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Case Report

Ruptured splenic artery aneurysm in late pregnancy

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ABSTRACT

The spontaneous rupture of the spleen in pregnancy is rare and is frequently misdiagnosed. Failure to recognize the condition can be fatal to mother and foetus at least in a peripheral hospital. In the last trimester of pregnancy it is easily confused with rupture of uterus or abruptio placenta.

Keywords: Spontaneous splenic artery aneurysm rupture, Spontaneous splenic rupture, Third trimester pregnancy, Haemorrhagic shock

INTRODUCTION

We describe below a case of spontaneous rupture of splenic artery aneurysm in the third trimester of pregnancy. The patient was posted for laparotomy for intraperitonal bleeding of undiagnosed origin. 11 units of blood were necessary for resuscitation. This happened in a peripheral hospital in Saudi Arabia where there was no blood bank. 8 units of blood were drawn freshly from donors during the surgical procedure and that enabled us to save the life of the mother. The foetus was already dead before the patient was brought to the operation theatre.

CASE REPORT

A 35 year old multipara of 40 weeks of gestational age, weighing 60 kg was admitted to the above hospital for safe confinement. Her previous five pregnancies were uneventful and all were normal vaginal deliveries conducted in our hospital. She had an uncomplicated course for this pregnancy also. She was non-diabetic, normotensive and afebrile on admission. The investigations showed a hemoglobin level of 11.5 g/dl, hematocrit 0.3, leukocyte count of 6.5 x 10^9/l, platelet count 200 x 10^9/l. Her serological test for toxoplasmosis, which was common in that part of the world, was negative. Blood group was O+ve.

At one am, on the second night after admission, obstetrician was informed of sudden loss of foetal heart beats in this patient who was in bed at that time and was not in labour. Examination by the obstetrician revealed that the patient was in haemorrhagic shock with a pulse rate 120 per minute and systolic arterial pressure of 70 mmHg and diastolic arterial pressure of 40 mmHg. Blood samples were taken for cross matching, haemogram, coagulation profile, before starting 1000 ml normal saline through 16 G cannula in antecubital vein. A bed side ultrasound showed a non-echogenic mass in abdomen and a dead foetus in the uterus. Hb was 5.5 g/dl, haemotocrit 0.25. The coagulation profile was normal. Since the patient was in shock, a surgical colleague also opined for urgent laparotomy and the patient was posted for immediate surgery.

On examination in the operation theatre, patient was very pale, fully alert and talking. She was complaining of generalized abdominal pain. Radial and brachial pulses were not felt. Axillary pulse was feeble and carotid pulse

was satisfactory. Oxygen inhalation of five litres per minute was started by mask, patient connected to SPO_2, ECG and NIBP monitors. Continuous drainage of urinary bladder done. One more 16 G cannula was inserted in other antecubital vein and one unit of uncross-matched O-ve blood started. Before induction 500 ml of haemaccel and 1000 ml ringer lactate were also given. Inj. metochlopramide 10 mg and ranitidine 50 mg i.v. were given.

Induced with ketamine 75 mg i.v. followed by succinyl choline 50 mg i.v. Oro tracheal intubation was achieved with 7 size cuffed tube applying Sellicks manoeuvre. Lungs were ventilated with 50% oxygen in nitrous oxide and an $etCO_2$ monitor connected. Pancuronium was used as a long acting neuromuscular blocking drug. Fentanyl was used as analgesic. Right internal jugular cannulation with 16 G cannula tried and abandoned after many unsuccessful attempts. One more 16 G cannula was inserted through right external jugular vein.

Through a lower midline incision, abdomen was opened. It was filled with blood and clots. After removal of blood and clots it was found that uterus was intact. A dead foetus was delivered through lower segment incision. Inj. ergometrine 0.2 mg and syntocinon 20 units added to a saline bottle, were given for uterine contraction. As source of bleeding was not found in the pelvis, right hypochondrium was explored after extending the abdominal incision upwards. Liver, gall bladder, portal system were found to be normal. When the left hypochondrium was explored a ruptured splenic artery aneurysm near the pedicle was found. Splenectomy and aneurysectomy was done. The splenic vein and the spleen appeared normal. Throughout the surgery, systolic arterial pressure remained below 80 mmHg and was fluctuating, sometimes unrecordable also.

After splenectomy blood pressure slowly started to rise. There were no ECG changes throughout the surgery. $etCO_2$ was in the range of 20 to 25 mmHg.

The duration of the surgery was about three hours and during this period two more units of stored O+ve blood, eight units fresh blood, 1500 ml of normal saline 1000 ml of 5% dextrose saline, 1000 ml of ringer lactate were given. 10 ml of 10% calcium gluconate were given after each three units of blood. Crystalloids were infused through warming device. Some bottles of crystalloids were administered using pressurized infuser bag. As it was very difficult to assess blood loss by conventional methods in this patient, a rough estimate of 3000 ml blood loss was made.

After the completion of surgery, patient became stable with a pulse rate of 100 per minute and systolic arterial pressure 95 mmHg and diastolic arterial pressure of 65. Patient was shifted to ICU and artificially ventilated for 36 hours. The urine output was only 50 ml at the end of surgery, but in the ICU, once the patient stabilized urine output increased to satisfactory levels.

The lab investigations at the end of surgery showed haemoglobin 9 gm/dl, hematocrit 0.3, platelet count 250 x 10^9/l, prothrombin time 16 seconds (10-12), partial thromboplastin time 40 sec (25-30), bleeding time 5 minutes, thrombin time 10 sec (9-11 sec) activated clotting time 150 sec (90-120 sec) calcium 2.5 mmol/l (2.2-2.5). Sodium 145 mmol/l, K 4.5 mmol/l. Since the ABG machine was out of order, pO_2, pCO_2, HCO_3, pH, lactate, BE could not be done.

Artificial ventilation discontinued and tracheal tube removed after 36 hours and thereafter it was uneventful. The spleen was sent for biopsy and the report came as normal splenic tissue.

DISCUSSION

The first case of rupture of spleen in pregnancy was reported in 1803 by Saxtorph.[1] Splenic artery aneurysm is more frequent in women than men in the ratio 4:1. In pregnancy 69% of rupture,[2] occur in third trimester, 2% in first trimester, 10% in second trimester, 13% in labour, 6% in puerperium.

Mortality of splenic artery aneurysm rupture in pregnancy is 75% for the mother and the for the fetus 95.[3] 12 cases have been reported so far in which both mother and fetus survived.[4] A rare case in which mother died and the premature infant survived has been reported.[5]

Splenic artery aneurysm is the most common in splanchnic arterial bed after aortic and iliac artery aneurysm.[6] Atherosclerosis or congenital defect in the arterial wall predispose to aneurysm. Arterial wall weakness increases when there is rise of systemic arterial pressure.

Sparkman,[7] categorized the other causes of splenic rupture in pregnancy as follows:

a) Traumatic, b) rupture of diseased spleen c) toxaemia of pregnancy and d) spontaneous rupture of normal spleen.

a) Traumatic

Most of the cases are easy to diagnose because there will be a positive history. But some cases may present as delayed rupture following minimal or forgotten trauma.

Rupture of diseased spleen

Diseases which affect spleen and cause rupture include hydatid cyst,[8] malaria, splenic angiosarcoma, actinomycosis, non-Hodgkin lymphoma, pancreatic pseudocyst, infectious mononucleosis, leukaemia, thrombosis of the splenic vein, portal hypertension.

b) Rupture due to toxaemia of pregnancy

In toxemia hypertension, thrombosis and diffuse angiitis predispose to vascular rupture.[9]

c) Spontaneous rupture of normal spleen

Can be defined as rupture of normal spleen occurring in absence of trauma. It is encountered mostly in third trimester though cases has been reported in early trimester, labor or in puerperium.[10] Possible factors include congenital malpositioning, short pedicle or a deeper location of the spleen.

Triggering factors include coughing, vomiting, and bearing down efforts in the second stage of labor. A few cases of spontaneous rupture after heparin treatment have been described in the literature.

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