# Interesting case of invasive mole with hemoperitoneum

Savita Kamble, Tejaswini Kale (Pingle)

Correspondence: Dr Savita Kamble, Associate Professor in Obstetrics and Gynaecology, B J Government Medical College, Pune, Maharashtra. India; Email - savik05@yahoo.in

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#### **ABSTRACT**

Trophoblastic diseases comprise a variety of biologically interrelated conditions which form a clinical spectrum from benign partial hydatidiform mole at the one end to the highly malignant choriocarcinoma at the other without any precise line of demarcation. A case of invasive hydatidiform mole also called as Chorioadenoma destruens presenting as an acute primary haemoperitoneum. The patient presented in shock with acute abdominal pain and signs of haemoperitoneum. Emergency laparotomy revealed a molar pregnancy perforating through the uterine fundus, resulting in massive haemoperitoneum. Hysterectomy was done as a life saving procedure considering the patient in shock, massive hemoperitoneum and irreparable damage to the uterus.

**Keywords:** Molar pregnancy, invasive mole, haemoperitoneum, trophoblastic embolisation.

Gestational trophoblastic neoplasms (GTN) are proliferative as well as degenerative disorders of placental elements and include complete or partial mole (90%), invasive mole (5-8%), choriocarcinoma (1-2%) and placental site tumor (1- 2%). Fifteen percent of complete mole can develop into invasive mole. But only 2-4% of the partial mole transform into this variety of trophoblastic tumor [1]. The incidence of GTN varies in different regions from 0.6-1.1 per 1000 pregnancies in Europe and north America to 2 per 1000 in Japan and 1 in 160 in India and middle east [2]. Invasive mole follows approximately 10 to 15 percent of complete hydatidiform moles [3]. They are characterized 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.

## Case report

Twenty year old female married since 8 months was brought in casualty of Sassoon general hospital, tertiary hospital Pune, in a state of shock. Patient came with history of pain in abdomen and vomiting since 1 which increased in severity since 2 days. month, Patient also had per vaginal bleeding breathlessness since morning on same day. Patient was apparently alright 2 months back. She went to private practitioner for 2 months amenorrhea, underwent dilatation and evacuation. Histopathology report was suggestive of vesicular mole and β- HCG levels were 35,641mIU/ml so patient was advised follow up by private practitioner but she did not go.

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On admission her general condition was poor. She was afebrile, conscious and oriented with pulse rate of 160/min feeble in volume and blood pressure of 70 mm of Hg systolic and respiratory rate of 30/min. Patient had severe pallor, no cyanosis, clubbing, icterus, oedema, lymphadenopathy. Her respiratory system examination revealed tachypnoea but no crepitations or rhonchi. Her cardiovascular system examination revealed normal heart sounds and tachycardia. On per abdominal examination there was generalised tenderness all over abdomen with distension. There was demonstrable guarding and rigidity and bowel sounds could not be heard. Her per speculum



Fig 1: Perforative mole over serosal surface of uterus

examination revealed minimal bleeding. On per vaginal examination cervix was closed with tenderness and bogginess present all over fornices but exact uterine size could not be assessed. Patient was immediately resuscitated in view of shock with intravenous colloid support. Her emergency ultrasonography was showing moderate free fluid in abdomen with internal echoes and solid areas along peritoneum suggestive of haemoperitoneum with clots. Uterine size was  $12\times8.9\times8.6$  cm. Endometrial echo as well as myometrium was not appreciated, uterus completely replaced by echogenic lesions with cystic contents extending up to cervix and fundal part of uterus. Bilateral ovaries being bulky with multiple theca lutein cyst. On ultrasonography guided tapping there was

frank blood present. Her urine pregnancy test was positive and her haemoglobin concentration was 3 gm% with normal bleeding time and clotting time. Her  $\beta$  HCG on admission report of which was traced postoperatively was 1,27,515 mIU.

From history, examination and sonography report perforative vesicular mole haemoperitoneum suspected. Patient was was immediately shifted to operation theatre for emergency exploratory laparotomy after written informed valid consent in view of invasive mole, uterine perforation, need of multiple blood transfusion. hysterectomy and Medical Intensive Care Unit (MICU) admission with need of ventilators support if required. General anaesthesia was given. Abdomen opened by midline infra-umbilical incision .There was evidence of haemoperitoneum of 500 ml, which was drained and 800 gm clot present in bilateral paracolic gutters. Evidence of perforative mole over serosal surface of uterus at many sites which were bleeding actively (Fig 1). There was evidence of bilateral theca lutein ovarian cyst around 5×6 cm. The decision of hysterectomy was taken as a life saving procedure considering the patient in shock, massive hemoperitoneum and irreparable damage to the uterus. Total abdominal hysterectomy with preservation of both ovaries was done.

Intraperitoneal drain was kept after peritoneal wash. Abdomen closed in layers after confirmation of hemostasis. Intraoperative 3 units of PCV and



Fig 2: Gross cut section of uterus

postoperative 2 units of PCV and fresh frozen plasma were transfused. Postoperatively vital parameters of the patient were stable. Uterus in gross cut section was showing completely distorted anatomy with no differentiation between endometrium and myometrium and completely replaced by vesicular mole (figure 2).

Hysterectomised specimen was sent for histopathological diagnosis in which section through endo-myometrium was showing molar villi covered by proliferating trophoblast infiltrating myometrium. It was diagnostic of persistent trophoblastic disease which was invasive mole type.

On postoperative day 3 suddenly patient became breathless. On examination her general condition was moderate, afebrile with pulse rate of 140 per min and blood pressure of 110/70 mm of Hg. Her respiratory rate was 40 per min with bilateral crepitation and SPO<sub>2</sub> was 58 %. There was no pallor, cyanosis, icterus,



Fig 3: Chest X- Ray

clubbing, lymphadenopathy, oedema. On cardiovascular examination there was tachycardia. Her per abdominal examination revealed normal findings. There was no per vaginal bleeding. Her urine output was adequate and drain output was minimal. Patient was shifted to MICU on non-invasive ventilator support. She was given propped up position with nasal oxygen and started on inj. Furosemide 60 mg iv stat and, Inj. Piptaz 4.5 gm. 12 hrly iv. Patient was investigated as complete blood count, β HCG prothrombin time, D-dimer , Chest –x-ray, arterial

blood gas and CT pulmonary angiography. Her β HCG was still high 67,395 mIU and portable chest x-ray suggested multiple fluffy opacities (figure 3).

CT pulmonary angiography was suggestive of diffuse extensive patchy ground glass opacities with interlobular interstitial thickening with no e/o filling defect noted in pulmonary vasculature. In view of trophoblastic embolisation patient was started on chemotherapy after oncology opinion. So patient was given etoposide + cisplatin for 5 days and repeat chemotherapy on day 22. Patient was monitored with haemogram, creatinine and  $\beta$  HCG after chemotherapy was over. Her  $\beta$  HCG dropped to 2100mIU post chemotherapy first cycle and subsequently normal.

## 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 intraperitoneal bleeding [4]. Direct vascular invasion and metastasis rarely occurs in invasive moles, the most common site reported is the lung [5, 6]. 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 curetting unless there is a sufficient myometrium to demonstrate the invasion. Here we had good sonographic diagnosis of complete replacement of uterus by echogenic lesion with cystic areas and haemoperitoneum. For such cases of invasive mole complicated by internal haemorrhage. Mitani et al [7] recommended partial resection for young women. They have reported five women treated this way, four of which subsequently delivered healthy babies by caesarean section. Goldstein et al [8] used local uterine resection together with bilateral internal iliac artery ligation in an attempt to achieve haemostasis and preserve fertility. But in this case, we had to go ahead with hysterectomy considering irreparable uterine damage and hemodynamic instability of patient. Use of chemotherapy in the management of invasive mole is debatable, with the evidence of spontaneous regression of metastatic mole in the literature [5, 6]. We did consider chemotherapy in our case as there was evidence of lung trophoblastic embolism and the  $\beta$ -hCG levels were high postoperatively and also patient was symptomatic.

Trophoblastic pulmonary embolisation usually occurs following hysterectomy for invasive mole or evacuation of a molar pregnancy when the uterus is larger than dates and the human chorionic gonadotropin levels are more than 1 lakh. Differential diagnosis is usually pulmonary embolisation, transfusion related acute lung injury and aspiration. It has a dramatic onset with dyspnoea, tachypnoea, bilateral pulomonary infiltrates and low PaO<sub>2</sub> levels. Treatment requires supportive measures only and intubation is rarely required. The clinical course is short lived with gradual improvement after 48 hrs and complete resolution in 72 hrs.

Self limited respiratory distress arises in 3-10% following molar evacuation, with the number rising to 25% when the uterus is larger than dates and the human chorionic gonadotropin levels are more than 1 lakh mIU/ml [9].

### Conclusion

Hydatiform mole has a potential for myometrial and vascular invasion, leading to uterine perforation and massive internal hemorrhage which can be life threatening. Therefore, to avoid such adverse consequences it is necessary to identify such cases by early first trimester ultrasound and strict follow up where the diagnosis of persistent gestational trophoblastic disease can be done at the earliest.

Conflict of interest: None. Disclaimer: Nil.

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# Savita Kamble<sup>1</sup>, Tejaswini Kale(Pingle)<sup>2</sup>

<sup>1</sup>Associate Professor, Department of Obstetrics and Gynaecology, BJ Government Medical College, Pune, Maharashtra, India; <sup>2</sup>Assistant Professor, Department of Obstetrics and Gynaecology, BJ Government Medical College, Pune, Maharashtra, India.