Myocardial Infarction in the 31st Week of Pregnancy - Case Report

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Summary: Duarte FP, O'Neill P, Centeno MJ, Ribeiro I, Moreira J - Myocardial Infarction in the 31st Week of Pregnancy - Case Report.

Background and objectives: The occurrence of acute myocardial infarction (AMI) during pregnancy is rare. The authors describe the case of MI in a 31-week pregnant woman and the importance of a multidisciplinary team for its approach.

Case report: Thirty-one week pregnant woman with history of smoking, alcoholism and hypertension was admitted after an episode of syncope. On admission she was conscious and asymptomatic, although hypotensive. The electrocardiogram showed marked ST-segment elevation in D1, aVL, V1-V6. The cardiac enzymes were positive. The transthoracic echocardiogram showed reduction in septal and left ventricular contractility and an ejection fraction of 30%. Angiography revealed proximal occlusion of the left anterior descending artery. After a non-successful balloon angioplasty, a metallic stent was placed. The patient started therapy with beta-blockers, aspirin and clopidogrel. As for the delivery, we chose to perform a cesarean section four weeks after MI. Clopidogrel was suspended seven days before delivery. The preoperative cardiac function was improved by infusion of levosimendan started the day before. Cesarean section occurred under epidural block. The intraoperative period showed no complications, except for mild hypotension easily corrected with phenylephrine. The Apgar score for the newborn was 9 / 10.

Conclusions: This is one of the few cases of myocardial infarction and angioplasty reported during pregnancy. The authors discuss the decisions taken by the multidisciplinary team consisting of anesthesiologists, obstetricians, neonatologists and cardiologists, particularly with regard to dual antiplatelet therapy, the type of delivery and anesthesia.

Keywords: Pregnancy Complications, cardiovascular; Myocardial Infarction; Angioplasty; Cesarean Section.

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INTRODUCTION

The occurrence of an acute myocardial infarction (AMI) during pregnancy is estimated at about 6 per 100,000 births [1]. Although it is a rare phenomenon, it is associated with high maternal-fetal morbidity and mortality.

In the first published meta-analysis, maternal morbidity was between 19% [2] and 37% [3]. It is currently estimated at 5% and appears to be higher during the third trimester of pregnancy [4].

There are several factors that contribute to the three to four times higher incidence of AMI during pregnancy, namely: increased volemia, increased cardiac output, increased heart rate and decreased oxygen supply to the myocardium caused by anemia, and physiological diastolic hypotension.

Maternal mortality is higher in the first 15 days after AMI. The risk of fetal mortality ranges from 9% to 13% [1].

Some independent risk factors for AMI during pregnancy have been identified: age over 35 years, hypertension, *diabetes mellitus*, smoking, pre-eclampsia, hemorrhage and postpartum infection [1,5,6].

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The authors describe a case of AMI during the third trimester of pregnancy, assisted by a multidisciplinary team that includes: obstetrician, anesthesiologist, cardiologist and neonatologist who performed the diagnosis, immediate stabilization with use of percutaneous angioplasty with stent placement and subsequent clinical optimization, aiming a safe delivery without complications.

CASE REPORT

Thirty-nine years old pregnant woman in the 31st week of pregnancy, gesta 4, para 4, was admitted to the Obstetrics Emergency Service for loss of consciousness in public.

The patient had a history of chronic hypertension, post-traumatic epilepsy for 4 years, chronic alcoholism, macrocytic anemia and smoking habits of 40 packs/year.

She was medicated in outpatient care with alpha-methyldopa and hydantoin with poor therapeutical adherence in consequence of severely low socioeconomical conditions.

On admission, the patient was asymptomatic, although hypotensive (mean arterial pressure of 53 mmHg). Continuous monitoring of electrocardiographic trace suggested ST-segment depression in D_{II}. A 12-lead ECG was then performed showing marked ST-segment elevation in V1-V6.

Analytically, the patient presented elevated cardiac enzyme panel with CPK of 5,236 ng.mL^{-1} and troponin of 10.77 ng.mL^{-1}. Worth mentioning the macrocytic anemia presented (hemoglobin 10 gr.dL^{-1}, Hct: 32%, MGH 100 fL), leukocytosis (15.6 x 103 cells.uL^{-1}) with neutrophilia (93%) and altered liver enzymology (AST 469 IU.L^{-1}, ALT 68 IU.L^{-1}, LDH 1,000 IU.L^{-1}).

Transthoracic echocardiography showed apical akinesia and severe hypokinesia of the mid and apical segments of the septum and anterior wall and with severely impaired global systolic function (LVEF 30%).

Coronary angiography revealed thrombotic occlusion of 100% in the proximal left anterior descending artery with TIMI 0 distal flow.

A primary angioplasty of the proximal anterior descending artery was performed with placement of metallic stent. Despite these measures, however, the distal flow remained very low (TIMI 1), so that after discussing the risk/benefit ratio, we opted for the administration of abciximab.

Given the high mortality during the first two weeks post-infarction and the absence of fetal distress, delivery was delayed four weeks after fetal lung development and maternal clinical and laboratorial optimization.

Surgical delivery meeting the pelvic presentation of the fetus was suggested as an alternative to the hemodynamic consequences of a potentially prolonged labor and the possibility of tubal ligation.

The pregnant woman was admitted to the Coronary Intensive Care Unit (CICU), which conducted the serial evaluation of the maternal cardiac function and the fetal well-being. Drug therapy with acetylsalicylic acid (ASA), clopidogrel, and carvedilol was initiated.

The value of troponin normalized after 20 days, while heart function did not presented any improvement. Clinically, the patient remained as class III of the New York Heart Association (NYHA).

A week before the scheduled date for delivery, clopidogrel was suspended in order to minimize blood loss and enable local/regional anesthesia. The remainder of the medication was continued until the morning of surgery.

Twenty-four hours before the cesarean section, levosimendan infusion at 0.35 $mg.h^{-1}$ was initiated, which was reduced to 0.175 $mg.h^{-1}$ on the day of surgery.

In face of the proposed surgery and the value of preoperative hemoglobin (9.9 $gr.dL^{-1}$), two units of packed red blood cell were reserved.

Beds in CICU and in neonatal ICU were arranged.

Upon arrival at the operating room, the patient was asymptomatic, in NYHA class II. The following criteria were continuously monitored: electrocardiographic trace, pulse oximetry, invasive arterial pressure, respiratory rate and fetal focus.

Cesarean section was performed under epidural anesthesia performed with titrated administration of 12 mL of ropivacaine at 7.5 $mg.mL^{-1}$ and the sufentanil at 1 $\mu g.mL^{-1}$, in order to get sensory block until T_6.

Levosimendan at 0.175 $mg.h^{-1}$ was maintained by perfusion, and oxygen supplementation via face mask at 5 $L.min^{-1}$.

The intraoperative progressed without complications, except for mild arterial hypotension (decrease in MAP not exceeding 20% of baseline) easily reversed with a 10 to 20 mg of phenylephrine *bolus* and suspension of the levosimendan perfusion.

After delivery, uterine massage was encouraged as to ensure hemostasis of the placental bed in order to reduce the need for oxytocin, which was initiated in slow infusion (40 U in 1000 mL of 5% dextrose in saline at about 80 $mL.h^{-1}$).

Tubal ligation was performed as previously informed consented.

Blood loss was sensitive (600 mL). Therefore, transfusion of one unit of packed red cell unit and about 300 mL of hydroxyethylamide was performed.

The male newborn was born with 2,500 g with an Apgar score of 9/10 at the first and fifth minutes, respectively.

The post-operative analgesia was ensured by operative wound infiltration with 10 mL of ropivacaine at 10 $mg.mL^{-1}$, 1 g of intravenous paracetamol infusion (8/8h), and epidural morphine perfusion at 0.12 $mg.h^{-1}$ through an elastomeric infuser for 48 hours.

In the immediate postpartum period, there were no obstetrical or anesthetic complications. There was no acute decompensation cardiac failure, remaining in NYHA class II.

Clopidogrel was reinstituted 48 hours postpartum, after removal of the epidural catheter.

The patient was discharged on the seventh day after childbirth, medicated with ASA, clopidogrel, captopril and carvedilol.

DISCUSSION

There are few cases of AMI reported in the literature, and even fewer angioplasty during pregnancy.

The initial approach of AMI during pregnancy does not differ greatly from the non-pregnant patient. However, it presents some peculiarities.

Pregnancy has traditionally been a relative contraindication to the use of thrombolytic agents, given the fear of maternal and fetal complications such as preterm delivery, placental abruption, fetal death, postpartum hemorrhage and teratogenicity.

There are about 200 published cases of thrombolytic therapy in pregnant woman for various reasons, including AMI [7]. The series of case published to date show favorable results. The maternal and fetal mortality associated with this treatment is estimated at 1% and 6%, respectively [8].

However, its use in the third trimester raises some ethical considerations, particularly with regard to the risk of any surgical procedure 10 days after its administration.

In the case described, we chose to administer abciximab as an adjuvant to angioplasty, since the flow distal to the stenosis did not improve significantly. There is only one published case of use of antiplatelet during pregnancy [9], also with no record of fetal complications.

The first option in respect of the pregnant woman's coronary revascularization must be a balloon angioplasty, since it requires only 2 to 4 weeks of dual antiplatelet therapy. This is an important aspect of a population that may require surgery at any time.

Another feature of the approach of AMI during pregnancy is associated with mandatory monitoring of fetal well-being through cardiotocograms and serial obstetric ultrasounds.

In the absence of fetal distress, delivery should be postponed at least three weeks after AMI, since maternal mortality is of up to two weeks post-MI, at a maximum [10].

According to published literature, the birth does not necessarily have to be surgical [11-13].

If normal delivery is chosen, effective analgesia during labor should be ensured in order to avoid physiological changes induced by pain, such as tachycardia, increased peripheral vascular resistance and increased myocardial oxygen needs. These patients have often received dual antiplatelet therapy, which imposes some limitations on the conduct of local/regional techniques, especially if labor is not programmed. Another concern is associated with sympathetic blockade and the consequent vasodilation and hypotension linked to neuroaxial blockade. The expulsive period should be shortened through instrumentation [14].

Although the cesarean birth is associated with greater blood loss, hemodynamic instability, and infections, we opted for cesarean section because of pelvic presentation of the fetus, in order to avoid a prolonged labor, and the possibility of proceeding with tubal ligation during the surgery. The tubal ligation was particularly important given the severely impaired left ventricular function, which strongly advised against another pregnancy [15], due to the nature of indigent pregnant women.

The choice of epidural anesthesia was based on the installation of more gradual blockade, compared to the subarachnoid block, and the possibility of making the post-operative analgesia through this pathway [16].

Although current guidelines advocate [17] dual antiplatelet therapy for at least four to six weeks after placement of a metallic stent, the decision to discontinue clopidogrel was taken jointly between anesthesia and cardiology, in order to minimize blood losses and neuraxial regional anesthesia, which should be the technique of choice instead of general anesthesia.

Some studies suggest that abrupt withdrawal of the drug results in a proinflammatory and prothrombotic state [18]. It has been further suggested that the maintenance of perioperative antiplatelet agents is not associated with increased blood loss [19]. However, the available information about the true impact of thienopyridines in non-cardiac surgery is still insufficient.

In complementation to the attempt of reperfusion by angioplasty and the introduction of medical therapy with antiplatelet agents and beta-blockers, the infusion of levosimendan was fundamental in the optimization of cardiac function in this patient. This is also true given the high risk of the proposed surgery in patients who had an extensive anterolateral MI for less than 25 days, with severe left ventricular dysfunction leaving sequelae.

Levosimendan is a calcium channel sensitizer used in the treatment of decompensated heart failure. This agent sensitizes troponin C to calcium, increasing calcium in the effect of myofilaments during systole and improving contraction without increased energy expenditure. For its beneficial effect, it is still determining its vasodilatory action through the opening of ATP dependent potassium channels. Through its inotropic and vasodilator effect, levosimendan enhances cardiac output without increasing myocardial oxygen requirements [20]. There are few reported cases of its use during pregnancy [21,22].

We can conclude that the approach of these patients is a challenge in diagnosis and therapy, and that it should be viewed in the context of a multidisciplinary team involving obstetricians, anesthesiologists, cardiologists and neonatologists.