linked

to short-term cardiovascular hazards that are consistently observed.[5] These hazards, particularly in the event of early-onset pre-eclampsia, may become apparent several years after birth.It is imperative that healthcare professionals make sure expectant patients do not experience any difficulties during their care.[6]The present study was conducted to assess risk factors for cardiopulmonary dysfunction in early-onsetsevere pre-eclampsia.

MATERIALS & METHODS

The present study consisted of 78 patients with severe pre-eclampsia diagnosed at less than 34 weeks of pregnancywho received expectant management to prolong theirpregnancy. All gave their written consent to participate in the study.

Data such as name, age, etc. was recorded. All patients received expectant management. Prophylactic magnesium sulfate was administered during the first 5 days of expectant management, intrapartum, and for 3 days postpartum. All patients received dexamethasone upon hospital admission. Maternal monitoring included blood pressure measurements every 4 hours, a clinical evaluation of symptoms at least twice daily, and a 24-hour urine analysis twice weekly. Patients were divided into 2 groups based on cardiopulmonary function. Group I was patients with normal cardiopulmonary function and group II was patients with decompensatory cardiopulmonary function. The clinical characteristics of patients in the two groups were compared. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Assessment of parameters

Parameters	Group I (40)	Group II (38)	P value
Fetal growth restriction	16	22	0.81
Required intravenous antihypertensive medications	12	15	0.92
Alanine aminotransferase, IU/L	25.2	30.4	0.71
Creatinine, µmol/L	57.3	78.1	0.04
Platelet count, cells/µL	170.2	165.2	0.89
Proteinuria, g/24 h	5.2	8.4	0.01
Plasma albumin, g/L	27.4	25.3	0.26
Ascites	8	27	0.01

Table I, graph I shows that fetal growth restriction was seen in 16 in group I and 22 in group II. 12 patients in group I and 15 in group II required intravenous antihypertensive medications. The mean alanine aminotransferase level was 25.2IU/Lin group I and 30.4IU/L in group II. The mean creatinine level was 57.3µmol/Lin group I and 78.1µmol/L. Platelet count was 170.2cells/µL in group I and 165.2cells/µL. The mean proteinuria was 5.2g/24 hin group I and 8.4g/24 h in group II. The mean plasma albumin was 27.4g/Lin group I and 25.3g/L in group II. Ascites was seen in 8 in group I and 27 in group II. The difference was significant (P< 0.05).

Graph I Assessment of parameters

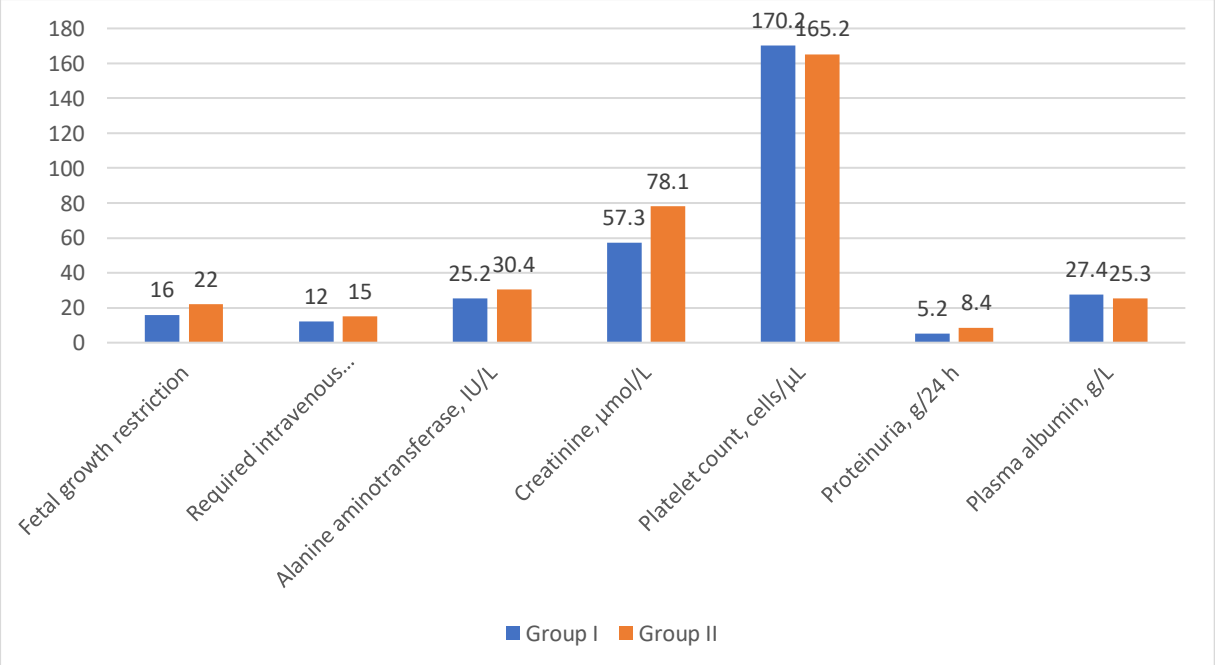

Table II Correlations between cardiopulmonary function and patient characteristics

Variables	Odd ratio	P value
Fetal growth restriction	1.5	0.72
Required intravenous antihypertensive medications	1.8	0.45
Alanine aminotransferase, IU/L	25.3	0.17
Creatinine, μmol/L	0.19	0.04
Thrombocytopenia	10.7	0.12
Proteinuria, g/24 h	0.62	0.05
Hypoproteinemia	0.12	0.16
Ascites	12.4	0.01

Table II shows that the serumcreatinine (P-0.04), increased proteinuria(P-0.05), and ascites (P- 0.01) were factors that demonstrated associationswith decompensation of cardiopulmonary function.

DISCUSSION

Cardiopulmonary dysfunction in early-onset severe pre-eclampsia can have serious consequences for both the mother and the fetus. Several risk factors contribute to the development of cardiopulmonary complications in this condition.[7] Early-onset severe pre-eclampsia, which occurs before 34 weeks of gestation, is associated with a higher risk of cardiopulmonary dysfunction compared to late-onset pre-eclampsia.[8]The severity of pre-eclampsia, including the degree of hypertension, proteinuria, and other associated symptoms, can influence the likelihood of developing cardiopulmonary complications.[9]Early-onset severe pre-eclampsia often involves multi-organ dysfunction, including renal, hepatic, and hematological dysfunction, which can contribute to cardiopulmonary complications.[10]The present study was conducted to assess risk factors for cardiopulmonary dysfunction in early-onset severe pre-eclampsia.

We found that fetal growth restriction was seen in 16 in group I and 22 in group II. 12 patients in group I and 15 in group II required intravenous antihypertensive medications. The mean alanine aminotransferase level was 25.2IU/L in group I and 30.4IU/L in group II. The mean creatinine level was 57.3μmol/L in group I and 78.1μmol/L. Platelet count was 170.2cells/μL in group I and 165.2cells/μL. The mean proteinuria was 5.2g/24 h in group I and 8.4g/24 h in group II. The mean plasma albumin was 27.4g/L in group I and 25.3g/L in group II. Guan et al[11]explored associations between patient characteristics and cardiopulmonary function among patients with early-onset severe pre-eclampsia being treated with expectant management. Patients were divided into two groups based on cardiopulmonary function, a decompensatory group and a normal group. Serum creatinine levels (P = 0.017), ascites (P = 0.001), and increased proteinuria (P = 0.015) were associated with decompensation of cardiopulmonary function during early-onset severe pre-eclampsia. Hypoproteinemia was associated with significantly increased odds of ascites occurring (odds ratio 3.16; 95% confidence interval 1.34–7.44) and the mean serum albumin level was higher in patients without ascites (P=0.001).

We observed that the serumcreatinine (P-0.04), increased proteinuria(P-0.05), and ascites (P- 0.01) were factors that demonstrated associationswith decompensation of cardiopulmonary function. Woods et al[12]reviewed the medical records of 190 patients and noted the presence or absence of large-volume maternal ascites, peripartum complications, laboratory data, and specific operative techniques.The incidence of large-volume ascites in patients with HELLP syndrome who underwent abdominal delivery was approximately 10% in classes 1, 2, and 3. Compared with HELLP syndrome patients without ascites, those with HELLP-associated ascites at surgery had a significant sixfold increase in the incidence of congestive heart failure and a ninefold increase in the incidence of adult respiratory distress syndrome, both of which usually became clinically apparent within 24 hours postpartum. Those HELLP syndrome patients without ascites at surgery developed congestive heart failure or adult respiratory distress syndrome infrequently, and more than 24 hours postoperatively. The limitation of the study is the small sample size.

CONCLUSION

Authors found that ascites and renal insufficiency were linked to cardiopulmonary dysfunction. More medical attention should be given to ascites while managing early-onset severe pre-eclampsia.

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