

## LETTERS TO THE EDITOR—BRIEF COMMUNICATION

**Preoperative diagnosis and successful laparoscopic management of intramural pregnancy: case report**

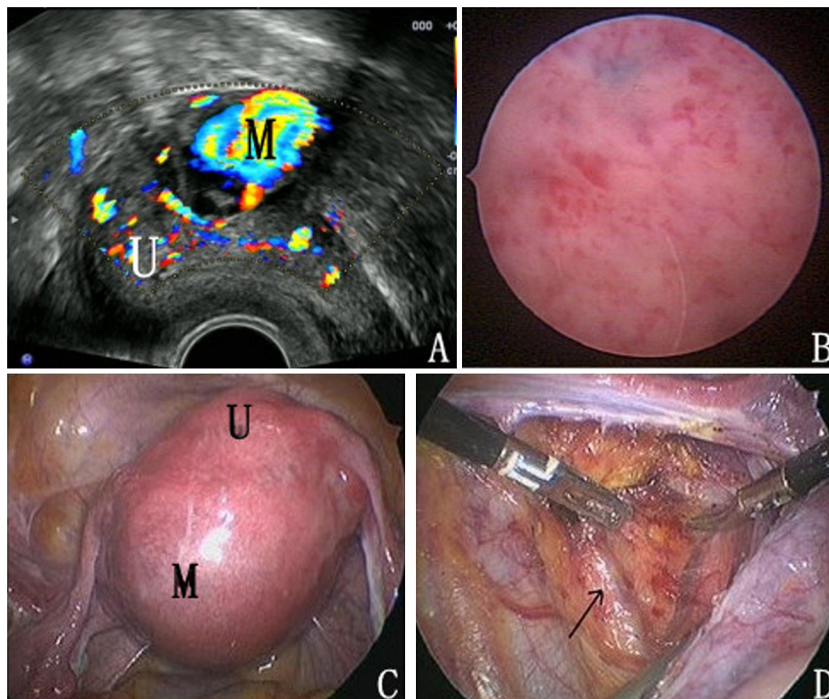

Dear Editor,

We would like to report a case of intramural pregnancy, diagnosed preoperatively. Laparoscopic unilateral uterine arterial occlusion was performed and the lesion was excised successfully without any complications.

A 34-year-old woman, gravida 2, para 1, presented to our gynecological clinic with lower abdominal pain and amenorrhoea for 57 days. She had a laparoscopic surgery performed due to endometriosis one year before. Diagnosed as “intrauterine pregnancy” at a local hospital, she underwent induced abortion, which failed. Gynecological examination revealed a slightly enlarged uterine body. The serum  $\beta$ -hCG concentration was 15000 mIU/ml. Transvaginal ultrasound scan revealed a less uniform myometrium echo and a heterogeneous echogenic area stretching to the serosa layer in the posterior uterine wall, measuring 3.7 cm  $\times$  3.3 cm. Doppler flow revealed high blood flow (Fig. 1A). Pelvic MRI showed an irregular-shaped signal within the myometrium in the left wall of the uterus. Around the lesion there

were multiple tortuous low signal flow-void vascular shadows. Hysteroscopy revealed an empty uterine cavity and thickened endometrium where a blue pigmented cyst could be seen (Fig. 1B). A diagnosis of suspected intramural pregnancy was made. Laparoscopy was therefore performed under general anesthesia. A bulging mass with an extremely thin serosa was seen arising from the left side of the posterior wall of the uterus (Fig. 1C).

The operation procedure was as follows: (1) the left uterine artery was separated from the anterior peritoneal layer of the left broad ligament and the main trunk was blocked using bipolar coagulation (Fig. 1D) in order to lessen the blood flow around the lesion; (2) 1 ml of vasopressin was then injected into the myometrium around the lesion; (3) a wedge-shaped incision was made over the bulging part of the posterior wall, and then dark reddish necrotic tissue suggestive of products of conception was carefully explored, confirming that it was connected neither with the uterine cavity nor with either of the fallopian tubes; (4) the lesion was removed and the defect was then repaired. The total operative time was 60 min, and the blood loss was 80 ml. Histopathology revealed villous and trophoblast cells in blood clots and the smooth muscle. Serum concentration of  $\beta$ -hCG was 643.45 mIU/ml on the 7th postoperative day and returned to normal on the 15th. Regular menstruation was achieved 7 weeks



**Fig. 1.** (A) Preoperative transvaginal ultrasound scan reveals a less uniform myometrium echo and a heterogeneous echogenic area stretching to the serosa layer in the posterior uterine wall with high blood flow. (B) Hysteroscopy reveals an empty uterine cavity with thickened endometrium. (C) Laparoscopic view shows a bulging mass with an extremely thin serosa arising from the left side of the posterior wall of the uterus. (D) The left uterine artery is blocked.

afterwards. No subsequent hemodynamic instability or hemorrhage was detected during the 3-months' follow-up, and ultrasonography at the end of the follow-up showed no change in the appearance of the uterus.

Intramural pregnancy is a rare form of ectopic pregnancy – a pregnancy implant within the myometrium, separate from the endometrial cavity and Fallopian tubes [1]. The exact cause is unclear. Possible risk factors include prior uterine trauma, adenomyosis, pelvic surgery, and in vitro fertilization [2]. As for this case, the previous history of surgery for endometriosis and the hysteroscopic findings indicated that the cause might be adenomyosis. In the past, the diagnosis could not be made until the time of surgery for uterine rupture [2]. In the present case, however, early detection was possible by liberal use of ultrasonography, MRI and hysteroscopy. A prompt and accurate diagnosis is crucial, avoiding complications such as uterine rupture, which is extremely dangerous and might require hysterectomy, resulting in subsequent loss of fertility [2]. As an ectopic pregnancy, treatment could be surgical, medical or expectant. For the medical approach, methotrexate comes first, and might even be confidently preferred before surgery in a ruptured ectopic with a hemodynamically stable patient [3]. For this case, the lesion size and  $\beta$ -hCG level indicated that it was in an active state, and the patient had the desire for future fertility, so we chose laparoscopic excision of the lesion. In the procedure, we occluded one uterine artery at the main trunk level first, which temporarily reduced the local blood supply and achieved satisfactory control of bleeding.

## References

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24 May 2013

<http://dx.doi.org/10.1016/j.ejogrb.2013.09.013>

## Euglycemic ketoacidosis in pregnancy and its management: case report and review of literature



Dear Editor,

Euglycemic ketoacidosis is an entity in which the blood glucose levels are within the normal range but the patient is ketotic with metabolic acidosis. This case consists of euglycemic ketoacidosis resulting from starvation in a previously healthy pregnant woman. We used fixed dose insulin for her successful management.

We conducted a review of the literature. Unfortunately there are not many cases with starvation ketoacidosis in pregnancy and their management is diverse.

## Case report

A 25-year-old, healthy nulliparous woman was admitted at 37 + 5 weeks with persistent vomiting and right sided loin pain. She was unable to tolerate any oral intake. On admission, examination was normal. Her urine dipstick showed glucose 1+ and ketones 3+ while her random plasma glucose was 5.7 mmol/L and ketones 2.7 mmol/L. Other blood results were: C-reactive protein (CRP) 14 mg/L, and leucocyte count  $15.2 \times 10^9/L$ . She was commenced on cyclizine and intravenous fluids.

She continued to vomit despite the above measures. Her CRP increased to 125 mg/L despite there being no infection. Her arterial blood gases (ABG) revealed severe metabolic acidosis. She was started on 10% dextrose but she still continued to deteriorate. A diagnosis of euglycemic ketoacidosis was made and the patient was monitored with ABG, blood ketones and blood glucose levels. She was commenced on 10% glucose at 125 ml/h in conjunction with Actrapid 7 ml/h. She showed quick recovery with a fixed dose regime of insulin rather than the older protocol of intravenous (IV) dextrose alone or sliding scale insulin. Once her bicarbonate levels were above 18 mmol/L and blood ketone levels were below 0.3 mmol/L, insulin was discontinued.

As her pregnancy was at term, a plan for induction of labour was made. Monitoring in labour would be as follows: (1) 10% glucose IV would be commenced to provide calories and prevent return to ketoacidosis; (2) hourly BM and ketones monitoring; (3) if her blood sugar levels on finger prick test were  $>7$  mmol/L or blood ketones  $>1.0$  mmol/L, fixed dose insulin would be started as per the diabetic ketoacidosis protocol; (4) following delivery, glucose  $\pm$  insulin infusion could be discontinued, but hourly blood sugar levels on finger prick and ketones for 12 h and then 4 hourly for 24 h would be monitored. If her ketone levels were above 0.3 mmol/L then venous blood samples would be checked. If her blood glucose was below 3.5 mmol/L, the diabetic team would be consulted.

She went into spontaneous labour and her ketones increased, and hence she was recommenced on 10% glucose at 125 ml/h and insulin at 5 units/h. She was started on IV syntocin (49 ml of Hartman + 10 units of syntocin at 0.6 ml/h) as there was no progression in labour. Cardiotocography (CTG) of the baby became suspicious with syntocin at 1.2 ml/h so it was stopped. After recovery with left lateral position, syntocin was started again at 0.6 ml/h and kept at the same rate until delivery. Because of a suspicious CTG after her cervix was fully dilated she was taken to theatre for trial of labour. A trial of forceps was successful and a healthy female child weighing 4140 g was delivered. Her Apgar scores were 6, 9 and 9 at 1, 5 and 10 min of life respectively. The mother's immediate postnatal period was uneventful.

Sliding scale insulin was started after delivery, with the patient receiving 7 units/h insulin and 10% dextrose at 125 ml/L, and care was given as planned. She went home the next day with an appointment for follow-up in the diabetic clinic in 6 weeks' time with an oral glucose tolerance test.

## Discussion

The patient denied any substance abuse. Diabetic ketoacidosis seemed unlikely as her blood sugar levels were normal (5.7 mmol/L) on admission. The patient had been unable to keep any food and water down so the diagnosis of starvation ketoacidosis was established.