International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Venkatesh M et al. Int J Reprod Contracept Obstet Gynecol. 2025 Oct;14(10):3590-3593
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20253118

Case Report

Acute pulmonary edema secondary to severe preeclampsia in a 47-year-old elderly gravida conceived via *in vitro* fertilization: a case report

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Received: 03 August 2025
Accepted: 05 September 2025

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ABSTRACT

Advanced maternal age and assisted reproductive technologies, such as in vitro fertilization (IVF) with oocyte donation, are recognized as risk factors for many disorders in pregnancy, including preeclampsia. Severe preeclampsia can, in rare instances, be complicated by acute pulmonary edema, posing significant risks to both the mother and the fetus. We report a rare case of a 47-year-old elderly primigravida with a BMI of 30 kg/m² (obese) who conceived via IVF and developed severe preeclampsia at 30+5 weeks of gestation with a hypertensive crisis that progressed to cause a life-threatening acute pulmonary edema. Immediate management included intravenous antihypertensives, diuretics, magnesium sulphate, and ventilatory support. Due to the deteriorating maternal and fetal status, an emergency lower segment caesarean section was performed in the intensive care unit. A 1.2 kg baby girl was delivered and admitted to the neonatal intensive care unit. The mother showed gradual postoperative improvement with stabilization of blood pressure and resolution of pulmonary symptoms. Her recovery period was aided by a multidisciplinary team involving obstetricians, anaesthesiologists, neonatologists, intervention specialists, and clinical psychologists. The case emphasizes the need for heightened vigilance and aggressive management in IVF pregnancies especially among women of advanced maternal age who are at increased risk of hypertensive disorders, fluid shifts, and cardiovascular stress. Risk assessment in elderly women undergoing IVF is crucial, along with vigilant monitoring and prompt multidisciplinary intervention to improve outcomes in high-risk pregnancies.

Keywords: Preeclampsia, Acute pulmonary edema, In vitro fertilization, Elderly primigravida, Multidisciplinary care, Lower segment caesarean section

INTRODUCTION

In vitro fertilization pregnancies are now increasingly common among women of advanced reproductive age and have been shown to carry a heightened risk for gestational hypertension, preeclampsia, and adverse obstetric outcomes. Hypertensive disorders of pregnancy affect up to 10% of pregnancies globally, with preeclampsia accounting for 3%–5% of these cases. Acute pulmonary edema is a rare but serious complication that occurs during pregnancy, with a reported incidence rate ranging from 0.08% to 1.5%.[1] Pulmonary edema is characterized by the excessive accumulation of extravascular fluid in the lung parenchyma, resulting in impaired gas exchange at the alveolar level. Clinically, it is diagnosed by the presence of the sudden onset of breathlessness, crackling rales, and/or decreased saturation. Severe preeclampsia is the most common cause of acute pulmonary edema during pregnancy. Some of the other causes include peripartum cardiomyopathy, multiple pregnancy, infections, and fluid overload.[2] The mechanisms involved in preeclampsia-induced pulmonary edema are largely undefined - high plasma volume, reduced plasma oncotic pressure, increased capillary permeability, and pulmonary capillary hydrostatic pressures are a few probable mechanisms.[3] Authors present a rare case of acute pulmonary edema in a 47-year-old primigravida conceived via in vitro fertilization with severe preeclampsia, highlighting the

importance of individualized risk stratification and multidisciplinary management in high-risk obstetric scenarios.

CASE REPORT

A 47-year-old booked elderly primigravida, been married for three years, conceived via IVF using a donor oocyte, was diagnosed with pregnancy -induced hypertension at 20 weeks of gestation and was managed with oral labetalol 100 mg twice daily. She was also a known case of hypothyroidism, on Thyronorm 75 mcg once daily. Her body mass index (BMI) was 30 kg/m^2, classifying her as obese. At 30 weeks and 5 days of gestation, the patient presented to the Outpatient Department with complaints of headache for 2 days. On examination, her blood pressure was found to be 220/100 mmHg. The patient was counselled for admission due to her severely elevated blood pressure and was also informed of the risks and complications related to severe preeclampsia. However, the patient and her attender initially refused admission, attributing the headache to lack of sleep over the past two days. After prolonged counselling, she was admitted to the labour ward and was given intravenous labetalol 20 mg as a stat dose for managing the elevated blood pressure. For eclampsia prophylaxis, she was given a loading dose of magnesium sulfate 4g over 20 minutes, according to the Zuspan regimen.

Thirty minutes post admission, the patient developed a sudden onset of severe breathlessness and cough. Her blood pressure was 170/110 mmHg. Oxygen saturation was 85% on room air. Bilateral coarse crepitations were heard in the lower lung fields, suggestive of flash pulmonary edema secondary to severe preeclampsia. She was given intravenous furosemide 40 mg and started on oxygen therapy via nasal prongs at 10 litres per minute for the immediate management of her symptoms. Due to further oxygen desaturation to 56%, the Medical Emergency Team (MET) was alerted, and the patient was urgently transferred to the Medical Intensive Care Unit (MICU). In the MICU, her condition continued to deteriorate, and fetal bradycardia was also noted.

The attenders were counselled by the team of senior obstetricians and anaesthesiologists regarding the patient's critical status and the need for intubation followed by emergency caesarean section in view of persistent fetal bradycardia unresponsive to resuscitative methods. The patient underwent intubation and was started on intravenous labetalol infusion. Due to the persistently elevated blood pressure of 260/200 mmHg, indicating hypertensive crisis, nitro-glycerine infusion (1 mg/ml) was initiated. Given the worsening maternal condition and evidence of fetal bradycardia, an emergency crash lower segment caesarean section (LSCS) was performed in the Intensive Care Unit. A baby girl weighing 1.2 kg was delivered. The baby only responded after two cycles of positive pressure ventilation and was admitted to the Neonatal Intensive Care Unit (NICU).

Postoperatively, the mother's blood pressure stabilized on nitro-glycerine infusion, achieving a target of 160/110 mmHg. She was started on a maintenance dose of magnesium sulfate (1 gram per hour for 24 hours) as per the Zuspan protocol. Her chest radiograph (Figure 1) showed bilateral perihilar opacities in a bat-wing distribution, suggestive of acute pulmonary edema. Mild cardiomegaly and increased vascular markings were also noted. She was started on an empirical antibiotic therapy of intravenous piperacillin-tazobactam and metronidazole. By postoperative day 2, she was weaned off ventilatory support. Ophthalmology opinion was obtained for papilledema and was found to be normal. Compression stockings were used as part of thromboprophylaxis. Soon, her parenteral antihypertensives were gradually withdrawn, and she was started on oral antihypertensive therapy consisting of labetalol 200 mg three times daily, clonidine 0.1 mg twice daily, and nifedipine 10 mg three times daily. A repeat echocardiogram showed normal cardiac function with an ejection fraction of 60%.

She was subsequently shifted to the postnatal ward. During the hospital stay, the patient recovered well. She received chest physiotherapy, and psychological counselling was also provided to reduce the risk of postpartum depression and post-traumatic stress disorder (PTSD).[4] The baby responded well in the NICU and was subsequently handed over to the mother. The recovery of the patient was aided by a multidisciplinary team involving obstetricians, anaesthesiologists, neonatologists, clinical psychologists and interventional specialists. The patient's oral antihypertensives were tapered accordingly before her discharge, and although she had a prolonged hospital stay of 28 days, she showed significant clinical improvement and was discharged with a healthy baby.

Figure 1: Chest radiograph of bilateral perihilar opacities in a bat-wing distribution, suggestive of acute pulmonary edema. Mild cardiomegaly and increased vascular markings noted.

DISCUSSION

While in vitro fertilization, particularly through oocyte donation, has emerged as a boon for treating infertility, it has also been recognized as an independent risk factor for hypertensive disorders in pregnancy, postpartum hemorrhage, and higher caesarean section rates.[5] In a large retrospective analysis of women aged 45–59 years (mean age 47.3), undergoing oocyte donation IVF cycles demonstrated that this approach is successful in enabling conception among women of advanced maternal age. However, obstetric complications were commonly seen, affecting 37.8% of the women who delivered, of which 2 cases of preeclampsia were reported.[6] This study underscores that, although IVF with donor oocytes can result in successful pregnancies in women of advanced reproductive age, it carries a significant risk of hypertensive disorders of pregnancy, including preeclampsia, which, in rare cases, as illustrated in the present case report, can progress to flash pulmonary edema, a life-threatening complication.

Obstetric outcomes that occur with increased frequency in women of advanced age include early pregnancy loss, ectopic pregnancy, multiple gestation, placenta previa, protraction/arrest disorders, and caesarean birth. Severe maternal morbidity and maternal mortality are also increased. Fetal/neonatal outcomes that occur with increased frequency in women of advanced age include fetal chromosomal abnormalities and some congenital anomalies, low birth weight (LBW) or preterm. There is also an increased risk of perinatal mortality.[7] A study conducted at a tertiary care centre reviewed 90,540 deliveries, among which 540 women (0.6%) were above the age of 45 years. Of these, 67 women (12.4%) developed preeclampsia, and 4 cases (6%) were further complicated by acute pulmonary edema. This study highlights that acute pulmonary edema is a rare but serious complication of preeclampsia, particularly in pregnancies that involve an advanced maternal age.[8]

Preeclampsia primarily stems from abnormal development of the placenta, characterized by failure of penetration of the cytotrophoblast cells into the myometrial segment of the spiral arteries, leading to local hypoperfusion and resulting in the release of various factors, including inflammatory cytokines and antiangiogenic proteins, that contribute to systemic endothelial response, manifested clinically as preeclampsia and intrauterine fetal growth restriction.[9] Potential serious maternal sequelae of preeclampsia include placental abruption, pulmonary edema, HELLP syndrome (characterized by microangiopathic hemolysis, elevated liver enzymes, and low platelet count), cerebral hemorrhage, hepatic failure, acute kidney injury, seizure (eclampsia) and death. Fetal and neonatal manifestations include fetal growth restriction, oligohydramnios, prematurity, small for gestational age, and stillbirth.[10] Acute pulmonary edema often necessitates admission to an intensive care unit and is also a major cause of mortality in women with preeclampsia. Pulmonary oedema may occur in up to approximately 3% of women with preeclampsia, with 70% of cases occurring after birth. Only 30% of cases of pulmonary edema in the setting of preeclampsia occur before delivery.[11] A landmark study by Sibai et al analyzed 37 consecutive cases of pulmonary edema in patients with severe preeclampsia or eclampsia over a 9-year period and reported an incidence of 2.9%.[12]

Acute pulmonary edema often presents with common symptoms that include breathlessness, orthopnoea, restlessness, and cough. Clinical signs may involve tachycardia, rapid breathing, auscultatory findings such as crackles or wheeze, an S3 gallop, heart murmurs, and decreased oxygen saturation. Radiographic findings on chest X-ray may reveal signs like upper lobe redistribution, Kerley B lines, and pulmonary infiltrates. Additional diagnostic tools, including arterial blood gas analysis and echocardiography, can aid in confirming the diagnosis. Management includes initiating non-invasive ventilation, if necessary, alongside prompt control of severely elevated blood pressure using intravenous antihypertensives. In cases where preeclampsia is complicated by pulmonary edema, nitro-glycerine (glyceryl trinitrate) is often preferred to be used. Diuretics such as furosemide are beneficial, and calcium channel blockers like nicardipine or nifedipine may be added, particularly when diastolic dysfunction is evident. Intensive monitoring and high-dependency care are critical for optimal outcomes. Additionally, consideration needs to be given to the delivery of the fetus if acute pulmonary oedema occurs in the antenatal period.[13]

Appropriate risk reduction strategies and proper long-term monitoring are essential to minimize the complications that may arise in the later stages of life.[11]

CONCLUSION

This case highlights a rare but life-threatening occurrence of acute pulmonary edema secondary to severe preeclampsia in an elderly primigravida conceived via in vitro fertilization. Pulmonary edema in pregnancy, though rare, demands immediate recognition and intervention due to its rapid progression and potential for maternal and fetal compromise. This case emphasizes the need for heightened vigilance and aggressive management in IVF pregnancies especially among women of advanced maternal age who are at increased risk of hypertensive disorders, fluid shifts, and cardiovascular stress. It also reflects the importance of adhering to national IVF regulations, including upper age limits and pre-conception counselling, to minimize maternal morbidity. Multidisciplinary team involvement and prompt obstetric interventions play a critical role in improving outcomes in such high-risk scenarios.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Venkatesh M, Mathimaaran S, Narayanan P. Acute pulmonary edema secondary to severe preeclampsia in a 47-year-old elderly gravida conceived via *in vitro* fertilization: a case report. Int J Reprod Contracept Obstet Gynecol 2025;14:3590-3.