PREVALENCE, CLINICAL PRESENTATION, DIAGNOSIS AND TREATMENT OF ACUTE PULMONARY OEDEMA IN SEVERE PREGNANCY-INDUCED HYPERTENSION AND ECLAMPSIA CASES IN TRIBAL POPULATION OF SOUTH RAJASTHAN

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ABSTRACT

BACKGROUND

Pulmonary oedema in severe pregnancy-induced hypertension is a life threatening complication with high maternal mortality, particularly in tribal population of South Rajasthan.

METHODS

Thirteen cases which occurred in the duration of two and half years were analysed through medical records and findings were recorded.

RESULTS

Maximum cases 10(76.92%) were in less than 20 years of age. 12 (92.30%) cases were nulliparous. Out of 13 cases of PIH, pulmonary oedema developed in 5 (38.46%) cases of eclampsia and 8 (61.54%) cases of severe pregnancy-induced hypertension. 10 (76.92%)cases were 28 to 30 weeks of gestation and 3 (23.08%) were 31 to 34 weeks of gestation. 8 (61.54%) cases were severely anaemic. 12 (92.30%) were unbooked cases.

CONCLUSION

Regular antenatal checkups, early diagnosis, prompt treatment of hypertension and pulmonary oedema and termination of pregnancy is required to prevent maternal death.

KEYWORDS

Pulmonary Oedema, Eclampsia, PIH, Pregnancy-Induced Hypertension, Hypertension in Pregnancy, Maternal Mortality Causes.

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INTRODUCTION: Prevalence of acute pulmonary oedema in pregnancy varies from as low as 0.08% to as high as 0.5%.[1] Pre-eclampsia remains an important cause of acute pulmonary oedema in pregnancy. Acute pulmonary oedema is a significant cause of morbidity and mortality in pregnant women and represents a form of decompensated acute cardiac failure.[2] It is characterised by symptoms of sudden-onset of breathlessness, orthopnoea, agitation and cough and signs like tachycardia, tachypnoea, crackles and wheeze on chest auscultation, cardiac S_3 gallop rhythm and murmurs and decreased oxygen saturation. Typical chest X-ray features include upper lobe redistribution, Karley-B lines and pulmonary infiltrates.

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Arterial blood gases (decreased P_aO_2), ECG and echocardiography may help establish the diagnosis. Preventive strategies include close clinical monitoring and restricted fluid administration. Immediate management of acute pulmonary oedema includes oxygenation, ventilation and circulation control with vasodilators.[3] Hypertensive disease of pregnancy affects approximately 15% of pregnant women.[4] Pulmonary oedema may occur in up to approximately 3% of women with pre-eclampsia, with 70% of cases occurring after birth. It is associated with excessive fluid administration and disease severity, including the presence of haemolysis, elevated liver enzymes and low platelets (HELLP), and eclampsia.[5] In addition to the usual management goals of stabilising the woman and treating the acute pulmonary oedema, consideration needs to be given to delivery of the foetus if acute pulmonary oedema occurs in the antenatal period. The underlying mechanism for the hypertension in this disease state remains unknown. Urgent reduction of critically high blood pressure with an intravenous antihypertensive agent is necessary. Nitroglycerin (glyceryl trinitrate) is recommended as the

drug of choice in pre-eclampsia associated with pulmonary oedema.[6]

It is given by intravenous infusion starting at 5 µg per min, gradually increasing every 3–5 min. to a maximum of 100 µg per min. Nitroglycerin can also be administered by sublingual spray (400 µg, 1–2 puffs every 5–10 min.). An alternative agent, sodium nitroprusside, is recommended in severe heart failure and critical hypertension; however, it should be used only with caution.[7] Reduction in systolic and diastolic blood pressure should occur at a rate of approximately 30 mmHg over 3–5 min. approximately to 140/90 mmHg. Intravenous furosemide (bolus 20–40 mg over 2 min.) is used to promote vasodilation and diuresis, with repeated doses of 40–60 mg after approximately 30 min. if there is an inadequate diuretic response (maximum dose 120 mg.)[8] If hypertension persists despite the combination of nitroglycerin or sodium nitroprusside and furosemide, then a calcium channel antagonist such as nicardipine or nifedipine may be considered. Labetalol, Prazosin as well as hydralazine can be used for control of high blood pressure.[9] Intravenous morphine 2–3 mg may also be given as a vasodilator and anxiolytic.[2]

MATERIAL AND METHODS: This being a retrospective study, the old records were reviewed by the authors for maternal age, parity, education, socioeconomic status and period of gestation. pre-existing maternal disease, obstetric complications, drug and medication use, tobacco use, maternal symptoms, physical findings, laboratory studies, imaging studies, fluid balance, tocolytic use and type, evidence of pre-eclampsia, infection, and therapeutic measures were also recorded. The timing of pulmonary oedema in pregnancy and gestation period and outcomes were recorded. The probable cause of the pulmonary oedema was determined by the authors based on history, physical examinations, and laboratory and radiologic findings. In a few cases, more than one attributable cause was possible, and in those cases, the most likely diagnosis was chosen. Pregnancy outcomes included gestational age at delivery, birth weight, mode of delivery, and Apgar scores. Applied therapies such as supplemental oxygen, need for ventilator support, fluid restriction, diuretic usage, cessation of tocolytics, antihypertensive use, termination of pregnancy, and anti-arrhythmic usage were recorded.

OBSERVATIONS AND RESULTS:

Age in Years	No. of Patients	Percentage
Less than 20	10	76.92
21- 25	2	15.38
26-30	1	7.70
31 and above	0	0.00
	13	100
Table 1: Age Distribution		

Maximum cases 10(76.92%) were in less than 20 years of age.

Parity	No. of Patients	Percentage
nulliparous	12	92.30
Para 1	1	7.70
Para 2	0	0.00
Para 3 and above	0	0.00
	13	100
Table 2: Parity		

12(92.30%) cases were nulliparous.

Associated Factors	No. of Patients	Percentage
Severe PIH	8	61.54
eclampsia	5	38.46
Gestational period 28- 30 weeks 31-34 weeks 35 weeks and above	 10 3 0	 76.92 23.08 0.00
Severe anaemia (6 g% and less)	8	61.54
Unbooked cases	12	92.30
Education less than 8th standard	12	92.30
Non-land owner	13	100.00
Table 3: Associated Factors		

Out of 13 cases of PIH, pulmonary oedema developed in 5(38.46%) cases of eclampsia and 8(61.54%) cases of severe pregnancy-induced hypertension. 10(76.92%) cases were 28 to 30 weeks of gestation and 3(23.08%) were 31 to 34 weeks of gestation. 8(61.54%) cases were severely anaemic. 12 (92.30%) were unbooked cases and had education less than eighth standard. All cases were landless belonging to lower socioeconomic status.

Clinical Presentation	No. of patients	Percentage
Catastrophic/unconscious/history of convulsions	5	38.46
Breathlessness, cough	12	92.30
Generalised anasarca	10	66.66
Disorientation, agitation	13	100
Tachycardia, tachypnoea	12	92.30
Diastolic Blood pressure more than 100 mm of Hg	13	100.00
Positive auscultation	13	100.00
Positive X-ray	12	92.00
Low Oxygen saturation (below 90%)	13	100.00
Table 4: Clinical Presentation		

5(38.46%) cases were brought to hospital with eclampsia in catastrophic or unconscious state or with history of convulsions at home. 12(92.30 5) cases presented with breathlessness cough, had tachycardia and tachypnoea and positive X rays. All cases were dis-oriented or agitated, had diastolic BP more than 100 mm of Hg, positive chest signs and low oxygen saturation.

Treatment	No. of Patients	Percentage
Standard Antihypertensive Lobate and nifedipine, diuretic furosemide, oxygen by mask, induction of labour, and vaginal delivery	8	61.54
Resuscitation, mag-sulf therapy, antihypertensives, diuretics, ventilator support, caesarean delivery	5	38.46
Outcome of foetus		
Still birth	3	23.08
Live birth	10	76.92

Table 5: Management

8(61.54%) cases were managed by antihypertensives, diuretics, oxygenation and termination of pregnancy. 5(38.46%) cases required resuscitation ventilator support antihypertensives, diuretics and termination of pregnancy by caesarean delivery. Outcome of foetus was 3(23.08%) still births and 10(76.92%) live births of preterm and extremely preterm new born babies.

DISCUSSION: In the present study, maximum cases 10(76.92%) were in less than 20 years of age. 12 (92.30%) cases were nulliparous. Out of 13 cases of PIH, pulmonary oedema developed in 5(38.46%) cases of eclampsia and 8(61.54%) cases of severe pregnancy-induced hypertension. 10(76.92%) cases were 28 to 30 weeks of gestation and 3(23.08%) were 31 to 34 weeks of gestation. 8(61.54%) cases were severely anaemic. 12(92.30%)were unbooked cases and had education less than eighth standard. All cases were landless belonging to lower socioeconomic status. 5 (38.46%) cases were brought to hospital with eclampsia in catastrophic or unconscious state or with history of convulsions at home. 12(92.305) cases presented with breathlessness, cough, had tachycardia and tachypnoea and positive x rays. All cases were disoriented or agitated, had diastolic BP more than 100 mmHg, positive chest signs and low oxygen saturation. 8(61.54%) cases were managed by antihypertensives, diuretics, oxygenation and termination of pregnancy. 5(38.46%) cases required resuscitation, ventilator support, antihypertensives, diuretics and termination of pregnancy by caesarean delivery. Outcome of foetus was 3(23.08%) still births and 10(76.92%) live births of preterm and extremely preterm new born babies.

Acute pulmonary oedema is the fourth most common cause of maternal mortality. It may occur during the antenatal, intrapartum or postpartum periods. Risk factors and predisposing conditions are Cardiovascular disease (hypertension, ischaemic heart disease, congenital heart disease, valvular heart disease, arrhythmias, cardiomyopathy), Obesity, Increased maternal age, Endocrine disorders (phaeochromocytoma and hyperthyroidism). Pre-eclampsia, Cardiomyopathy, Sepsis, Preterm labour, Amniotic fluid embolism, Pulmonary embolism, β-Adrenergic tocolytic agents, Corticosteroids, Magnesium sulphate, Illicit drugs including cocaine, Positive fluid balance > 2000 mL and Multiple gestation. The mean maternal age of those diagnosed with pulmonary oedema was 27.6±6.4 years, with a range of 18–42 and a mean gestational age at diagnosis of 31.5±6.8 weeks.[10] Fifty-one women (0.08%) were diagnosed with acute pulmonary oedema during pregnancy or in the postpartum period. The mean patient age at the time of diagnosis was 27.6±6.4 years.[11] The mean gestational age at the time of diagnosis was 31.5±6.8 weeks. The diagnosis of pulmonary oedema was made during the antepartum period in 24 patients (47%), the intrapartum period in seven (14%), and the postpartum period in 20(39%). The most common attributable causes were tocolytic use (13 patients [25.5%]), cardiac disease (13 patients [25.5%]), fluid overload (11 patients [21.5%]), and preeclampsia (nine patients [18%]). Those with fluid overload identified as the likely aetiology had a significantly greater mean positive fluid balance (6022±3340 mL).[12] All patients whose pulmonary oedema was secondary to tocolytic use received multiple simultaneous tocolytic agents; the most common combination was intravenous magnesium sulphate and subcutaneous terbutaline. Six of the 13 women with cardiac disease were found to have previously undiagnosed structural heart disease.

The most common causes of pulmonary oedema are the use of tocolytic agents, underlying cardiac disease, fluid overload, and preeclampsia. Preeclampsia was the cause of pulmonary oedema in 17.4% of patients in this report.[12] These patients are at an increased risk for the development of pulmonary oedema due to underlying endothelial damage and decreased colloid osmotic pressure, which cause leakage into the pulmonary interstitial or alveolar space. Combined with the left ventricular dysfunction and increase in peripheral vascular resistance found in preeclampsia patients, pulmonary oedema develops. The development of pulmonary oedema appears to be influenced by maternal age, parity, and pre-existing essential hypertension.[13] Two women in this report with chronic hypertension and superimposed preeclampsia developed pulmonary oedema.

CONCLUSION: Maternal mortality in pulmonary oedema in severe pregnancy-induced hypertension cases can be reduced and requires regular antenatal care, early diagnosis and prompt treatment of hypertension as well as pulmonary oedema by diuretics and ventilator support or oxygen therapy.

REFERENCES:

1. Motwani MM, Shah SS, Mehta AC. Pulmonary oedema in severe pre-eclampsia (a case report). J Postgrad Med 1989;35(3):183-185.
2. Sibai BM, Mabie BC, Harvey CJ, et al. Pulmonary oedema in severe preeclampsia- eclampsia: analysis of thirty-seven consecutive cases. Am J Obstet Gynecol 1987;156(5):1174-1179.
3. Dennis AT, Solnordal CB. Acute pulmonary oedema in pregnant women. Anesthesia 2012;67(6):646–659. DOI: 10.1111/j.1365-2044.2012.07055.
4. Benedetti TJ, Kates R, Milliams V. Hemodynamic observations in severe pre-eclampsia complicated by pulmonary oedema. Amer J Obstet & Gynaecol 1985;152(3):330-334.
5. Donnelly JF, Lock FR. Causes of death in five hundred thirty-three fatal cases of toxemia in pregnancy. Amer T Obstet & Gynaecol 1954;86:184-190.
6. Cunningham FG, Pritchard JA, Hankins D, et al. Peripartum heart failure: idiopathic cardiomyopathy or compounding cardiovascular events? Obstet & Gynaecol 1986;67(2):157-168.
7. Hankins GDV, Wendel GD, Cunningham FG, et al. Longitudinal evaluation of hemodynamic changes in eclampsia. Amer J Obstet & Gynaecol 1984;150(5 Pt 1):506-512.
8. Henderson DW, Vilos GA, Milne KJ, et al. The role of Swan-Ganz catheterization in severe pregnancy - induced hypertension. Amer J Obstet & Gynaecol 1984;148:570-574.
9. Phelan JP, Yurth DA. Severe preeclampsia Peripartum hemodynamic observations. Amer J Obstet & Gynaecol 1982;144:17-22.
10. Engelhardt T, MacLennan FM. Fluid management in pre-eclampsia. International Journal of Obstetric Anesthesia 1999;8(4):253-259.
11. Cantwell R, Clutton-Brock T, Cooper G, et al. Saving mothers' lives: reviewing maternal deaths to make motherhood safer: 2006-2008. The eighth report of the confidential enquiries into maternal deaths in the United Kingdom. British Journal of Obstetrics and Gynecology 2011;118(Suppl 1):1–203.
12. Dunne C, Meriano A. Acute postpartum pulmonary oedema in a 23-year-old woman 5 days after Cesarean delivery. Canadian Journal of Emergency Medicine 2009;11(2):178–181.
13. Sciscione AC, Ivester T, Largoza M, et al. Acute pulmonary oedema in pregnancy. Obstetrics and Gynecology 2003;101(3):511–515.