Invasive molar pregnancy and MRI: What every radiologist must know

Poster No.: C-1200
Congress: ECR 2010
Type: Educational Exhibit
Topic: Genitourinary - Female
Keywords: Gestational trophoblastic disease, Invasive Mole, MRI
Keywords: Genital / Reproductive system female, Obstetrics (Pregnancy / birth / postnatal period), Pelvis
DOI: 10.1594/ecr2010/C-1200

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Learning objectives

• To review the relevant pathophysiology and natural history of malignant Invasive Mole within the spectrum of Gestational Trophoblastic Disease (GTD)

• To depict relevant MR imaging features of GTD in the initial exam and follow-up.

• To emphasize in the inestimable use of MRI in detection of myometrial tumoral invasion and assessment of extrauterine spread.
Background

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1. THE SPECTRUM OF GESTATIONAL TROPHOBLASTIC DISEASE

1.1 Definition and introduction

The term Gestational Trophoblastic Disease (GTD) describes a spectrum of placental lesions that arise from pregnancy, characterized by abnormal trophoblastic proliferation, that vary in their clinicopathological behaviour.

GTD can be divided in two main groups, the benign form of GTD, Hydatidiform Mole, a localized and benign disease that usually resolves with uterine evacuation (although around 10% develop Persistent Gestational Trophoblastic Disease [PGTD]), and malignant forms of GTD, called Persistent Gestational Trophoblastic Disease (PGTD) or Gestational Trophoblastic Neoplasia, which can be non-metastatic (Invasive Mole) or metastatic (Choriocarcinoma). There is another form of PGTD, which is the least common, called Placental Site Trophoblastic Tumor (PSTT).
GTD classification (Fig 2) on page 14 - Benign Gestational Trophoblastic Disease: Hydatidiform mole

- Complete Hydatidiform Mole
- Partial Hydatidiform Mole

- Malignant Gestational Trophoblastic Disease: Persistent Gestational Trophoblastic Disease or Gestational Trophoblastic Neoplasia

- Non-metastatic: Invasive Mole
- Metastatic: Choriocarcinoma
- Placental Site Trophoblastic Tumor

GTD can be **early detected** by increased levels of a hormonal marker, $\beta$-HCG, produced by trophoblast cells, that is very important in the diagnosis as well as in follow up.

**Clinical manifestations, sonography, $\beta$-HCG and pathologic findings permit the diagnosis of GTD.** It is important to remark the need of a systematic pathologic diagnosis in every non-evolutive pregnancy, as a warranty of the diagnosis of GTD.

After treatment of an Hydatidiform Mole, $\beta$-HCG levels usually decrease progressively. When high $\beta$-HCG titres persist following evacuation of a mole, the patient is treated with chemotherapy without further pathologic diagnosis. Such a case is diagnosed as **PGTD**.

Imaging has an important role in GTD diagnosis and follow up. Initially, US can exclude normal intrauterine pregnancy, gemellar pregnancy or ectopic pregnancy. CT can be used to asses metastasis, and also to asses parametrial invasion. But neither US and CT are sensitive to locally invasive disease, and this is the most important role of MRI.

**1.2 Epidemiology**

The prevalence of GTD depends on geography, and for unknown reasons, varies with race. It is more common in Asian and Latin America with rates of 1/240-400 pregnancies, than in Occident, with rates of 1/1000-2000 pregnancies.
Maternal age is a risk factor: mothers over 35 years and under 20 years have higher risk. Previous molar event and spontaneous abortions also increase the risk.

1.3 #-HCG

#-HCG serum level is a sensible indicator of the presence of the disease, and is useful in the follow up and for planning the therapy.

The #-HCG is an hormone produced by syncytiotrophoblast, detected in plasma and urine within the 9 days of conception. Normally, levels of #-HCG arise reaching a peak at 9-12 weeks, with 100.000 mUI/ml and declining to a plateau until birth. Levels of # 200.000mUI/ml are very suggestive of the GTD.

The function of #-HCG is maintaining the corpus luteum, and stimulating ovarian production of progesterone and estrogens. When the placenta begins to produce these steroids, the secretion of #-HCG declines. Therefore, #-HCG concentration in the urine or serum is directly related to the number of viable trophoblastic cells, which are increased in GTD.

2. PATHOPHYSIOLOGY AND NATURAL HYSTORY OF GTD

2.1 Normal embryonic development and placental formation (Fig 3) on page 14

A brief review of the normal embryonic development and placental formation is useful to understand the pathophysiology of the disease.

The process is divided in several phases.

- **Day 1. Fertilization** is the fusion of genetic material from sperm and ovum into a single nucleus, and formation of the zygote. It takes place in the ampullary portion of the tuba.

- **Day 2. Cleavage** are the early series of mitotic divisions of the zygote, in which the number of cells increase quickly, and the cells become smaller.
**Day 3. Morula** (from latin, *morum* from its resemblance with a mulberry) is the name of the zygote after 3 or 4 divisions, it is a solid ball of cells. In this state the morula is about to enter the uterine cavity.

**Day 5. Blastocyst** is a hollow ball of cells surrounding a central cavity, with an outer covering of cells, *trophoblast* that will become the *chorion*, which forms de fetal portion of the placenta, and an *inner cell mass*, which will contribute to the formation of the embryonic body. *Blastocel* is the internal fluid-filled cavity.

**Days 6-7. Implantation** is the adhesion of the blastocyst to the endometrium with the inner cell mass oriented toward the endometrium (embryonic pole); it penetrates into endometrium to get blood supply and nutrients.

**Day 9** the blastocyst is buried into endometrium. The trophoblast develops, specially at embryonic pole.

The trophoblast has three components:

- **Cytotrophoblast**: a stem cell with high mitotic activity without hormonal function.
- **Syncytiotrophoblast** has hormonal activity, syntesis of beta subunit of human chorionic gonadotropin (#-HCG).
- **Intermediate trophoblast**: takes part in endometrial invasion and implantation.

**Second month**, trophoblast develops a large number of villi in the embryonic pole, called *corion frondosum*, and has a radial appearance.

**Third month.** Development of the placenta is complete.

**Fourth month.** The placenta is formed by two portions.

- **Chorion** is the *embryonic (fetal) portion* of the placenta. The *chorionic villi* are finger-like structures covered by trophoblast, that containing fetal arterial plexuses supplied by umbilical artery, that extend into *intervillous spaces* (maternal blood sinuses), where they are immersed in maternal blood. They provide the surface for the exchange of oxygen and nutrients with the maternal circulation.
• The maternal portion of the placenta is the endometrium just beneath the blastocyst called the decidua basalis, which lines the intervillous space. This decidual changes have been induced by the fetal trophoblastic invasion of the endometrium.

Placenta functions are:

- Diffusion of nutrients and oxygen from maternal blood, toxin excretion.

- Protective barrier against most microorganisms.

- Hormone secretion.

2.2 Benign gestational trophoblastic disease

The term "hydatiform" comes from the cystic appearance of moles, that resemble to hydatid cyst in an Echinococcosis. Hydatid derives from hydatis (Greek "a drop of water"), referring to the watery contents of the cysts. Mole comes from mola (Latin = millstone/ false conception).

It represents the 80% of all GTD. It is a common complication of gestation, that occurs in one of 1000-2000 pregnancies in US.

There are two types of Hydatidiform Mole, with different histologic and genetic features: Complete form, the commonest, in which no fetus is formed, and Partial form, with an abnormal or demised fetus. A rare type is the coexistence of a mole and a normal pregnancy (normal fetus and placenta).

It is a non invasive process characterized by proliferation and hydropic swelling of the villi, that have prominent central acellular space, that macroscopically correlate with the fluid filled vesicles.

Although some grade of myometrial invasion may exist in the Hydatidiform Mole, the evacuation of a molar gestation has a cure rate between 70-90%.
The diagnosis usually is done after uterine aspiration after a spontaneous incomplete abortion. After that, patient must be monitorized with serial #-HCG levels. If #-HCG levels persist high, the patient is diagnosed of PGTD. (Fig 4) on page 15

In the next lines, we briefly resume and compare the most important findings of Partial and Complete Hydatidiform Mole.

**Complete Hydatidiform Mole** (Fig 5) on page 16

**Pathology.** (Fig 6) on page 17 Enlargment and hydropic transformation of all villi, trophoblastic proliferation (hyperplasia); complex multicystic mass, classically "bunch of grapes" appearance. Absent embryo or fetus and absent gestational sac.

**Gametogenesis.** Diploid karyotype, that derives from sperm. Ovocyte has lost its material. The embryo is lost early. Uncommon coexistence of fetus.

**Symptoms.** Spontaneous abortion of 2\textsuperscript{nd} trimester, uterus large for date or rapidly enlarging, strong bleeding, preeclampsia.

**Imaging**

- **US** (transabdominal, transvaginal) Uterine enlargement. Ecogenic central uterine mass, "snowstorm" or "granular" appearance, caused by anecoic or cystic spaces (hydropic villi). Nonspecific appearance, incomplete miscarriage can appear identical. Normal interface between abnormal trophoblastic tissue and myometrium. No identifiable fetal tissue or gestational sac is seen. Teca-lutheinic ovarian cysts due to hyperstimulation by BHCG. *(Fig 7 on page 18 and 8 on page 19)*
- **MR** Not used in routine. Asses extension into myometrium or extrauterus. Nonspecif findings, can mimic retained products of conception (RPOC).

**Therapy** Mole evacuation

- In woman < 40 years with gestational wish, dilatation and suction curettage (permit microscopic diagnosis).
- In selected cases (>40 years, no wish to conserve fertility, uterine pathology associated, risk of uterine perforation or uncontrollable bleeding) abdominal hysterectomy with adnexal preservation is an alternative therapy. Hysterectomy reduces the risk of developing PTGD but a risk of 3-5% remains.

**Follow-up** with serial mesurements of #-HCG.
Higher risk (15%) in developing persistent trophoblastic disease (10% of patients will develop invasive mole, 5% choriocarcinoma)

Partial Hydatidiform Mole (Fig 9) on page 20

Pathology. Normal and hydropic villi. Less trophoblastic proliferation (hyperplasia) and less tumoral volume than complete form. Abnormal fetal tissue. (Fig 10 on page 21 and 11 on page 22)

Gametogenesis. Triploid karyotype with maternal and paternal genetic material (one ovum with two sperm). The fetus is also tryploid and has severe abnormalities.

Symptoms. Spontaneous abortion of 1st trimester, uterus small for age, moderate bleeding

Imaging

• US (transabdominal, transvaginal): fetus with severe structural abnormalities, grow restriction, oligoamnios. Placenta containing numerous cystic spaces (hydropic villi).
• MR. Not used in routine; assess extension into myometrium or extrauterus; nonspecific findings, can mimic retained products of conception (RPOC)

Therapy. Mole evacuation

• In woman < 40 years with gestational wish, dilatation and suction curettage (permit microscopic diagnosis). If fetal tissue prevents suction and curettage, medical abortion can be indicated.
• In selected cases (>40 years, no wish to conserve fertility, uterine pathology associated, risk of uterine perforation or uncontrollable bleeding) abdominal hysterectomy with adnexal preservation is an alternative therapy. Hysterectomy reduces the risk of developing PTGD but a risk of 3-5% remains.

Follow-up with serial measurements of #-HCG.

Lower risk (<5%) than Complete Hydatidiform Mole in developing Persistent Gestational Trophoblastic Disease.
2.3 Persistent Gestational Trophoblastic Disease

Persistent Gestational Trophoblastic Disease can occur in persistent or rising #-HCG levels in absence of a pregnancy, after the following situations:

- evacuation of a benign Gestational Trophoblastic Disease (60%)
- abortion (30%)
- normal or ectopic pregnancy (10%)

PGTD diagnosis is based in #-HCG serum levels, and hystologic or radiologic evidence is not indispensable.

**Work-up** in patients with PGTD include: physical examination, weekly of #-HCG levels, hematologic and serum chemistry, as well as radiologic studies, that may include radiologic studies. This work-up permits a clinical staging, which is important in the disease management. *(Fig 13) on page 24*

Several systems have been used to **classify the severity of PGTD**. Recently a new International Federation of Gynaecologists and Obstetricians (FIGO) **scoring system** has been developed and most centers use this to enable better comparison of patient response and outcome. With this scoring systems patients are divided in two therapeutic groups, those with low FIGO score (# 6 ) have a low risk of developing disease resistant to single drug therapy, while those with high FIGO score (>6) require multiagent combination therapy. *(Fig 14 on page 25, 15 on page 26 and 16 on page 27)*

**Invasive mole (Fig 17) on page 28**

A Complete or Partial Hydatidiform Mole that invades the myometrium is termed invasive mole. It is **locally aggressive and invasive, but rarely metastasizes**.

**All cases of invasive mole are sequelae of hydatidiform moles.** It can be diagnosed on US or on a rising of #-HCG after uterine evacuation.

Patient with persistent or rising of #-HCG serum levels after treatment of complete hydatidiform mole presumably has an invasive mole unless there are clinical and/
or radiologic evidence for metastasis, which would traduce the existence of choriocarcinoma.

Approximately 15% of complete moles are associated with or precede invasive moles. The pathologic diagnosis of invasive mole is rarely made because most cases are treated medically, without hysterectomy.

**Pathology.** Hydropic villi and trophoblast proliferation and penetration into the myometrium. Macro and microscopic invasion into myometrium and blood vessels. *(Fig 18) on page 29*

**Imaging**

- **US.** Central uterine mass similar and indistinguishable to complete hydatidiform mole; sometimes demonstrable myometrial invasion. Mass shows very high blood flow, specifically high diastolic flow, result of decreased vessel tone in the proliferation neoplasm. *(Fig 19 on page 30 and 20 on page 31)*
- **MR.** Heterogeneous, hypervascular mass that distort the normal zonal anatomy; extraterine extension seen as abnormal signal intensity in myometrium or parametrium.

**Therapy.** Chemotherapy in the majority of cases. Hysterectomy if invasive disease to prevent uterine perforation in selected patients with no wish to conserve fertility.

**Choriocarcinoma (Fig 21) on page 32**

Very aggressive malignancy with high metastatic risk.

- 50% of choriocarcinoma arise from complete hydatidiform mole; this group have a good response to chemotherapy and have a better prognosis than other groups of risk (may be secondary to a careful follow up that these patients undergo).
- 25% arise from normal pregnancies
- 25% develops after spontaneous abortion or ectopic pregnancy

**Pathology.** Extensive necrosis and hemorrhage. Absence of villi is very important and diagnostic, and helps in the differential with other forms of GTD. Early and extensive vascular invasion, that result in metastatic disease. **Metastasis** are hematogenous, and are frequent in lungs, vagina, brain, and less common in vulva, kidneys, liver, ovaries, and bowel.
Imaging

- **US.** Central uterine mass, heterogeneous due to areas of extensive necrosis and hemorrhage; sometimes demonstrable myometrial invasion. Mass shows very high blood flow.
- **MR.** Heterogeneous, hypervascular mass that distort the normal zonal anatomy; extrauterine extension seen as abnormal signal intensity in myometrium or parametrium.
- **CT.** Chest CT and head CT to detect metastasis.

**Therapy.** Chemotherapy in the majority of cases. Hysterectomy if invasive disease to prevent uterine perforation in selected patients with no wish to conserve fertility.

**Placental site trophoblastic tumor**

It is composed of intermediate trophoblastic cells, that proliferate and form a mass. These cells have a role in establishing uteroplacental circulation. The tumor is usually confined to uterine corpus but may metastasize, and can invade lymph nodes (uncommon in other types of GTD).

PSTT is typical of women of childbearing age. It may develop after normal pregnancy, abortion or molar pregnancy. It presents with vaginal bleeding.

Because it contains small quantity of syncytiotrophoblastic tissue, **#-HCG are normal or mildly elevated.** Instead, the cell tumor secretes human placental lactogen. Unlike choriocarcinoma, it has a lower sensitivity to chemotherapy.

MR imaging findings are nonspecific, and similar to other types of PGTD, and MR is useful to depict location, size and extent of the tumor. The diagnosis is made with biopsy.

This tumor is considered a different entity, and is not classified under the FIGO staging system.

PSTT is relatively **chemoinsensitive**, so when the disease is localized to the uterus hysterectomy is recommended.

2.4 Visual overview of GTD pathophysiology (Fig 22) on page 33
Fig 2. GTD classification

**Benign Gestational Trophoblastic Disease**
- Complete Hydatidiform Mole
- Partial Hydatidiform Mole

**Malignant or Persistent Trophoblastic Gestational Disease (PGTD)**
- Non-metastatic Invasive mole
- Metastatic
  - Choriocarcinoma
  - Placental site trophoblastic tumor (PSTT)

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Fig. 0

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Fig. 0

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Fig. 6. Complete hydatidiform mole

Photomicrograph (H-E stain). Diffuse villous enlargement, with round borders, central cysterns (*), circumferential trophoblast proliferation (black arrows) and atypia.

Fig. 0

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Sagittal and axial images from transvaginal ultrasound show enlarged uterus with an heterogeneous echotexture. A central uterine mass is seen (arrows), with numerous small anechoic spaces. No identifiable fetus. The endometrium cannot be identified. These findings are consistent with an hydatidiform mole.

**Fig. 0**

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Fig 8. US Hydatidiform Mole

Sagital images from transvaginal ultrasound show enlarged uterus with a large central uterine mass is seen (arrows). No identifiable fetus was seen. Note the intense power Doppler enhancement of the mass.

Fig. 0

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Fig. 9. Partial Hydatidiform mole

<table>
<thead>
<tr>
<th>Partial Mole</th>
<th>69, XXY or 69 XXX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetus Present</td>
<td></td>
</tr>
<tr>
<td>Villous edema</td>
<td>Variable</td>
</tr>
<tr>
<td>Trophoblast proliferation</td>
<td>Focal, moderate</td>
</tr>
<tr>
<td>Presentation</td>
<td>Abortion</td>
</tr>
<tr>
<td>Uterus</td>
<td>Small for gestation age</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Moderate</td>
</tr>
<tr>
<td>Teca-luteinocyst</td>
<td>Rare</td>
</tr>
<tr>
<td>Complication</td>
<td>Rare</td>
</tr>
<tr>
<td>PGTD</td>
<td>&lt;5%</td>
</tr>
</tbody>
</table>

Fig. 0

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Fig. 10. Partial hydatidiform mole

Photomicrograph (original magnification 15x, hematoxylin-eosin [H-E] stain). Extensive villous enlargement (black arrows) and scattered normal villi (green arrows).

Fig. 0

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Fig 11. Partial hydatidiform mole

Photomicrograph (original magnification 80x, H-E stain). Enlarged villi with scalloped borders, and moderate, circumferential trophoblast proliferation {green arrows}. Scattered normal villi can also be seen (black arrows).

Fig. 0

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### Fig 12. Partial Mole versus Complete Mole

<table>
<thead>
<tr>
<th></th>
<th>Partial Mole</th>
<th>Complete Mole</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cariotype</strong></td>
<td>69, XXY or 69 XXX</td>
<td>46, XX or 46, XY</td>
</tr>
<tr>
<td><strong>Pathology</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Fetus</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>• Villous edema</td>
<td>Variable</td>
<td>Diffuse</td>
</tr>
<tr>
<td>• Trophoblast proliferation</td>
<td>Focal, moderate</td>
<td>Diffuse, severe</td>
</tr>
<tr>
<td><strong>Clinical presentation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Diagnosis</td>
<td>Abortion</td>
<td>Molar pregnancy</td>
</tr>
<tr>
<td>• Uterine volume</td>
<td>Small for gestation age</td>
<td>Large for gestation age</td>
</tr>
<tr>
<td>• Vaginal bleeding</td>
<td>Moderate</td>
<td>Heavy</td>
</tr>
<tr>
<td>• Teca-lutehnic cyst</td>
<td>Rare</td>
<td>15-25%</td>
</tr>
<tr>
<td>• Medical Complications</td>
<td>Rare</td>
<td>&lt;25%</td>
</tr>
<tr>
<td><strong>PTGD</strong></td>
<td>Approximately &lt;5%</td>
<td>Approximately 20%</td>
</tr>
</tbody>
</table>
Fig 13. Work-up for PGTD staging

- History and physical examination
- Weekly β-HCG titers
- Hematologic and serum chemistries
- Chest film (or thorax CT if metastasis are suspected)
- Brain MRI or CT
- Liver US or abdominal CT
- **PELVIC MRI**
- PET if the latter exams do not detect M1 and HCG is high.

*Source: SEGO*

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**Fig. 0**

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Fig. 0

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Fig 15. STAGING

- **Stage I**: PGTD strictly limited to the uterine body
- **Stage II**: PGDT extended to adnexa and vagina, but limited to genital structures
- **Stage III**: PGTD extended to lungs, with or without genital involvement.
- **Stage IV**: metastasis in other locations different to lungs

Additional note: *Clinical staging in PGTD recommended by FIGO on 2000.*
Fig. 0

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Fig 17. Invasive Mole

Invasive Mole

- All from Hydiliform Moles
- Locally Aggressive
- Low metastatic risk
- Penetrates into the myometrium
- Hydopic Villi and trophoblast proliferation
- Highly Chemosensitive

Fig. 0

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2. PATHOPHYSIOLOGY AND NATURAL HISTORY OF THE GTD

Fig 18. Invasive mole

Photomicrograph (original magnification x12, H-E stain). Dilated villi surrounded by hyperplastic trophoblast (black arrows) invading through the myometrium (green arrows).

Fig. 0

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Sagittal transvaginal sonographic view shows an hypervascular myometrial mass. Sagittal T2 WI shows a well defined round mass with a hypointense halo, within the anterior myometrial wall. Note myometrial diffuse hypersignal and loss of the normal anatomy zone.

Fig. 19. US with MRI correlation

Fig. 0

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Abnormal pulsed wave Doppler sampling in a patient with active PGTD at initial diagnosis and one month later after chemotherapy shows low impedance waveforms and marked myometrial color flow Doppler signal.
Fig. 21. Metastatic PGTD: Choriocarcinoma & PSTT

Metastatic PGTD

Choriocarcinoma >>> PSTT

- 50% from Complete Mole
- 25% Normal Pregnancy
- 25% Spontaneous abortion or Ectopic pregnancy

Very Agressive
High Metastatic Risk
Absence of Villi in Choriocarcinoma
Intermediate Trophoblastic cells in PSTT
High vascular invasion in Choriocarcinoma

Chemosensitivity
Choriocarcinoma >>> PSTT

Fig. 0

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Fig. 0

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3. MRI IN GTD

With its excellent soft tissue resolution, MRI is able to demonstrate the boundaries of myometrium and tumor involvement more accurately than any other imaging technique and is useful in the assessment of extrauterine disease in the persistent gestational trophoblastic disease. In this work we will focus on the role of MRI in Invasive Mole.

MRI is also useful when there is tumoral myometrial extension and scarce endometrial extension, where uterine curettage may not be diagnostic. It is used as a presurgical map.

Pelvic MRI can exclude uterine source for elevated #-HCG in extrauterine germ-cell tumors or occult neoplasm, specially when there is no response to medical treatment.

3.1 MRI protocol (Fig 1) on page 38

We perform MR imaging with 1.5-T unit (General Electric). A phased array coil is used.

Our standard pulse sequences and planes for MR imaging of GTD are:

- Scout: axial, coronal and sagital

1-Axial of the pelvis: T1-WI (SE Respiratory compensation or FSE) and Fat Saturation T2 (FSE). Slice thickness: 8mm. Include the whole pelvis.

2-Sagital of pelvis: T2-WI (FSE). Slice thickness: 4mm. Including the whole uterus and sacrum

3-Axial to corpus uteri: T2-WI and Fat Sat T2 (FSE) Slice thickness: 3mm.

4-Coronal to corpus uteri: T2-WI (FSE) Slice thickness slice: 3mm.

5- Fat Saturation T1-WI (FSE or FSPGR, breath hold) precontrast and dynamic postcontrast. Axial to corpus uteri and delayed sagital postcontrast.

3.2 MR findings of GTD
MRI relevant imaging aspects/checkpoints in initial imaging are:

- Uterine volume (in three dimensions)
- Tumor location and extent
- Visibility of normal uterine zonal anatomy (endometrium, myometrium, junctional zone)
- Tumor MR signal
- Boundaries between tumor and myometrium
- Tumoral vascularity and state of arterial and venous structures (engorgement of hypogastric, adnexal and uterine vessels)
- Extratumor extension beyond myometrium to peritoneum, extension to adnexa
- Presence and characterization of adnexal cysts.
- Pelvic metastasis

**Hydatidiform Mole**

Not routinely used

- Nonspecific findings, can mimic RPOC
- Heterogeneous mass in uterine cavity, low signal intensity on T1, high signal intensity on T2, strong contrast enhancement. Focal areas of hemorrhage and cystic spaces. The mass is surrounded by normal myometrium, seen as an hypointense layer. Fetal tissue with structural abnormalities and growth restriction in partial mole.

**Invasive Mole and Choriocarcinoma**

These two entities are indistinguishable at imaging

- Intrauterine mass, heterogeneous signal on T2 images; may show necrotic or hemorrhagic zones (Fig 2) on page 38. As an hypervascular tumor, has avid contrast enhancement, that represents areas of viable tumor (Fig 3 on page 39 and 4 on page 40)
- Signal voids representing dilated vessels within the tumor, in the myometrium and parametrium (Fig 5) on page 41
- Myometrial invasion is seen as high signal areas within the myometrium, distorting the junctional zone. These areas enhance after contrast administration. (Fig 6 on page 45 and 7 on page 44)
- Indistinct boundaries between tumor and myometrium (Fig 8) on page 43. Presence and size of teca-lutheinic cysts (Fig 9) on page 42
- Parametrial spread and metastatic pelvic disease, seen as high signal and enhancing areas.
• Enlarged pelvic lymph node

MR imaging may resemble RPOC, but BHCG levels help distinguishing GTD from RPOC, where hormonal levels are normal or slightly elevated.

3.3 Post-chemotherapy findings

MRI indications during chemotherapy are the absence of decrease of the BHCG serum levels, but can also be used to assess tumoral response. MRI findings after therapy depend on the chemotherapeutic cycle and response.

- Progressive recovery of normal uterine zonal anatomy (Fig 10) on page 46
- Decreased uterine and adnexal vascularity
- Development of intralesional hemorrhage (Fig 11) on page 47
Return to normal uterine volume (Fig 12 on page 48 and 13 on page 49)

After treatment patients may develop vascular abnormalities, associated with residual heterogeneous scarring. These uterine vascular malformations can appear as residual tortuous and coiled vessels within a thickened myometrium.
Fig. 0

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3. MRI IN GTD

Fig 2. Myometrial mass

Sagittal and Coronal T2WI. Right parasagittal myometrial mass with heterogeneous T2 signal.

Myometrial mass, heterogeneous signal on T2 images

Fig. 0

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Fig. 0

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Fig. 4. Dynamic post-contrast MRI

Sagittal Fat saturated T1 before and after contrast administration. See the early intense contrast enhancement of the mass, and good tumor-myometrial contrast. Compare another patient in late dynamic phase, in which myometrial enhancement masks tumoral enhancement.

Dynamic contrast enhanced MRI permits a good tumor-myometrial contrast on early dynamic phase. Enhancement areas relate to the amount of active trophoblastic tissue and correlate with BHCG levels.

Fig. 0

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Coronal to uterus T1 Fat Saturated Contrast enhanced and Sagital T2 WI, shows ill-defined enhancing parasagital mass, with hypointense areas in all sequences which represent vascular flow voids. Note large teca-lutein cysts (arrowheads). See a small intramural myoma in the anterior uterine wall ( ).

**Signal voids** representing dilated vessels within the tumor

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**Fig. 0**

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Fig. 9. Teca-lutheinic cysts

Axial T2 Fat saturation and Sagital T2WI shows large teca-lutheinic cysts in patient with invasive mole.

Presence of large teca-lutheinic cysts

Fig. 0

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3. MRI in GTD

Fig 8. Indistinct margins

Sagittal T2WI of different patients. See heterogeneous myometrial ill defined masses.
See teca-luteinic cysts (arrowheads).

Indistinct boundaries between tumor and myometrium

Fig. 0

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3. MRI IN GTD

Fig 7. Diffuse myometrial T2 WI hypersignal

Sagital T2 WI in a patient with a PGTD. See myometrial hyperintense mass and a diffuse increased myometrial signal, and loss of normal zonal architecture. Compare this findings with Sagital T2 WI of a same patient.

**Diffuse increased myometrial signal and loss of normal zonal architecture on T2 WI, may reflect diffuse myometrial tumoral involvement, but it is nonspecific**

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Fig. 0

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Coronal to uterus T2 WI. Ill-defined myometrial mass, partial junctional zone disruption (area between arrow that shows subtle hypersignal). Hyperintense well defined cystic focus suggestive of necrotic or hemorrhagic changes within the mass.

Myometrial invasion is seen as high signal areas in T2 within the myometrium, distorting the junctional zone

Fig. 0

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3. MRI IN GTD

Fig 10. Chemotherapy positive response

Axial STIR before and after chemotherapy. Good response with decreased uterus size, decreased uterine vascularity and no visible tumor in follow-up MRI. BHCG levels had significantly decreased.

Recovery of normal uterus size, decreased uterine vascularity, recovery of the normal MR zonal anatomy

Fig. 0

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Fig. 11. Chemotherapy undeterminate changes

Sagittal T2 WI before and one month after chemotherapy. Although BHCG levels had significantly decreased, the patient was reimaged because transvaginal US showed no significant changes. MR showed a decreasing in uterus size, recovery of normal myometrial signal but no changes in tumor size. However, tumor showed higher central signal intensity, a thicker hipointense halo and morphologic changes.

Recovery of normal uterus size and myometrial signal intensity, and undeterminate changes in tumor MR appearance

Fig. 0

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Fig 0

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Transverse transvaginal sonographic view with transverse T2 WI correlation shows an ill defined myometrial mass. See hypoechoic areas within the mass, which may represent necrosis, and anechoic tubular left parametrial structures that represent tumoral vascularity seen as signal voids in MRI.
Conclusion

- MRI is an inestimable tool for detecting myometrial tumoral invasion and extrauterine spread in invasive mole disease.

- Knowledge of the pathophysiology and natural history of the disease, and familiarity with typical imaging characteristics as described in this work allow to make a confident diagnosis.
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References


