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Shock After a Caesarian Section for Placenta Previa

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CASE REPORT

A 34-year-old lady, pregnant at 32 weeks of gestation, with a history of bronchial asthma treated with bronchodilators was admitted in the Obstetrics Department with uterus contractions and a tamponading placenta previa. She was initially treated with tocolytics and a caesarean section was planned three weeks later.

After the caesarean section and the placenta excision, severe hemorrhage occurred which made a hysterectomy necessary. During the operation there was an iatrogenic rupture of the posterior urinary bladder wall, as well as rupture of both ureters. The urologist who was called performed repair of bladder and bilateral ureterostomy.

Due to severe blood loss, requiring transfusion with 8 blood and 6 fresh frozen plasma units, the patient was transferred to the intensive care unit (ICU) intubated and on norepinephrine infusion (67 µg/min). Upon the ICU admission she had an arterial blood pressure of 170/80 mmHg and a heart rate of 110/min. The ECG showed sinus rhythm without repolarization abnormalities. A complete blood count examination showed a hematocrit of 27.2%, a hemoglobin of 9.1 g/dl, and a white cell blood count of 13200/µl. The arterial blood gas examination showed pH = 7.49, pCO₂ = 25 mmHg, pO₂ = 162 mmHg, HCO₃ = 19 mmol/l, and oxygen saturation of 99.4% (on inspired oxygen fraction of 50%). The biochemistry tests were normal.

In the ICU the patient received additional 3 blood and 6 fresh frozen plasma units. She had intravascular catheters in both internal jugular veins but the left one was not in place and was removed. She had been intubated for a period of 20 hours, during which there was a gradual decreasing dose and discontinuation of vasopressor agents. The patient was extubated and had low grade fever (T: 37.3°C). After extubation, she had sinus tachycardia 120/min, her blood pressure was 100/65 mmHg and the arterial blood gas examination showed a pH of 7.42, pO₂ 113 mmHg, pCO₂ 30.5 mmHg, HCO₃ 19.4 mmol/l, lactate 4.2 mmol/l on supplemental O₂ of 50% delivered via a Venturi mask.

One hour later, the patient had sudden unexpected tachycardia and hypotension with drop of blood pressure to 70/50mmHg. At this time arterial blood gases were as follows: pH 7.35, pCO₂ 32.5 mmHg, pO₂ 75 mmHg, HCO₃ 17.6 mmol/l, saturation 94.3% (while on O₂ mask at 50%) and lactate of 3.4 mmol/l. The hematocrit was 34.6%. A chest x-ray in the recumbent position showed no evidence of pneumothorax and no other apparent abnormalities. The right jugular catheter was in the lower right atrium. ECG showed sinus tachycardia 170/min with no repolarization abnormalities and low amplitude of QRS complexes. At this point blood pressure was supported with vasopressors and fluids. Intravenous bolus of adenosine was given to discern a possible sinus node re-entrant tachycardia; this led to transient sinus bradycardia

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55/min, and a blood pressure of 60/40 mmHg with dizziness. Atropine was administered and heart rate and blood pressure returned to previous values two minutes later.

Echocardiography was promptly performed which showed a large pericardial effusion with signs of cardiac tamponade, and an emergency echo-guided pericardiocentesis ensued via a subxiphoidal approach. A total of 800 ml of chylous fluid was drained. In order to exclude intrapericardial administration of parenteral nutrition, aspiration from the right jugular catheter was done and normal venous blood was obtained. The analysis of the pericardial fluid showed a lactate dehydrogenase (LDH) value of 116U/l, triglycerides at 1255 mg/dl, total protein at 2.1g/dl, while the fluid cell count yielded 150 white blood cells with 81% neutrophils and 19% lymphocytes.

The next day the patient underwent chest and abdominal computed tomography, immunological and virological tests and mantoux skin test all of which were negative. The fluid culture was also negative. There were no clinical signs of subclavian or internal jugular vein thrombosis. The patient was put on diet high in mid-chain triglycerides and had no relapse during an 8-month follow-up.

DISCUSSION

The pericardial fluid in chylopericardium is milky and opaque as it comprises chyle, the normal content of the lymphatic vessels and the thoracic duct. Although the cholesterol content is high, chylopericardium should not be confused with cholesterol pericarditis in which the fluid contains cholesterol crystals, said to have a glittering "gold paint" appearance but is otherwise clear.

Chylopericardium is a rare cause of pericardial tamponade. As in the present case, the fluid is rich in triglycerides, contains lymphocytes, and has negative cultures. Chylopericardium may be primary (idiopathic) or, more frequently, secondary. Causes are multiple and include trauma, mediastinal

neoplasms, lymphangioliomyomatosis, thrombosis of the subclavian and internal jugular vein, tuberculosis, thoracic surgery, Behçet syndrome, acute pancreatitis and idiopathic chylopericardium which is an exclusion diagnosis.

Based on the results of all diagnostic tests performed, a most plausible cause of chylopericardium in the present case might be a trauma of the thoracic duct due to inappropriate puncture of the left internal jugular vein.

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