

## Idiopathic pulmonary edema in pregnancy

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### ABSTRACT

Acute pulmonary edema in pregnancy is associated with increased risk of maternal and fetal morbidity and mortality. The normal physiological changes during pregnancy as well as the use of tocolytic agents, underlying cardiac diseases, fluid overload and pre-eclampsia predispose the development of pulmonary edema which is usually self limiting. In those cases where no immediate and obvious cause of pulmonary edema is readily apparent, all the possible investigations should be performed to rule out underlying pathology.

**Key words:** pulmonary edema, pregnancy, fluid overload, tocolytic agent

### INTRODUCTION

Pulmonary edema is defined as the accumulation of fluid in the pulmonary interstitial spaces and alveoli; thereby preventing the adequate diffusion of both oxygen and carbon dioxide. Although an uncommon event in pregnancy, it is associated with an increased risk of maternal and fetal morbidity and mortality.<sup>1</sup>

Several risk factors have been identified such as preeclampsia or eclampsia,<sup>2</sup> use of tocolytic therapy,<sup>3</sup> severe infection,<sup>4</sup> cardiac disease,<sup>5</sup> iatrogenic fluid overload,<sup>6</sup> and multiple gestation.

Normal physiological changes that occur during pregnancy are increase in cardiac output beginning as early as 5<sup>th</sup> weeks reaching its peak by 31<sup>st</sup> weeks. Systemic vascular resistance reduces to a maximum between 16 to 24 weeks. There is an increase in plasma volume by 45% whereas red cell volume increases by 20-30%, leading to hemodilution. Pregnancy results in faster resting heart rate, bounding pulses, widened pulse pressure, low blood pressure, and warm extremities. Pericardial impulse is hyperkinetic and shifted laterally and upwards. In the intrapartum period, cardiac output further increases by 12 to 31% in first stage which can be attributed to the sympathetic stimulation, tachycardia and increased myometrial oxygen consumption. It further increases by 49% in second stage. During labour, maternal bearing down efforts results in about 300 to 500ml of blood autotransfused from the uterus to systemic circulation. These physiologic changes associated with pregnancy

may predispose the patient to pulmonary edema.<sup>7</sup>

### CASE REPORT

A 33 year old woman, married for 12 years, gravida 5, para 4, at 29<sup>th</sup> weeks of gestation with polyhydramnios and hypothyroidism presented with complaints of cough with expectoration since one month, swelling in lower limbs since five days and difficulty in breathing since last three days. There was no history of any tocolytic agent usage, hypertension or cardiac disease. She had regular cycles. There was no past history of hypertension, cardiac disease, bronchial asthma or tuberculosis. Nothing significant was revealed in family history. She revealed that she had similar complaints in her previous pregnancies. So it was thought of a case of recurrent idiopathic pulmonary odema.

On examination, she was afebrile and all the vitals were stable; and there was no pallor, cyanosis, clubbing, jaundice or lymphadenopathy. Clinically she was euthyroid. Cardiovascular examination did not reveal any abnormalities. On auscultation, bilateral coarse crepitations were present. Uterus was overdistended with head of fetus downwards, and fetal heart sound was normal.

Laboratory Findings- Hemogram values were within normal limits. Ultrasonography demonstrated single live intrauterine pregnancy corresponding to 30 weeks gestation with no abnormalities. Chest X-ray suggested cardiomegaly. Echocardiography showed concentric left ventricular hypertrophy with normal left ventricular systolic function. Ejection

fraction was 60%, grade 1 Left ventricular diastolic dysfunction, trivial MR, mild TR, and moderate PAH (PASP -57mm of Hg) with small pericardial effusion.

She was treated with IV antibiotics, diuretics, oxygen and other supportive measures. After 2 weeks of treatment amniocentesis was done for polyhydramnios. She was symptomatically better following the procedure. At 33 weeks she went into spontaneous labour and delivered vaginally a live male baby weighing 1.92 kg. Her postpartum period was uneventful.

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## AUTHOR NOTE

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## REFERENCES

1. Sibai BM, Mabie WC, Harvey CJ, Gonzalez AR. Pulmonary edema in severe preeclampsia-eclampsia: Analysis of 37 consecutive cases. *Am J Obstet Gynecol.* 1987;156:1174-9.
2. Benedetti TJ, Kates R, Williams V. Hemodynamic observations in severe preeclampsia complicated by pulmonary edema. *Am J Obstet Gynecol.* 1985;152:330-4.
3. Davies AE, Robertson MJS. Pulmonary edema after administration of IV salbutamol and ergometrine. *Br J Obstet Gynaecol.* 1980;87:539-41.
4. Donnelly JF, Lock FR. Causes of death in 533 fatal cases of toxemia of pregnancy. *Am J Obstet Gynecol.* 1954;68:184-7.
5. Clark SL. Structural cardiac disease in pregnancy. In: Clark SL, Phelan JP, Cotton DB, eds. *Critical care obstetrics.* Oradell, New Jersey: Medical Economics Books, 1987:92.
6. Hankins GD, Havith JC, Kuehl TJ. Ritodrine hydrochloride infusion in pregnant baboon sodium and water compartment alterations. *Am J Obstet Gynecol.* 1983;147:254-9.
7. Metcalfe JL, Ueland K. Maternal cardiovascular adjustments to pregnancy. *Prog Cardiovasc Dis.* 1974;16:363-74.