An Unusual Cause of Abdominal Pain in Pregnancy

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CASE

A 33 year old G4P1 presented at 32 weeks gestation generally unwell with abdominal pain and nausea. Past medical history was significant for Type 2 diabetes and a history of recurrent falls over the last 3 months (most recent, one week prior). There was no history of abdominal trauma or vaginal bleeding.

On examination the patient had vital observations as follows: T 35.9, HR 125, BP 120/70 sats 98% RA. Abdomen was soft, with tenderness in the right iliac fossa, and no uterine or renal angle tenderness. Mild irregular tightenings were palpable. Speculum examination showed a long closed cervix, with a weakly positive fetal fibronectin and no evidence of bleeding or rupture of membranes. CTG was abnormal with a baseline of 160 and reduced variability but improved with fluid resuscitation. Multiple bruises were noted on the lower limbs which the patient attributed to previous falls.

Bloods showed Hb 119 WCC 15.4 Plt 268 Neut 12.5. UEC and LFTs normal. CRP 5, lactate 2.6 and blood glucose 8.6. Urine dipstick showed leukocytes and nitrites. The patient was reviewed by the obstetric, ICU and general surgical team. The provisional diagnosis was intra-abdominal sepsis secondary to appendicitis, with a differential diagnosis of pyelonephritis. The patient was started on broad spectrum IV antibiotics and fluid resuscitated with the plan for abdominal ultrasound the following morning.

Over the course of the night the patient developed oliguria and became diaphoretic with worsening tachycardia and pain. Examination showed an increase in RIF tenderness , and a soft non tender uterus. CTG was recommenced and pathological with a baseline tachycardia, reduced variability and shallow decelerations. Bedside ultrasound showed copious free fluid throughout the abdomen and a fetal bradycardia. Decision was made for urgent delivery and exploratory laparotomy with the surgeons available.

The patient underwent a midline laparotomy and emergency lower uterine segment Caesarean section. A 2L haemoperitoneum was noted on entry. The baby was delivered via a T incision for transverse lie, with clear liquor and no evidence of abruption. The general surgeon was in attendance and identified the ruptured spleen as source of bleeding. The patient underwent a splenectomy, blood transfusion and admission to ICU. The post-operative course was complicated by a return to theatre (bleeding splenic hilar vessels) and sepsis. Splenic and portal vein thrombosis was diagnosed on a postoperative CT angiogram, requiring anticoagulation.

During rehabilitation the patient was noted to have an ataxic gait. MRI demonstrated a cystic spinal cord lesion extending from C3 to T7, suggestive of astrocytoma, differential ependymoma. The patient was discharged from the surgical ward on day 19 with ongoing follow up with neurosurgery.

Histopathology showed a 565g spleen 150 x 115 x 60mm, with focally ruptured capsule and underlying haematoma 100 x 45 x 20mm with multifocal intraparenchymal haemorrhage. Remaining parenchyma was normal. No malignancy was identified.

DISCUSSION

Spontaneous splenic rupture during pregnancy is a rare but serious event. Amongst those case reports available, it often presents as left upper quadrant pain and haemodynamic instability necessitating delivery. In the presence of uterine tightenings and/or abnormal cardiotocography the presumed diagnosis is usually placental abruption[1]. The diagnosis of splenic rupture is made following discovery of significant haemoperitoneum on entry, with an intact uterus and clear liquor.

Associated factors reported in the literature include increased maternal age and multiparity, with greatest incidence in the 3rd trimester and puerperium[2]. Diagnosis is often delayed due to absence of trauma and a low index of suspicion.

In a 2012 systematic review of splenic rupture in the absence of trauma or previously diagnosed diseases, pregnancy associated splenic rupture occurred most commonly in a normal pregnancy (22/38 cases), followed by splenic ectopic pregnancy (9/38)[3]. Proposed mechanism for spontaneous splenic rupture have included repeated splenic torsion, obstruction of the collateral drainage, or portal vein thrombosis and spasm of the splenic vein causing congestion. Physiological processes related to pregnancy include increased circulating blood volume, enlargement of spleen and relatively reduced peritoneal cavity[4].

The history of falls secondary to spinal cord lesion may possibly have precipitated our patient's presentation with splenic rupture. Trivial trauma with delayed rupture has been reported, although infrequently. Although the patient did not sustain significant injuries aside from bruising to her lower limbs, perhaps the combination of the fall, gravid uterus and associated pregnancy physiological changes contributed to this extremely rare presentation of abdominal pain.

REFERENCES

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