CASE REPORT

Invasive Mole Presenting as Acute Haemoperitoneum

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Abstract

We report a case of invasive hydatidiform mole presenting as an acute primary haemoperitoneum. The patient presented with acute abdominal pain and signs of haemoperitoneum. Emergency laparotomy revealed a molar pregnancy perforating through the uterine fundus, resulting in massive haemoperitoneum. The serum beta chorionic gonado-tropin (ß-hCG) levels regressed spontaneously following evacuation of the molar pregnancy.

Key words

Molar pregnancy, Invasive mole, Haemoperitoneum

Introduction

Invasive mole follows approximately 10 to 15 percent of complete hydatidiform moles (1). They are characterised by the persistence of edematous chorionic villi with trophoblastic proliferation invading into the myometrium. The presence of villi in the trophoblastic tissue differentiates an invasive mole from choriocarcinoma. We describe a case of a invasive hydatidiform mole perforating through the uterus resulting in a massive haemoperitoneum.

Case Report

A 26-year-old female, presented to the emergency services of AIIMS Hospital, New Delhi with complaints of acute abdominal pain of 4 hours duration associated with vaginal bleeding. She had amenorrhoea of 8 weeks duration but her previous cycles were regular. Urine test for pregnancy was positive. On examination she was pale, hypotensive and had tenderness all over her abdomen. On bimanual pelvic examination the uterus was soft and bulky, fullness and tenderness could be elicited through all the fornices.

Following resuscitative measures she was taken up for an emergency laparotomy with a provisional diagnosis of a ruptured ectopic pregnancy. Abdomen was opened by a midline vertical incision and 2 liters of haemoperitoneum was evacuated. There was a profuse haemorrhage from the uterine fundus and the perforated area resembled trophoblastic tissue. The uterus was soft and enlarged to a size of 10 weeks pregnancy, both the adenxae were normal. Uterine evacuation was performed using a suction curette through the uterine fundus and local uterine wedge resection was performed to achieve homeostasis, uterus was closed with 1-0 vicryl. Intraoperatively patient received 2 units of packed cell transfusion and 20 units of continuous oxytocin infusion. During postoperative period she received 2 more units of blood transfusion and made an uneventful recovery.

The pathological diagnosis was invasive mole and microscopy showed the lesion was a complete hydatidiform mole. There was a transmural infiltration of the myometrium (Fig. 1).

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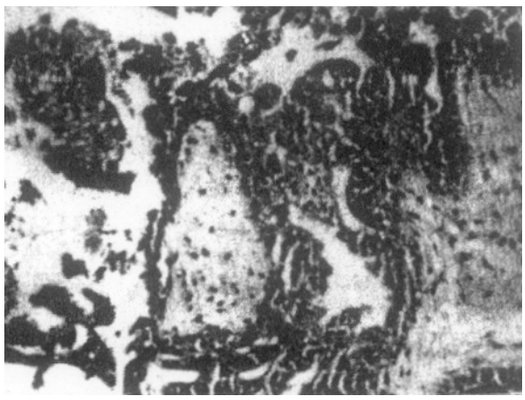

Fig. 1. Microphotograph showing chorionic tissue with marked proliferation of syncitial and cytotrophoblast.

The postoperative metastatic work-up including serum biochemistry, chest x-ray and upper abdominal ultrasonography did not reveal any evidence of metastasis. In view of low levels of serum ß-hCG, she was kept under surveillance with serial quantitative ß-hCG estimation to consider chemotherapy if ß-hCG levels remain in plateau or rise. Postoperatively ß-hCG level was 68 mIU/ml and it fell rapidly to 20 mIU/ml at one week and <5mIU/ml at two weeks following surgery. The subsequent ß-hCG levels were negative on regular follow up visits. Two years later she conceived spontaneously and had delivered a healthy baby at term by an elective cesarean section.

Discussion

Complete hydatidiform moles are recognized to have a potential for developing uterine invasion or distant metastasis. Invasive mole may perforate through the myometrium resulting in uterine perforation and intra-peritoneal bleeding (2). Direct vascular invasion and metastasis rarely occurs in invasive moles, the most common site reported is the lung (3, 4).

The diagnosis of invasive mole rests on the demonstration of complete hydatidiform mole invading the myometrium or the presence of villi in the metastatic lesion. Myometrial invasion is difficult to document on pelvic ultrasound and also in uterine curettings unless there is a sufficient myometrium to demonstrate the invasion.

Intra-operative management options are limited due to the acute presentation. Uterine evacuation is the treatment of choice in many circumstances but use of oxytocin to attain homeostasis is controversial. Mitani *et al* (5) recommended, partial resection for young women if invasive moles are complicated by internal haemorrhage. They have reported five women treated this way, four of which subsequently delivered healthy babies by caesarean section. Goldstein *et al* (6) used local uterine resection together with bilateral internal iliac artery ligation in an attempt to achieve haemostasis and preserve fertility. In our patient we also performed local uterine wedge resection following uterine evacuation to achieve haemostasis.

Use of chemotherapy in the management of invasive mole is debatable, with the evidence of spontaneous regression of metastatic mole in the literature (3, 4). We did not consider chemotherapy in our case as there was no evidence of metastasis and the ß-hCG levels were low and thereafter declined rapidly and became negative. Pronounced degenerative changes in the trophoblast along with hyalinization were found to correlate with low or declining levels of ß-hCG.

References

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