(*Acta Anaesth. Belg., 2018, 69, 165-168*)

Acute respiratory distress in a twin pregnancy : a case report

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Abstract : When multiple obstetric complications come together in a pregnant patient with undocumented non-obstetric comorbidities, a sudden and unexpected clinical presentation may dramatically change the therapeutic strategy.

We describe the case of a 35-yr-old primigravid woman with a twin pregnancy who developed severe acute respiratory distress at 32 weeks of gestation during hospitalization for the indication of a cervical shortening. She received tocolytics and corticosteroids for fetal lung maturation and presented signs of mild pre-eclampsia during hospitalization. Because maternal condition worsened despite supportive measurements, an emergency caesarean section was performed under general anesthesia. Postoperatively, echocardiography revealed a previously unknown mitral valve insufficiency, which was thought to be chronic as stable findings were observed 4 weeks postpartum. The presence of pre-eclampsia in a patient with chronic mitral valve insufficiency and the use of a combination of betamethasone and tocolytic treatment were thought to be the triggering factors for the dramatic worsening of her hemodynamic status.

Due to the rising incidence of risk factors for preterm delivery and pre-eclampsia situations, in which the combination of tocolysis and fetal lung maturation is indicated, such a complication might theoretically become more frequent. As illustrated by our case, this can complicate management and may unexpectedly result in cardiopulmonary decompensation during pregnancy. A multidisciplinary approach is needed to insure an optimal outcome for the mother and the child. When treatment with a combination of tocolytics and corticosteroids is indicated in a pre-eclamptic patient, a diagnostic echocardiography and continuous monitoring of arterial oxygen saturation with pulse oximetry should be considered.

Keywords : pregnancy ; DCDA twin ; acute respiratory distress ; pre-eclampsia ; mitral valve insufficiency ; corticosteroids ; tocolysis ; fetal lung maturation ; caesarean section ; general anesthesia.

Introduction

We present a case of sudden cardiorespiratory collapse in a previously uncomplicated twin pregnancy. Written consent was obtained from the patient to publish her case.

Case report

A 35-yr-old primigravid woman with a dichorionic diamniotic (DCDA) twin pregnancy was admitted to the gynecology department at a gestational age of 32 weeks because of cervical shortening (19 mm). Her medical history was unremarkable and her home medication consisted of folic acid. Cardiotocography showed normal fetal heart rates with reactive traces and no regular contractions. Vital signs were normal. Urine dipstick was positive for proteins and a 24-hour urine collection revealed a borderline negative proteinuria of 297 mg/24hours (normal range < 300).

Forty-eight hours after admission, an even shorter cervix (14 mm) was measured with clinical and tocographic signs of prodromal labor. Therefore, tocolysis with intravenous (IV) atosiban and steroids for fetal lung maturation [2 injections of betamethasone 12 mg with a 24-hour interval) were provided, see timeline in Figure 1 (appendix)].

On day 4 of hospitalization, the patient awakened due to the onset of dyspnea, orthopnea and chest pain. Clinical examination showed normal breath sounds, a respiratory rate of 22/min, normal

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heart sounds, a blood pressure of 148/93 mmHg, a pulse rate of 72 bpm, a peripheral saturation of 93-97% sitting upright, and no peripheral edema. Arterial blood gas (21% O_2) suggested a pregnancy-induced hyperventilation with limited hypoxia. ECG examination was normal. Blood results showed a hemoglobin concentration of 8.4 g/dL (normal 11.7-15.5), uric acid 7.0 mg/dL (2.6-6.0), normal platelet count, normal liver and renal function, normal PT and aPTT, negative troponins, and negative proBNP. An emergency chest CT with IV contrast revealed only minimal pleural effusion and excluded pulmonary embolism. With oxygen delivery at 5 L/min and accentuated upright position, the patient was comfortable and saturation increased to 93%. As technical investigations were inconclusive, obstetricians opted for close monitoring of maternal-fetal parameters.

However, the patient's clinical condition worsened with progressive dyspnea and inspiratory basal crepitations on lung auscultation. Peripheral saturation dropped to 88% despite supplemental oxygen, and blood pressure was elevated (systolic 140-155 mmHg, diastolic 90-100 mmHg) with clinical hyperreflexia. Venous blood sample showed that the hemoglobin and platelets dropped respectively from 8.4 to 7.9 g/dL and from 189 to 142 x10^9/L, uric acid increased to 8.2 mg/dL. The combination of clinical signs of pulmonary edema, hyperreflexia, hypertension, borderline proteinuria on admission and suggestive lab results made pre-eclampsia the most presumable diagnosis. Intravenous magnesium sulfate ($MgSO_4$) was started to prevent seizures. Continuous cardiotocography showed normal traces.

Because maternal respiratory distress further worsened, an emergency cesarean section was performed. General anesthesia was administered (rapid sequence induction with propofol 2 mg/kg, rocuronium 1.2 mg/kg and remifentanil 100 µg IV; maintenance with sevoflurane, remifentanil and oxygen FiO_2 80%) because of severe orthopnea and low arterial O_2 saturation. Two healthy infants were delivered after an uneventful intraoperative course.

Postoperatively, mechanical ventilation was continued at the intensive care unit (ICU) because of low oxygen saturation due to pleural effusion and pulmonary edema. Transesophageal echocardiography (TEE) showed normal left and right ventricular systolic function with mitral insufficiency (MI) grade 2-3/4, tricuspid insufficiency (TI) grade 1/4 and minimal aortic insufficiency (AI); there was no evidence of shunt, pulmonary hypertension or thrombotic material in the heart. After antihypertensive (nicardipine and labetalol) and diuretic treatment, blood pressure and oxygenation returned to normal and extubation was indicated 4 hours postoperatively.

Four days postpartum, transthoracic echocardiography (TTE) showed a normal left and right ventricular systolic function and a MI grade 2-3/4. The chest X-ray revealed a discrete lung effusion.

The remaining postpartum period was eventless. Blood pressure normalized, without need for antihypertensive medications. One month after delivery, the results of ambulatory TTE were comparable, with a MI 2/4.

Discussion

To our knowledge, this is the first reported case of acute respiratory distress in a pregnancy due to a combination of corticoid administration, tocolysis, pre-eclampsia, and MI.

Severe pre-eclampsia alone can cause pulmonary edema due to an increased sympathetic tone, decreased colloid oncotic pressure, elevated afterload and increased capillary permeability (1) In this case however, pre-eclampsia was only mild.

Pregnancy places an increased burden onto the cardiovascular system. A maternal heart disease occurs in approximately 0.5 to 4% of all pregnancies and is associated with considerable morbidity and mortality rates (2,3). In pregnant women with known heart diseases, strict prenatal care and early risk stratification during pregnancy are essential to improve pregnancy outcome (2,4). Mitral regurgitation during pregnancy is usually due to a mitral valve prolapse (prevalence 2.4% in the general population) (5,6) or rheumatic valvular disease (7,8). Reduced left ventricular afterload and significant fall in systemic vascular resistance explain why even severe MI is well tolerated during an otherwise uncomplicated pregnancy (4,7). In contrast, acute onset MI is poorly tolerated (4). Asymptomatic patients with MI do not require therapy during pregnancy (7). However, patients with symptoms of left heart failure can be treated with diuretics and digoxin (7). Hydralazine and organic nitrates can be used for vasodilatation during pregnancy (7).

Because prior echocardiography was absent, it is impossible to prove that her MI was chronic. However, the TTE performed four days and four weeks postpartum (when the patient was asymptomatic and discharged from the ICU/hospital) showed the same severity of MI (grade 2-3/4) as the TEE immediately postpartum, while there was

still a need for mechanical ventilation. Therefore, we believe that this asymptomatic MI was already present before conception.

Preeclampsia in combination with chronic MI has a detrimental effect on the cardiovascular system. An increased sympathetic tone with resulting elevated afterload will increase the severity of mitral regurgitation. Borges et al. demonstrated that pregnancy causes unfavorable structural alterations associated with hemodynamic overload in women with MI (9). We hypothesize that, in our patient, these maladaptive cardiovascular changes in combination with pre-eclampsia and pre-existing MI elicited left heart failure with pulmonary edema after the administration of tocolytics and corticosteroids. Rapid delivery played a crucial role in the stabilization of the patient, by reversing the physiologic pregnancy-associated cardiovascular adaptations and the pathologic changes associated with pre-eclampsia.

Corticosteroid therapy before 34 weeks of gestation is safe and efficient in patients with pre-eclampsia (10,11) (repeated after 24 hours and then once a week). In contrast, the combination of corticosteroids and tocolysis in pre-eclamptic patients is associated with an increased risk of maternal pulmonary edema (12-14). Therefore, when corticosteroids and tocolysis are indicated in pre-eclamptic patients, a diagnostic echocardiography and continuous pulse oximetry monitoring should be considered.

The diagnosis of MI as atriggering factor in this case was only made after delivery and stabilization of the maternal condition. The differential diagnosis focused primarily on pulmonary embolism and pulmonary edema due to the presence of known risk factors for these complications (respectively the relative immobilization during pregnancy and pre-eclampsia in combination with tocolytics/ corticosteroids). Moreover, the absence of a relevant medical history or known familial cardiomyopathy made MI in this case less presumable. In this report, we want to emphasize a key message: clinicians should be aware of possible previously undocumented but relevant cardiac comorbidities that may be the triggering factor for cardiopulmonary decompensation during pregnancy. Only a multidisciplinary approach, with an open and sound communication between gynecologist, anesthesiologist, intensivist and cardiologist will insure an optimal outcome for the mother and the child.

Appendix

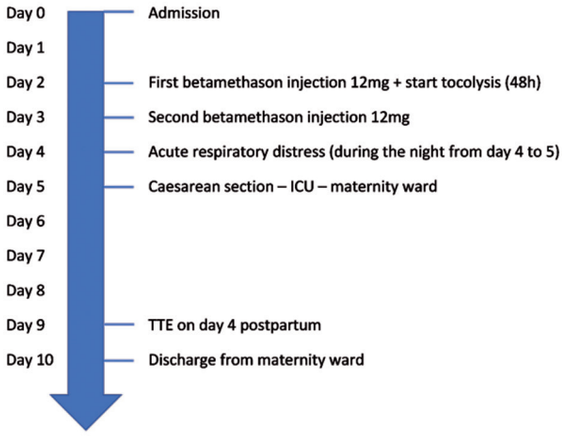

Figure 1. — Timeline.

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