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Anesthetic management of maternal Mirror syndrome

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ABSTRACT
Mirror syndrome (Ballantyne syndrome, triple edema, maternal hydrops, pseudotoxemia) is a rarely diagnosed condition associated with pregnancy that can be life-threatening for both the mother and fetus. There is limited literature on its pathogenesis and anesthetic management, making prevention and treatment complex. The duration of pregnancy and severity of maternal or fetal presentation often determines outcome. We describe the anesthetic considerations of a morbidly obese parturient with Mirror syndrome.

Keywords: Mirror syndrome; Cesarean section; Combined spinal–epidural anesthesia

Introduction

Mirror syndrome is a rare obstetric entity where a mother 'mirrors' the edema of the hydropic fetus and placenta. Pathogenesis of the condition is unknown. It is hypothesized that it is due to a maternal inflammatory response from trophoblastic debris[1] or increased placental production of proteins leading to vascular leakage.[2,3] Increased maternal morbidity and mortality are seen with Mirror syndrome.[4–6] If correction of the underlying fetal abnormality is not possible, current treatment is to deliver the hydropic fetus and placenta. Mirror syndrome has similar features to those of preeclampsia and may confound decision-making. We present the case of a parturient with Mirror syndrome and her anesthetic management.

Case report

A 42-year-old obese G2P1 woman at 34 weeks and five days of gestation presented with bilateral lower extremity pitting edema with profound vulvar edema. Weight gain fluctuated between 3 and 4 kg throughout pregnancy with total weight gain of 17 kg: a sudden increase of 11 kg over one week was considered significant. Evidence of fetal hydrops was found on ultrasound during a routine antenatal consultation and the patient was transferred to our hospital for further evaluation by the maternal-fetal medicine team. The pregnancy was complicated by abscesses on her pannus, nephrolithiasis and a recently diagnosed urinary tract infection. Her past medical history included severe vitamin A deficiency with concomitant night blindness, depression and anemia. Surgical history consisted of a gastric bypass 13 years previously, biliary-pancreatic diversion and an anal fissure repair. The patient denied any anesthetic complications. Her obstetric history consisted of a spontaneous vaginal delivery at term without neuraxial anesthesia eight years earlier. Current medications were prenatal vitamins, vitamin D3, vitamin A and iron. The patient reported anaphylaxis to zolpidem. Routine prenatal screening was negative for chromosomal abnormalities.

Initial laboratory values ordered by the obstetric team were normal (white blood cell count 16×10^9/L with no bandemia, hemoglobin 11.5 g/dL, hematocrit 34.9%, platelets 173×10^9/L, glucose 4.3 mmol/L,

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sodium 137 mmol/L, potassium 3.9 mmol/L, chloride 104 mmol/L, carbon dioxide 24 mmHg, urea 3.9 mmol/L, creatinine 72 μmol/L, calcium 2.1 mmol/L, uric acid 0.39 mmol/L, alkaline phosphatase 146 IU/L, alanine aminotransferase 46 IU/L and aspartate aminotransferase was 47 IU/L). A urine specimen showed 2+ leukocyte esterase, trace hemoglobin and no proteinuria. The maternal-fetal medicine team confirmed the diagnosis of severe fetal hydrops on ultrasound. All serologic studies to evaluate a cause for fetal hydrops were negative except for parvovirus immunogloblulin G. A maternal echocardiogram was normal. The pediatric cardiology team performed a fetal echocardiogram, but maternal body habitus made the study difficult. A diagnosis of a non-immune fetal hydrops and maternal Mirror syndrome was made.

The patient's weight was 108.4 kg and her height 170 cm (body mass index of 38 kg/m^2). Her systolic pressure was between 90 and 108 mmHg and diastolic pressure between 51 and 60 mmHg with a heart rate of 65–80 beats/min, respiratory rate of 16 breaths/min, temperature of 36.6°C and oxygen saturation of 99% on room air. The patient was cognitively intact. Physical examination included a class II Mallampati airway with full range of motion of her neck and no evidence of oral soft tissue edema. Her lungs were clear to auscultation and the cardiac examination was normal. Lumbar landmarks were not palpable due to large body habitus but the midline could be identified. The patient had non-erythematous pitting edema of the lower extremities. The fetal heart rate ranged from 120 to 140 beats/min with good variability. A 24-h urine specimen showed a creatinine of 1.4 g and protein of 165 mg.

On day two of admission the maternal-fetal medicine team decided to induce labor due to worsening maternal edema and concern for fetal compromise. A decision was made to not place an epidural catheter during induction of labor due to concerns about the patient's borderline systolic blood pressures of 90–95 mmHg. Over the next 24 h, the fetal heart tracing demonstrated recurrent late decelerations with moderate variability, and the patient's cervix failed to dilate past 6 cm. The decision was made to perform a cesarean section. Repeat examination did not show any change in airway classification or expansion of lower extremity edema into the pre-sacral or lumbar areas. A repeat hematocrit was 30.6%.

A phenylephrine infusion of 40 μg/mL was prepared, and the patient was given oral sodium citrate 20 mL. Upon entering the operating room, the patient was transferred to the operating room table, placed in a sitting position and connected to a 3-lead electrocardiogram, pulse oximeter and blood pressure cuff set to cycle at 3-min intervals. Cephazolin 2 g and a bolus of lactated Ringer's solution 1500 mL were administered through the single 18-gauge intravenous catheter in the upper extremity. Initial blood pressure was 141/82 mmHg with a sinus tachycardia of 101 beats/min. The patient was anxious. It was challenging to determine the exact location of the intervertebral spaces secondary to body habitus and abrupt progression of lumbar edema noticed only after the transfer to the operating table. Using landmark techniques, without ultrasound guidance, a combined spinal–epidural was performed with some technical difficulty and 0.75% hyperbaric bupivacaine 2 mL, fentanyl 15 μg and preservative-free morphine 150 μg were administered at what was considered to be the L3-4 interspace. A 19-gauge epidural catheter was threaded 5 cm past the tip of the Tuohy needle but with resistance. The patient was immediately placed supine with left uterine displacement. A T4 level of anesthesia was confirmed. Oxygen was delivered via nasal cannula throughout the surgery. Blood pressure immediately decreased to 80/51 mmHg, and despite multiple 40 μg/mL boluses of phenylephrine the patient remained hypotensive. The phenylephrine infusion was started which maintained her blood pressure at pre-neuraxial block values. After delivery of the fetus, the umbilical cord was clamped and oxytocin 20 U in lactated Ringer's solution 1 L was rapidly infused. Estimated blood loss was 1500 mL and one unit of packed red blood cells was transfused. A total of 3 L lactated Ringer's solution was given during the entire procedure. At the conclusion of surgery the epidural catheter was removed. Blood pressure readings continued to improve and the phenylephrine infusion was discontinued after the systolic pressures remained above 100 mmHg. A postoperative hematocrit was 34.3%. The patient did not require intravenous postoperative analgesia in the post-anesthesia care unit and was discharged to the postpartum floor once appropriate criteria were met.

On postoperative day 2, the patient developed erythema and pruritus over her lower extremities and was started on cephalexin for suspected cellulitis. Since the patient's systolic blood pressure had stabilized to 120–125 mmHg, the obstetricians administered intravenous furosemide 20 mg on postoperative day 4 to help reduce the lower extremity edema. The patient continued to do well and was discharged on postoperative day 11.

The neonate was edematous at birth and required intubation by the neonatal team. Apgar scores were 1, 3 and 7 at 1, 5 and 10 min, respectively. In the neonatal intensive care unit, the neonate required high oscillatory ventilation with nitric oxide and inotropic support. Evidence of disseminated intravascular coagulation required transfusion of multiple blood products. An echocardiogram identified total anomalous pulmonary venous return without obstruction, pulmonary hypertension, bidirectional patent ductus arteriosus and an apical ventricular septal defect. The neonate died on day 25.