BEDSIDE SONOGRAPHY PRIMER: Ultrasound Diagnosis of a Molar Pregnancy

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ABSTRACT
A case of a middle-aged female presenting with vaginal bleeding and elevated beta hCG (human Chorionic Gonadotropin). Transvaginal ultrasound images were consistent with a molar pregnancy. This case highlights the benefit of including emergency ultrasound in the clinical management of suspected Gestational Trophoblastic Disease (GTD).

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CASE PRESENTATION
A 48-year-old Asian female presented to the emergency department at approximately five weeks gestational age, by last menstrual period (LMP), with vaginal bleeding. One week prior she had passed a large clot with tissue. She continued to have cramping and bleeding over the next week, and saw her family doctor who found her to have an elevated beta hCG level. On emergency department presentation, her beta hCG level was measured at 305,533 mIU/mL. A transvaginal ultrasound revealed a “snowstorm” appearance, with clustered grape-like cystic structures within a hypoechoic uterus, characteristic of a hydatidiform mole.1 The patient was consented for suction dilatation and curettage and was taken to the operating room.

Figure 1. Sagittal view of the uterus. Note the grape-like cystic structures within the hypoechoic uterus (white arrow) and absence of fetal tissue. The transducer used for this image contains a damaged crystal resulting in a black streak artifact (white asterisk).

Gestational Trophoblastic Disease (GTD) encompasses four main pathologies: molar pregnancy (complete hydatidiform mole (CHM) and partial hydatidiform mole (PHM)), invasive mole, choriocarcinoma, and Placental Site Trophoblastic Tumor (PSTT). The term Gestational Trophoblastic Neoplasia (GTN) is a subset of GTD and refers to the latter three pathologies (invasive mole, choriocarcinoma, and PSTT). If left untreated, the mortality rate nears 100%.2

This patient received a diagnosis of GTD based on ultrasound imaging. There are two general types of molar pregnancy: complete hydatidiform mole (CHM) and partial hydatidiform mole (PHM). Complete moles are predominantly diploid androgenetic, from one haploid sperm that fertilizes an empty ovum (70-90%) and duplicates. Most PHMs are triploid, due to a normal egg that is fertilized by two sperm. CHMs tend to have higher hCG levels (>100,000 mIU/ml) than with PHMs (<100,000 mIU/ml). Another common differentiating factor between PHM and CHM is the presence of embryonic or fetal tissue. PHMs show clear signs of fetal or embryonic tissue, specifically the presence of villous blood vessels, proliferation of hyperplastic cytotrophoblast, and syncytiotrophoblast, while these are absent in CHMs.2 In this case, the patient’s hCG level and lack of fetal tissue led to a diagnosis of a complete mole.

In general, the incidence of molar pregnancy is 1 in 1500 live births.3 Several risk factors have been identified for molar pregnancy; age <20 or >40 nearly doubles the risk, prior molar pregnancy cause a 10-20 fold increase), previous spontaneous abortion can increase the risk by 2-3 fold, and diet that is low in animal fat and beta-carotene. In addition there appears to be significant regional variation: studies from North America, Australia, New Zealand, and Europe show an incidence between 0.57 to 1.1 per 1000 live births, while studies in Southeast Asia and Japan suggest an incidence as high as 2 per 1000 live births. Lastly, ovulation induction increases the risk of a combined pregnancy that consists of both a normal pregnancy and a molar pregnancy.2

The initial presentation for molar pregnancy usually consists of abnormal and sometimes massive uterine bleeding, as was seen in this case. However, due to elevated hCG, a patient may also present with acute hyperthyroidism, which can distract from a proper diagnosis.

Definitive treatment is operative removal of the pregnancy, usually with emergent dilatation and curettage, as patients may become unstable due to blood loss. A rapid diagnosis is therefore essential. While it is true that elevated hCG levels are an important finding of GTD, hCG levels alone can seldom differentiate a CHM from a normal intrauterine pregnancy or other pathologies. The diagnostic gold standard of GTD has traditionally been histological analysis. However, histological
morphology alone is not completely reliable and is only possible with tissue removed during surgery. Genetic testing can be used to differentiate between a diploid sample and triploid, but also requires surgical tissue sample. As such, ultrasonography greatly aids in the early diagnosis of molar pregnancy, and has significantly decreased the morbidity and mortality of this condition. The classic ultrasound finding is the characteristic “snowstorm” appearance and clustered grape-like cystic structures within a hypoechoic uterus, as was visualized in our patient.

Following dilation and curettage, post-operative complications can include toxemia, hyperthyroidism, and trophoblastic embolization. In particular, patients who are hyperthyroid secondary to the molar pregnancy must be managed carefully due to the increased risk of thyroid crisis during general anesthesia.

Once the mole is removed and the patient stabilized, the primary goals of treatment become prevention and early detection of a subsequent choriocarcinoma. In general, CHM’s are more associated with choriocarcinoma (15-20%) than PHM’s (1-5%). All patients treated for molar pregnancy should be monitored regularly for elevated hCG levels. Also, as the presence of one molar pregnancy elevates the risk of a subsequent one, it is important to counsel the patient to seek medical attention sooner rather than later in the event of vaginal bleeding or missed period.

In this case, the patient was found to have an invasive mole that was removed during surgery. This was confirmed on histologic diagnosis. She then underwent actinomycin D chemotherapy, followed by methotrexate. Her hCG level was monitored once per week and plateaued quickly once chemotherapy was begun. At her last follow-up, 6 months after her initial diagnosis, her beta-hCG level was >5.0 miU/ml.

REFERENCES