Primary Pulmonary Hypertension In Pregnancy: Vaginal Delivery With Epidural Labour Analgesia

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Abstracts: Primary pulmonary hypertension is a very rare, progressive, incurable disease, the only curable option being heart lung transplant. When pregnancy is associated with pulmonary hypertension due to any cause, it carries very poor prognosis with mortality rate ranging from 30-50%. More risk is involved during labour & peripartum period, as labour pain with hypercarbia, hypoxia, acidosis, increases sympathomimetic responses and pulmonary vascular resistance which could be fatal to parturient. Epidural labour analgesia with painless vaginal delivery attenuates these responses & improves survival rate. It also helps in accommodating the auto transfused blood in postpartum period due to controlled vasodilatation & so avoiding right ventricular failure. Case report: We report a case of a primigravida patient with primary pulmonary hypertension who was advised therapeutic abortion, but she continued with pregnancy and underwent vaginal delivery with epidural analgesia & was continuously haemodynamically monitored non-invasively during labour & postpartum period. [Mankar S et al NJIRM 2012; 3(4) : 135-138]

Key Words: Primary pulmonary hypertension, Pregnancy, Epidural labour analgesia

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Introduction: Incidence of primary pulmonary hypertension in general population is very low i.e. 2 cases per million[1]. Hence there are few studies in view of management of primary pulmonary hypertension with pregnancy. The method of delivery of these women is controversial. Most authors consider mortality to be higher after LSCS[2]. There are various studies showing lot of complications with invasive methods of monitoring[3,4,5]. Our case is unique as we conducted our case with all non-invasive methods of monitoring including 2D Echo. While doing this we kept all invasive modalities ready including all emergency drugs & preparation for LSCS by extending epidural segmental block level or under GA if necessary.

Case history: We report a case of 32 weeks primigravida, a known case of primary pulmonary hypertension. She was advised therapeutic abortion at 12th week of gestation, but she continued pregnancy accepting all risks explained to her. Later she stopped follow up and was on irregular treatment with T. Sildenafil 25 mg BID & T.Torsemide 10 mg OD. As pregnancy advanced she had progressive increase in breathlessness. At 32 weeks gestation she was admitted in ICU with labour pains and grade IV dyspnoea. On general examination - Weight -53kg, Height-153cm, Heart rate was 120 /min with sinus tachycardia, Blood pressure 130/80 mm Hg, Respiratory rate 40 /min, decreased Oxygen saturation i.e. SPO_2 - 86% on room air, raised JVP 8 cm above clavicle, bilateral pedal oedema up to ankle. On CVS examination she had parasternal heave, accentuated P^2 component of 2nd heart sound, with mild systolic murmur of tricuspid regurgitation.

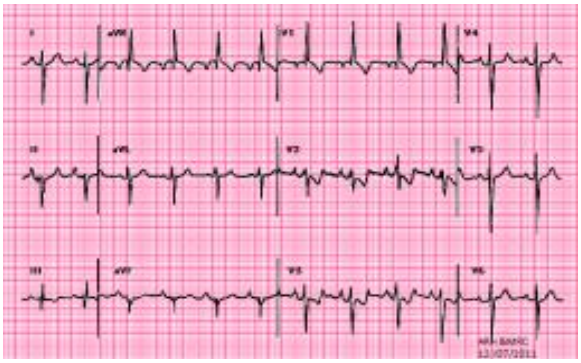


Fig-1 Ecg showing tall R waves in right leads, right axis deviation & right ventricular hypertrophy with strain patern

Chest Radiogram taken after proper shielding of abdomen showed perihilar haze with prominent pulmonary arteries [fig.2]. ECG showed right axis deviation & right ventricular hypertrophy, tall R wave in lateral leads. [fig.1]. 2D echo showed normal intracardiac anatomy but right atrial & ventricular dilatation [fig.3], impaired right

ventricular systolic function with tricuspid regurgitation. Right ventricular end systolic pressure was 85 mm Hg which directly reflected the pulmonary arterial pressure. Left heart was normal. T. Bosentan 62.5mg BID was started by the cardiologist. We continuously administered Oxygen by ventimask at 6 l/min and non-invasively monitored her HR, ECG, RR, SPO_2, urine output, foetal heart rate (till delivery of baby) since admission of patient and during post partum period also.

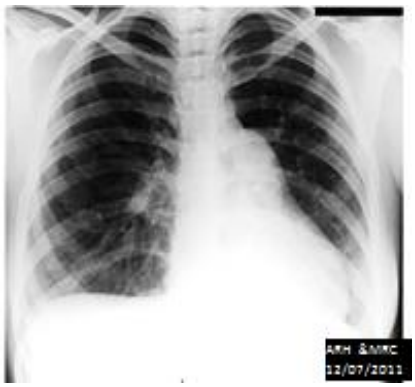


Fig-2 Prominent pulmonary vasculature with peripheral pruning, perihilar haze

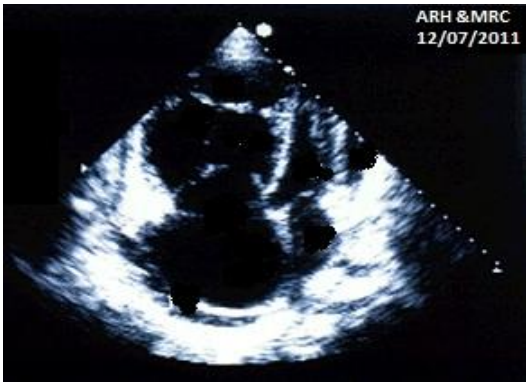


Fig-3 Dilation of right atrium and right ventricle, right ventricular end systolic pressure 85 mmHg, normal left ventricular function

2D Echo was done by cardiologist on admission, after epidural analgesia, post delivery of foetus, after 4hrs of delivery and before shifting from ICU to ward.

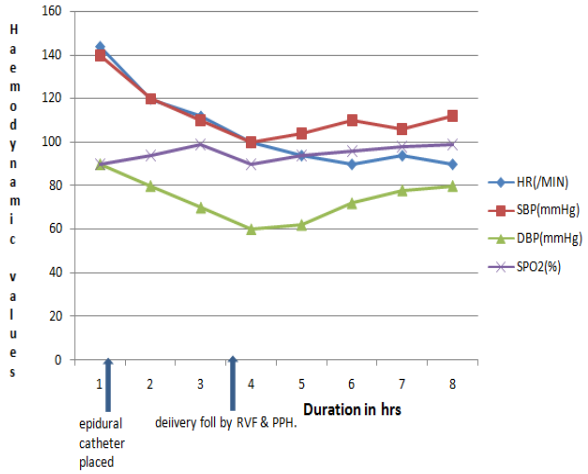
After proper counselling, psychological preparation of patient and relatives, written informed consent was taken for epidural labour analgesia. Patient's haemodynamics were noted i.e. HR-140/min, BP-140/90 mm Hg (increased during contraction), RR-44/min, SPO_2- 96% to 98%. 18G epidural catheter was introduced through 18G Tuohy needle ,when cervix was 2-3cm dilated in L3-4 space in left lateral position. After test dose of 3ml of 1.5% adrenalized (1:200000) Lidocaine, 6ml of 0.125% Bupivacaine was given. Top up doses of 3ml of 0.125% of Bupivacaine was administered at 30 minute intervals. After 3 hrs & 45 min she delivered a low birth weight female child of weight 1600 gm whose Apgar scores improved progressively with 7,8,9 at 1, 5, 10 min respectively.

Post delivery, as expected due to auto transfusion, she developed mild RVF as evidenced by decreased SPO_2, increased RR, increased filling of right heart by 2D Echo, increased JVP 10 cm above clavicle, which responded to oxygen & diuretics - Inj. Torsemide 40 mg iv. She also developed mild postpartum haemorrhage which was treated with uterine massage, infusion of Oxytocin 20 U in 200ml Ringer Lactate, & T. Misoprostol 400 µg per rectal. Twelve hrs after removing the epidural catheter we administered inj. Low molecular weight Heparin 60 mg subcutaneous BID and on 3rd day T. Warfarin 5mg OD and shifted from ICU to Ward on 3rd day with stable haemodynamics. Later she was discharged on 7th day on T. Sildenafil, T. Bosentan, T. Torsemide & T. Warfarin with above doses.

Discussion: Primary pulmonary hypertension is defined as sustained elevation of pulmonary artery pressure (mean >25mm Hg at rest & >30 mm Hg on exertion) in the absence of a demonstrable cause[1,3,4,6]. Pathophysiology of PPH is pulmonary arteriopathy, vascular remodelling, vasoconstriction & thrombosis in pulmonary vasculature. Histologically we see plexogenic pulmonary arteriopathy, thromboembolic, veno-occlusive types.

There are many newer modalities available for treatment of PPH but none totally cure . These are also not available at peripheral level like our institute. We have T. Sildenafil

(phosphodiesterase-5 inhibitor which relaxes pulmonary vascular smooth muscle) & T. Bosentan (ETA endothelin receptor blocker). Oxygen therapy replaces the decreased levels of blood oxygen and causes pulmonary vasodilatation decreasing PVR. Diuretics remove extra fluid from tissues & blood and help in easy breathing. Other modalities like prostaglandin I_2, Nitric Oxide are not available at our institute.



In pregnancy there are physiological changes in haemodynamics with hypercoagulable states which aggravate the PPH. In labour contraction forces up to 500 ml blood from systemic to pulmonary circulation thus increasing preload. Pain & expulsive effort of labour increases the HR, BP, RR, O2 consumption etc[7]. Principles of delivery are to avoid increases in pulmonary vascular resistance, keep normal preload, afterload & haemodynamics[3].

Advantages of epidural analgesia are to reduce pain, O2 consumption and so haemodynamic consequences of labour[4]. EA causes no increase in pulmonary vascular resistance due to decreased pain and stress of labour, and with advantage of controlled vasodilatation for auto-transfusion[8]. In our case, as there was no obstetric indication for surgery, after proper counselling of patient epidural analgesia was initiated for normal vaginal delivery. It was found that after giving epidural analgesia there was gradual improvement in HR, BP and SPO2.

Labour & spontaneous delivery places a lot of strain on the heart. Death often occurs during delivery or early postpartum period mainly because of RVF. And as was expected our patient also developed mild RVF, which was managed by diuretics and oxygen. Patient also developed mild postpartum haemorrhage which was treated by uterine massage, Oxytocin, & Misoprostol (Pg E1 analogue- causes pulmonary vasodilation[2]).

Since admission we continuously monitored the patient by all non-invasive methods[9]. For clinical management and research purposes, there has been an increasing awareness of potential complications of invasive monitoring[5]. Non-invasive methods of cardiac output measurement used in obstetric anaesthesia have provided valuable information on maternal and foetal well-being and haemodynamics in critical care setting and during regional anaesthesia for caesarean delivery. These techniques include Transthoracic echocardiography[5, 9]. So we kept invasive modalities ready if required which included central line for CVP, arterial line for invasive blood pressure, Swan Ganz catheter for PCWP measurement but fortunately we did not require them. We had a standby cardiologist who monitored heart status intermittently with 2D Echo and other parameters. Hence when there was increase in right heart filling after delivery we managed the developing RVF by giving diuretics timely to avoid increasing preload & restricting IV fluids[10,11].

Anticoagulation was started to prevent thromboembolism and was continued for 3 months[6].

From this case report we can safely assume that though the avoidance or termination of pregnancy in parturient with primary pulmonary hypertension is the best option, pregnancy can be continued with risks explained and can be successfully managed with epidural labour analgesia with continuous peripartum haemodynamic monitoring.

In conclusion patients of primary pulmonary hypertension can undergo normal vaginal delivery with epidural labour analgesia & vigilant monitoring in presence of good teamwork by anaesthesiologist, obstetrician, cardiologist & nursing staff.

Key messages: Though primary pulmonary hypertension is rare, incurable and fatal disease with high mortality in pregnancy, these patients can undergo normal vaginal delivery with epidural labour analgesia & vigilant monitoring in presence of good teamwork by anaesthesiologist, obstetrician, cardiologist & nursing staff

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