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Case report

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Preeclampsia complicated with pulmonary edema: a case report

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Abstract

Pulmonary edema is one of the most serious complications of preeclampsia that should be ruled out in case of dyspnea in a pregnant woman, especially in the context of preeclampsia. It is an indication of urgent pregnancy-termination. Many theories have been proposed to explain this phenomenon, such as hypervolaemia, left ventricular failure and pulmonary capillary leakage, but it is still not well understood. The prognosis is generally good after adequate management and symptoms completely disappear within a few days of delivery. We report a case of a 30 year old woman with pulmonary edema that complicated preeclampsia and discuss the management options.

Introduction

Pulmonary edema is a very rare complication of pregnancy. It is more often seen as a complication of preeclampsia. The physiopathology of this association is not well understood. It is a life-threatening condition that needs prompt treatment and pregnancy termination. We report the singular case of a 30 year old woman that has developed severe preeclampsia, complicated with pulmonary edema on the third trimester of her pregnancy, and do a review of literature.

Patient and observation

We report the case of a 30-year-old woman, with no particular pathological history, gravida 4 parity 3. She was referred for preeclampsia complicating a 39 weeks pregnancy. Upon arrival to the emergency room, she was conscious, BP at 17/10 cmHg. She reported the presence of neurosensory signs (headache, tinnitus and visual fog sensations). She also had stage III dyspnea. Heart rate was at 102, and saturation at 82%. She had peripheral edema, with no other signs of heart failure. On pulmonary auscultation, there were audible crackles located at the bases of the lungs. The patient was not in labor. The ultrasound revealed a monofetal pregnancy with positive heart activity, a cephalic presentation, an amniotic fluid in normal quantity, a fundic placenta and an estimated fetal weight at 3059g. Chest X-ray revealed alveolar opacities located in the perihilar areas, related to acute pulmonary edema. Echocardiography showed that cardiac cavities were of normal size and function, and confirmed the absence of valvulopathy. The biological tests had shown a slight anemia (9.2g/dl) but the renal and liver functions were normal and there were no other test abnormalities. The patient was hospitalized in maternal intensive care unit, where she was put under non-invasive ventilation, magnesium sulfate, nicardipine and furosemide. After stabilization of the patient, a C-section was performed, with the extraction of a male, healthy newborn with an APGAR at 10/10th and a weight of 2kg 900g. There were no post-operative complications. The evolution was marked by the disappearance of the dyspnea within 72 hours, and stabilization of the blood pressure.

Discussion

Preeclampsia is mainly caused by an abnormal development of the placenta, with a failure in the penetration of the cytotrophoblast cells into the myometrial segment of the spiral arteries [1]. Local hypoperfusion results in release of various factors, including inflammatory cytokines and antiangiogenic proteins, including the soluble form fms like tyrosine kinase 1 (sFlt 1) and soluble endoglin (s-Eng) as well as low circulating maternal concentrations of vascular endothelial growth factor (VEGF) and placental growth factor (PLGF) [2] that contribute to systemic endothelial response, manifested clinically as preeclampsia and intrauterine fetal growth restriction [1]. The short-term cardiovascular complications of preeclampsia with severe features (PEC) include heart failure, pulmonary edema, and stroke [3]. A recent prospective observational study, has shown that PEC was associated with higher right ventricular systolic pressures and decreased right ventricular longitudinal systolic strain. For left-sided cardiac parameters, there were some differences in mitral septal velocity, left atrial area size and septal wall thickness [4]. Pulmonary edema (PE) is a life-threatening condition that can be associated with preeclampsia. It accounts for only 0.08% [5]. It is more often caused by severe preeclampsia [6], which is associated in 2.9% with pulmonary edema [4]. PE may be associated with many clinical symptoms (breathlessness, orthopnoea, agitation and cough) and signs (tachycardia, tachypnoea, crackles and wheeze on chest auscultation, cardiac S3 gallop rhythm and murmurs, decreased oxygen saturation). Typical chest X-ray features include upper lobe redistribution, Kerley-B lines and pulmonary infiltrates [7].

Many mechanisms have been proposed to explain the pathogenesis of pulmonary oedema in severe

pre-eclampsia including hypervolaemia, left ventricular failure and pulmonary capillary leakage [8]. Pulmonary oedema could be due to a combination of these factors. However, it is thought that increased systemic vascular resistance induces significant changes in loading conditions of the ventricular myocardium contributing to diastolic filling abnormalities and to the development of an ischemic substrate with the potential for development of heart failure, pulmonary edema and/or sudden death [2]. A retrospective review demonstrated that acute pulmonary oedema in hypertensive pregnant women was strongly associated with increased intravenous fluid administration in the units that have unrestricted fluid policies for women during labour, c-section and magnesium sulphate seizure prophylaxis [8]. The use of intravenous fluids in women with preeclampsia remains controversial, although preeclampsia results in a state of initial intravascular hypovolaemia [8]. The treatment of pulmonary edema in PE is similar to those of non-pregnant patients: oxygen therapy, water restriction, intravenous furosemide (80 mg initially) and central hemodynamic monitoring. Reduction in afterload is obtained with the use of vasodilators (hydralazine, nifedipine) [9]. Non-invasive ventilation should be tried as the initial technique before tracheal intubation [10]. Urgent decrease of critically high blood pressure using an intravenous antihypertensive medication is also necessary. Nitroglycerin (glyceryl trinitrate) is recommended for management of pregnant woman with acute pulmonary oedema [8].

Other alternative agents are: sodium nitroprusside, intravenous furosemide, calcium channel antagonist such as nicardipine or nifedipine, Prazosin as well as hydralazine may also be considered [8]. Reduction in systolic and diastolic blood pressure should occur at a rate of approximately 30 mmHg over 3-5 min followed by slower reductions to blood pressures of approximately 140/90 mmHg. (bolus 20-40 mg over 2 min) [8]. The occurrence of pulmonary oedema indicates the urgent termination of pregnancy [9]. Since most pulmonary edema cases in pregnant women are associated with difficult-to-control hypertension, the prevention of this complication goes by the prevention of preeclampsia [9]. It has been shown that the use of calcium and low-dose aspirin are considered effective in clinical practice. Calcium supplementation (calcium carbonate, 1,000-2,000 mg/day) and the use of small daily doses (50-170 mg) of aspirin for at-risk groups are the only alternatives that have shown some degree of effectiveness in randomized clinical trials [9]. Restrictive fluid policies with median total fluid administration of 2100 ml in the peripartum period may also be a way of preventing pulmonary edema [11].

Conclusion

Acute pulmonary edema is a leading cause of death in women with pre-eclampsia. It is a rare entity. The management relies on antihypertensive treatments and on prompt delivery. Caution should be taken when administrating fluids to preeclampsia patients to prevent PE. Prognosis is generally good and return to normal occurs within a few days.

Competing interests

The authors declare no competing interests.

Authors' contributions

All the authors have read and agreed to the final manuscript.

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