Journal of Clinical Research in Pain and Anaesthesia
ISSN: 2689-6141

Case Report *Volume 2 Issue 1*

Anaesthesia Management of Severe PIH Patient with IUFD in Acute Pulmonary Oedema for Normal Delivery Using Labour Analgesia - A Case Report

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Received Date: February 04, 2020; **Published Date:** February 12, 2020

Abstract

Pregnancy induced hypertension is a hypertensive disorder, which occurs in 5% to 7% of all pregnancies. Number of Stillbirths and IUFD in mothers due to severe preeclampsia is rising in developing countries. A 40 year-old Primi-gravida patient with severe PIH developed acute pulmonary oedema due to fluid overload and intrauterine fetal death at 29th week of gestational age delivered vaginally by using labour epidural anasthesia. She was monitored carefully inside OR and intensive care Unit with invasive lines and supported with NIV for 24 hours. She was managed by multi-disciplinary team with proper and clear management plan and discharged from the hospital on the 4th postpartum day in good general condition.

Keywords: Pregnancy induced hypertension; Stillbirths and IUFD; Pulmonary oedema; Fetal death

Abbreviations: IUFD: Intrauterine Fetal Demise; PIH: Pregnancy Induced Hypertension; DIC: Disseminated Intravascular Coagulopathy; NIV: Non-Invasive Ventilation; PCWP: Pulmonary Capillary Wedge Pressure; CVP: Central Venous Pressure; LV: Left Ventricular; HR: Heart Rate; BP: Blood Pressure; RR: Respiratory Rate.

Introduction

Acute pulmonary oedema in pregnant women is a life-threatening event. The common attributable causes being iatrogenic fluid overload (21.5%) and preeclampsia (18%). The Scottish Confidential Audit of Severe Maternal Morbidity, one of the largest maternal morbidity audits, reported that acute pulmonary oedema was the fourth most common form of maternal morbidity [1]. The incidence of stillbirths and neonatal deaths in mothers who suffered severe preeclampsia was 22.2/1000 and 34.1/1000, respectively, in the UK with a higher incidence in developing countries [2]. In India the incidence of preeclampsia is 7.6% during pregnancy of which 3.3% is severe preeclampsia. Delivery of the infant and placenta is the only effective treatment [3]. A combination of mifepristone and a prostaglandin preparation is usually recommended as the first-line intervention for induction of labour [4]. In a patient of IUFD, delivery of the fetus within 24 hours becomes necessary to prevent development of Disseminated Intravascular Coagulopathy (DIC) and other complications. We wish to report a successful management of 29 weeks Primigravida patient with severe Pregnancy induced hypertension (PIH) and established pulmonary oedema with IUFD using epidural anaesthesia for normal vaginal delivery.

Case History

A 40 years old Primi gravida with 29 weeks of gestation (conceived after In Vitro Fertilization) with history of severe Pregnancy Induced Hypertension (PIH) during pregnancy, admitted in nearby nursing home with absent fetal movements. She was diagnosed to have IUFD on Ultrasonography, hence decision was taken to induce labour & deliver vaginally. In view of low Hb- 7.2 gm%, 2units of whole blood was transfused in the same hospital. Patient developed breathlessness and bilateral crepitation noticed on auscultation with declining Oxygen saturation. Hence she was shifted to Jupiter Hospital for further management.

On admission, in view of severe PIH with established acute Pulmonary oedema, patient was immediately shifted in Intensive Care Unit. Vitals on admission were HR- 106/min, NIBP- 180/100 mmHg, SpO_2- 94% with 4lit Oxygen by mask, RR- 30/min and patient had cough with respiratory distress. She was given Oxygen via Hicon mask and intravenous nitro-glycerine infusion (0.05mcg/kg/min) to maximise Oxygenation and control of systemic blood pressures respectively. Baseline investigation showed Hb - 9 gm%, normal coagulation profile, Platelets (1.90 lac) and 2D-echocardiography (fair right and left ventricular functions), negative D-Dimer. Invasive monitoring with left radial Arterial catheter & right internal jugular venous catheter established for better hemodynamic monitoring. After multidisciplinary discussion involving Obstetrician, Cardiologist. Anaesthesiologist and Intensivist, it was decided to proceed with vaginal delivery in OR, so that immediate operative intervention could be done in case of cardiorespiratory decompensating. Patient was shifted to OR after taking written informed consent in view of emergency endotracheal intubation if required with possibility of postoperative ventilator support, IV furosemide 40mg bolus given to extract the extra fluid collection from lungs.

Inside OR patient was started on non-invasive ventilation (NIV) on Pressure control mode using PEEP- 8 cm H_2O , FiO_2- 100% in view of pulmonary oedema. Full hemodynamic monitoring established and vitals were HR- 28/min, Invasive BP- 160/100 mmHg (with NTG), RR- 30/min, SpO_2 – 100% and auscultation of chest- frank Crepitation. She was in active labour on Oxytocin infusion. Intravenous Labetalol infusion started at 2mg/Hr and NTG discontinued. As the coagulation profile and platelet count were normal, labour analgesia was initiated with 18G epidural catheter under all aseptic precaution inserted at L3- L4 intervertebral space in sitting position to facilitate patient's efforts. Epidural top-up Inj Lignocaine 2% given in incremental doses after test dose. After patient was made comfortable, labour pain reduced, respiratory rate reduced to 22-24/min, HR- 100/min, BP-150/100mmHg, and SpO_2 – 100%. PEEP titrated and escalated gradually up to 16 cm H_2O and simultaneously FiO_2 reduced till 80%. She delivered normally with forceps assistance. I.V Morphine 7mg given in increments to aid treatment of pulmonary oedema. Patient was shifted back to ICU continuing NIV and IV labetalol with controlled hemodynamic parameters. She was gradually weaned off NIV support the next day and shifted toward after uneventful recovery.

Discussion

The patient described has severe PIH and developed pulmonary oedema in late pregnancy due to fluid overload with blood transfusion received in view of low Haemoglobin. Compared with non-pregnant adults, healthy pregnant women demonstrate an increased cardiac output, increased heart rate, increased blood volume, physiological anaemia, decreased systemic vascular resistance and decreased blood pressure [5]. Acute pulmonary oedema; represents a form of decompensated acute cardiac failure. The underlying mechanism of acute pulmonary edema in these circumstances depends on the underlying hemodynamic state of the pregnant woman. Not only are there cardiac structural and functional abnormalities, but there are also alterations in fluid balance associated with hypoproteinemia [6-8]. There is often preserved left ventricular ejection fraction, indicating significant cardiac reserve; however, the heart is still unable to generate a large enough cardiac output to deliver oxygen to the vital organs.The associated elevated pulmonary venous pressure at rest reduces lung compliance, increases the work of breathing and results in the subjective feeling of breathlessness [9-11].

Preeclampsia is a disorder of widespread vascular endothelial malfunction & vasospasm due to increase in vasoactive substances like thromboxane, endothelia & intracellular free calcium as well as deficiency of vasodilators like nitric oxide & prostacyclin. These type of patients usually have hyperdynamic status with normal to high Cardiac Output, normal Pulmonary capillary wedge pressure (PCWP) and Central venous pressure (CVP). Despite normal filling pressure, intravascular fluid volume is reduced (30 to 40%). They have high chances of developing in to pulmonary edema due to reduced colloid oncotic pressure [12]. Our patient had pulmonary oedema due to fluid overload in a background of PIH.

IUFD is more common in severe PIH due to spasm of uteroplacental circulation leading placental insufficiency. IUFD mother can have many complications the commoner are [13]

A. Infection: due to rupture of membranes, infection especially by gas forming organism like CL. Welchi may

occur.

B. Blood Coagulation Disorder: if the fetus remains more than 4weeks there is possibility of defrination from silent DIC.

In this case the uncontrolled hypertension and already developed acute pulmonary oedema could have worsened through sympathetic nervous system activation, because of acute vasoconstriction and increased afterload due to redistribution of fluid from the peripheral circulation to the pulmonary vessels [14]. In addition to the usual management goals of stabilizing the woman and treating the acute pulmonary oedema, consideration needs to be given to delivery of the fetus if acute pulmonary oedema occurs in the antenatal period.

Hence the goal of treatment is as follow:

1. Decreased Left Ventricular (LV) Preload
2. Decreased LV Afterload
3. Decreased or prevent Myocardia Ischaemia
4. Maintain oxygenation & ventilation with clearance of pulmonary oedema
5. Treat or avoid Infection
6. Early delivery of baby to prevent DIC

Fine balance of Vitals like Heart Rate (HR), Blood Pressure (BP), Oxygen Saturation (SPO_2), Respiratory Rate (RR), Temperature and Urine output are very important. Preoperative Anaesthesia Check rarely required considering a medical emergency triggering emergency response. Despite the risk of endotracheal aspiration due non-fasting status and delayed gastric emptying in pregnant mothers, Non-invasive Ventilation should be tried as initial technique which gives us following advantages

1. Provides increased inspired O_2 concentration.
2. Displaces the fluid from alveoli in to pulmonary vessels and then to systemic circulation.
3. Decreases work of breathing
4. Decreases the need for tracheal intubation

Acute rise in B.P should be prevented as [15]:

1. Nitroglycerine can be given by S.L. spray/ infusion
2. Sodium Nitroprusside infusion
3. Cardioselective B-Blockers, Nifedipine, Hyralazine.
4. Magnesium can be given as prophylactically if there are signs of impending eclampsia

Epiduaral Analgesia has great role in such type of patients. Preloading of the circulation is commonly performed prior to epidural blockade to prevent hypotension. However, in this patient, there was already clinical evidence of fluid overload, and over- infusion of fluids seemed dangerous, hence Fluid balance in the perioperative period was strictly monitored. The drug-induced tachycardia may be clinically misinterpreted a sign of hypervolemia. Drug-induced tachycardia, associated with fluid overload in late pregnancy, precipitated cardiac failure. We believe epidural blockade represents a rational form of therapy in this situation considering the following advantages:

1. It acts as a sympatholytic by reducing the maternal stress as a result of labor pain relief and venodilatation thus expanding the intravascular space
2. Pain relief in labour by blocking nociceptive input and sympathetic efferent, release of catecholamines, β-endorphins, ACTH, and reduction of cortisol, thereby reducing uterine hyperactivity and in between hypoactivity, uncoordinated uterine contractions is converted to normal contractions, leading to balance of maternal respiratory rate and amplitude thus maintaining maternal acid base balance [16,17].
3. Decreases blood pressure in preeclampsia.
4. In addition to these physiologic benefits, epidural labor analgesia also provides patients with psychologic comfort and alleviation of excessive pain and ensures that they leave the hospital more satisfied.

Conclusion

Acute pulmonary edema in pregnancy is a life threatening emergency. Early intervention with multidisciplinary approach reduces postoperative morbidity and mortality while dealing with complications of PIH. Epidural Analgesia should be considered in absence of HELLP syndrome. NIV has a significant role in treating Acute Pulmonary oedema. Normal Vaginal delivery is better than caesarean section in IUFD.

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