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Case Report

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Invasive hydatidiform mole with pulmonary and vaginal metastases: A rare case with MRI and CT findings

J Boutaleb[1]*; H Riache[1]; S Loubaris[1]; B Beqqali[1]; R Alem[2]; H Errihani[2]; N Allali[1]; L Chat[1]; S El Haddad[1]; E Manal[3]; L Najat[3]

[1]Radiology Department, Pediatric Hospital, Chu Ibn Sina Rabat, Morocco.

[2]Dermatment of Oncology, National Institute of Oncology, Chu Ibn Sina, Morocco.

[3]Department of Anatomopathology, Rabat Children's Hospital, Faculty of Medicine and Pharmacy, Mohammed V University Rabat, Morocco.

***Corresponding Author: Boutaleb Joud**

Radiology Department, Pediatric Hospital, Chu Ibn Sina Rabat, Morocco.

Email: joud.boutaleb@hotmail.fr

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Abstract

We present a rare case of invasive hydatidiform mole (IHM) in a 52-year-old woman with persistent vaginal bleeding and elevated β-hCG following evacuation of a complete hydatidiform mole. Imaging revealed a large uterine lesion with myometrial invasion, vaginal extension, and pulmonary and vaginal metastases. This case highlights the importance of imaging in detecting local invasion and metastases in IHM. Early diagnosis and appropriate chemotherapy remain key to managing this aggressive condition, even when metastases are present.

Introduction

Gestational Trophoblastic Neoplasia (GTN) encompasses a heterogeneous group of malignant disorders with distinct pathogenesis, morphological characteristics, and clinical presentations. These conditions can arise following any type of pregnancy. Among them, invasive mole is the most common form, characterized by myometrial and/or lymphovascular invasion by trophoblastic cells associated with molar villi [1]. Other rarer forms include choriocarcinoma, placental site trophoblastic tumor, and epithelioid trophoblastic tumor [2]. Invasive mole most often develops after a prior diagnosis of hydatidiform mole, with initial presentation as an invasive mole being particularly rare. The incidence of hydatidiform mole varies between 0.57 and 2 per 1,000 pregnancies, depending on the region. Post-molar GTN occurs in approximately 15-20% of complete moles and 1–5% of partial moles [3]. Identified risk factors include a β-hCG level >100,000 mIU/ml, excessive uterine enlargement, and the presence of theca lutein cysts ≥6 cm [4]. The clinical presentation of invasive mole is often nonspecific, including abnormal uterine bleeding, uterine enlargement, pelvic pain, and symptoms related to β-hCG stimulation. While local invasion is common, uterine perforation leading to hemoperitoneum is rare. Metastases are observed in approximately 15% of cases, with the lungs, vagina, liver, and brain being the most commonly affected sites [3]. Diagnosis relies on β-hCG monitoring, imaging, and histopathological analysis. GTNs have a high potential for local invasion and metastasis but are highly curable, with survival rates approaching 100% with appropriate treatment. Chemotherapy remains the mainstay of treatment, but hysterectomy may be required in cases of severe hemorrhagic complications or resistance to medical therapy [5].

Case report

A 52-year-old married woman, mother of three children (P4G3), with no significant medical history and still menstruating (with irregular cycles), presented with a history of heavy vaginal bleeding that began two months prior. Pelvic ultrasound revealed the presence of an end cavitary mass with multiple vesicles, creating a "honeycomb" appearance. Unfortunately, the ultrasound images were unavailable. Serum β-hCG levels were measured at 225,000 mIU/mL. The patient underwent

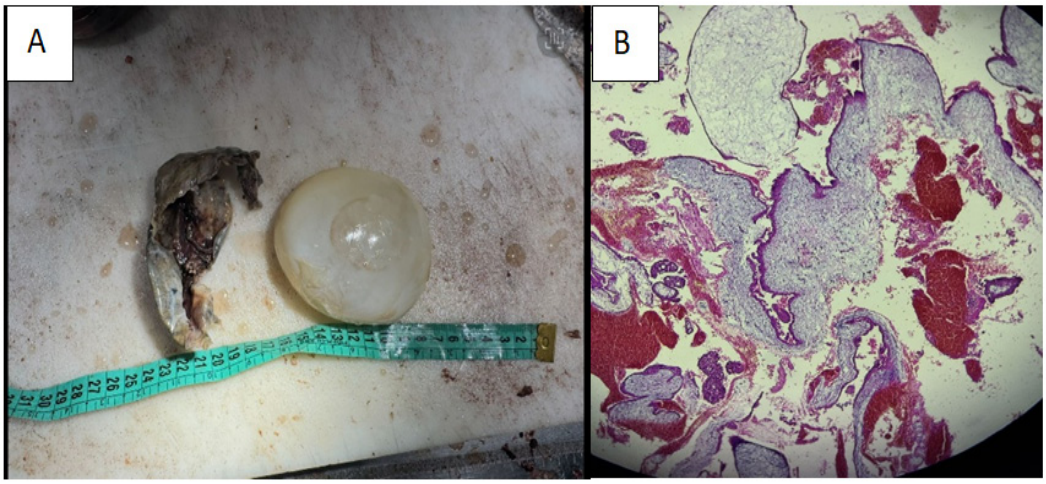

Figure 1: (A) Macroscopic image showing a translucent cystic vesicle measuring approximately 11 cm, suggestive of a complete hydatidiform mole, accompanied by placental debris. The characteristic "grape-like" appearance is due to hydropic degeneration of chorionic villi.
(B) Histological section stained with hematoxylin and eosin (H&E), demonstrating a complete hydatidiform mole with edematous, grape-like chorionic villi, marked trophoblastic proliferation, and absence of fetal blood vessels—hallmark features of this pathology.

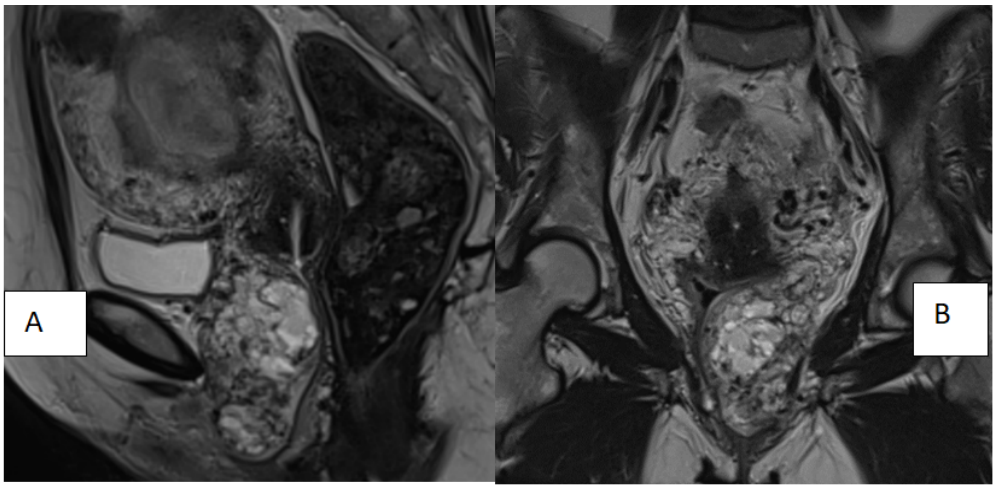

Figure 2: Sagittal T2-weighted **(A)** and coronal T2-weighted **(B)** images showing a poorly defined, irregularly contoured, circumferential lesion occupying the entire uterine cavity, predominantly tissue-like, with high T2 signal intensity.

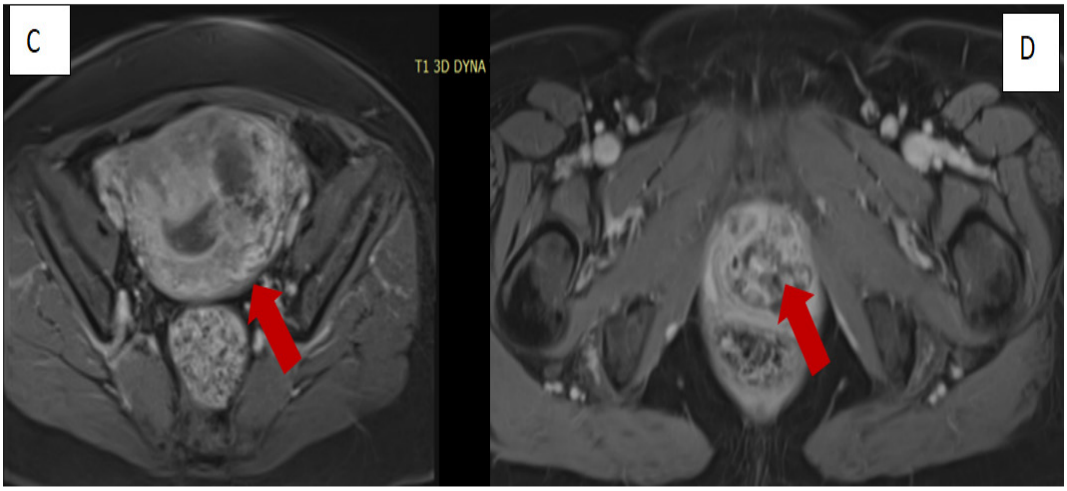

Figure 3: Axial T1-weighted images after gadolinium injection **(C,D)** showing heterogeneous enhancement of the lesion (red arrow).

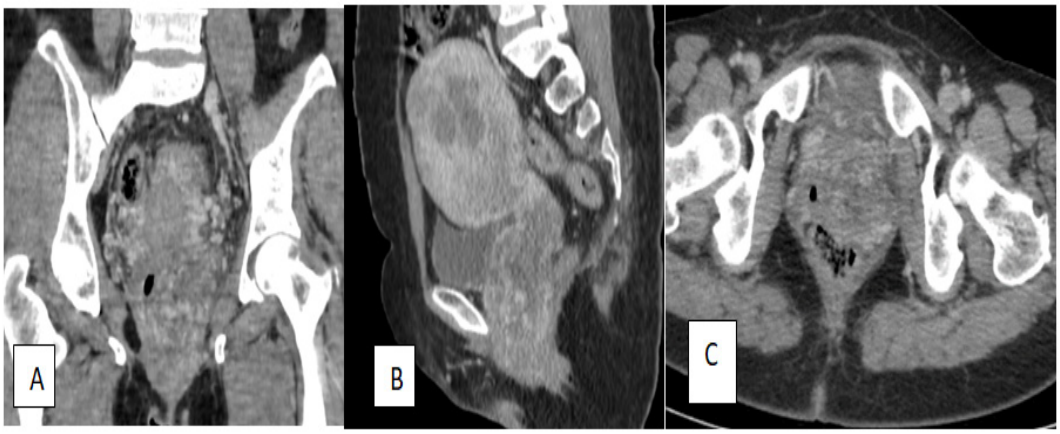

Figure 5: CT scan showing the enlarged uterus measuring 1160 × 95 × 120 mm (HxAPx1), with a poorly defined endocavitary lesion of irregular, circumferential contours occupying the entire uterine cavity.

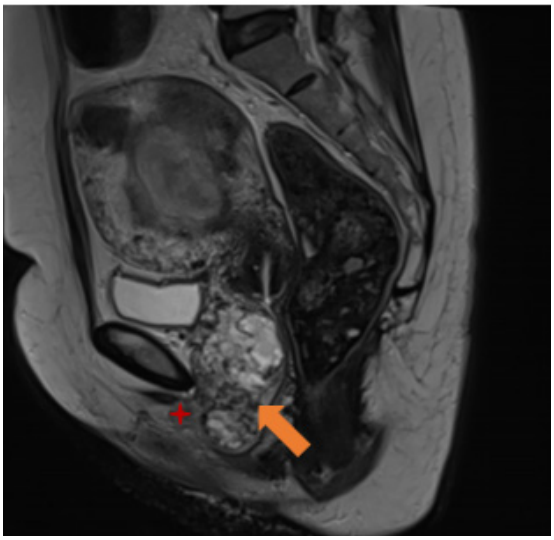

Figure 4: Sagittal T2-weighted image showing the extent of the lesion reaching the lower third of the vagina (orange arrow) and infiltration of the adjacent fat (red star).

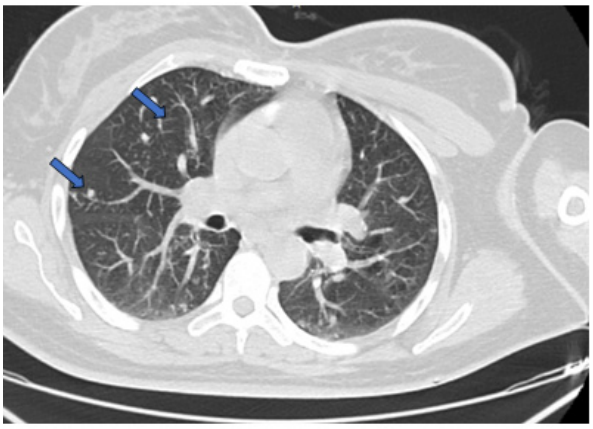

Figure 6: Axial lung window image demonstrating pulmonary nodules suggestive of secondary lesions (blue arrow).

uterine aspiration, and a follow-up ultrasound showed a clearly visible empty endometrial cavity. The aspirated material was sent for pathological examination, which confirmed the diagnosis of a complete hydatidiform mole (Figure 1). However, a subsequent β-hCG measurement revealed an increase to 155,760 mIU/mL. The clinical course was further complicated by the recurrence of vaginal bleeding two weeks after the procedure, with another β-hCG test showing a new rise to 225,000 mIU/mL. Laboratory tests showed mild anemia (Hb: 10 g/dL), mild thrombocytopenia (platelets: 140,000/µL), and a normal white blood cell count (WBC: 5,000/µL). Liver function tests (AST/ALT) were within normal limits, with serum creatinine at 0.9 mg/dL and normal urea levels. Renal function was normal, as indicated by normal creatinine and electrolytes, and the coagulation profile (PT and aPTT) was also normal. A pelvic MRI revealed an enlarged uterus measuring 130 × 90 × 116 mm, with a poorly defined, irregular endocavitary lesion occupying the entire uterine cavity. The lesion was predominantly solid, displaying low T1 and high T2 signal intensity, (Figure 2) with internal fluid components, no diffusion restriction, and heterogeneous enhancement after gadolinium injection, extending approximately 19 cm in height (Figure 3). Associated intra- and perilesional vascular ectasia with signal voids was noted. The lesion infiltrated the myometrium up to the serosa without exceeding it, extended inferiorly to the lower third of the vagina with disruption of the T2 hypointense vaginal wall signal and adjacent fat infiltration. It reached the posterior bladder wall without intraluminal protrusion, contacted the pubis without cortical irregularity, and abutted the anterior abdominal wall while preserving the separation plane (Figure 4). A TAP scan was performed as part of the distant staging workup, which revealed an enlarged uterus measuring 1160 × 95 × 120 mm (HxAPx1), with a poorly defined endocavitary lesion of irregular, circumferential contours occupying the entire uterine cavity. The lesion was predominantly tissue-based, with heterogeneous enhancement following injection and fluid-filled areas. It extended to the lower third of the vagina, with adjacent fat infiltration, and reached the posterior bladder wall without endoluminal protrusion (Figure 5). Bilateral dense pulmonary nodules suggestive of secondary lesions were also observed (Figure 6). Given the secondary pulmonary and vaginal lesions, the patient was classified as FIGO stage > 7 (FIGO 2000 classification) and was prescribed polychemotherapy based on the EMA-CO regimen:

- EMA Cycle:

o Day 1: VP16 100 mg/m², Methotrexate (MTX) 100 mg/m², Actinomycin D (Actino) 0.5 mg/m².

o Day 2: VP16 100 mg/m², Actinomycin D 0.5 mg/m², Folinic acid 15 mg twice/day.

- CO Cycle:

o Day 8: Vincristine 1 mg/m², Cyclophosphamide 600 mg/m² (Day 1 = Day 14).

The patient received eight cycles of EMA-CO chemotherapy, which led to a progressive decline in serum β-hCG levels from 0.891 to 0.6 mIU/mL, indicating a favorable biochemical response. Following the completion of treatment, the patient was placed under close surveillance with weekly β-hCG monitoring for three months, followed by biweekly monitoring thereafter, in accordance with post-molar Gestational Trophoblastic Neoplasia (GTN) follow-up protocols.

Discussion

Hydatidiform mole refers to an abnormal pregnancy characterized by varying degrees of trophoblastic proliferation (both cytotrophoblast and syncytiotrophoblast) and vesicular swelling of placental villi associated with an absent or an abnormal fetus/embryo. Two syndromes of hydatidiform mole have been described based on both morphologic and cytogenetic criteria [6]. Complete hydatidiform moles undergo early and uniform hydatid enlargement of villi in the absence of an ascertainable fetus or embryo, the trophoblast is consistently hyperplastic with varying degrees of atypia, and villous capillaries are absent. Approximately 90% of complete moles are 46, XX, originating from duplication of the chromosomes of a haploid sperm after fertilization of an egg in which the maternal chromosomes either inactive or absent. The other 10% of complete moles are 46, XY, or 46, XX, as a result of fertilization of an empty ovum by 2 sperm (dispermy).

Trophoblastic neoplasia (invasive mole or choriocarcinoma) follows complete mole in 15-20% of cases. Partial hydatidiform moles demonstrate identifiable fetal or embryonic tissue [7], chorionic villi with focal edema that vary in size and shape, scalloping and prominent stromal trophoblastic inclusions, and a functioning villous circulation, as well as focal trophoblastic hyperplasia with mild atypia only. Most partial moles have a triploid karyotype (usually 69, XXY), resulting from the fertilization of an apparently normal ovum by 2 sperm [7].

Invasive mole is a benign tumor that arises from myometrial invasion of a hydatidiform mole via direct extension through tissue or venous channels. Approximately 10-17% of hydatidiform moles will result in invasive mole, and about 15% of these will metastasize to the lungs or vagina. Invasive mole is most often diagnosed clinically rather than pathologically based on persistent hCG elevation after molar evacuation and is frequently treated with chemotherapy without a histopathologic diagnosis [8].

The pathogenesis of invasive mole, particularly in perimenopausal women, remains unclear. It is believed to be linked to the immature spontaneous ovulation of oocytes, which leads to decreased fertility in perimenopausal and eventually postmenopausal patients. Typically, invasive mole metastases occur in the vagina, lungs, and brain due to the invasion of molar tissue into the venous system [9], although rarer metastases have been reported in the epidural space and bladder. Seckl et al. (2000) noted that locally invasive gestational trophoblastic neoplasia develops in 15% of patients and metastasis in 4% after evacuation of a complete mole, with less frequent occurrences after partial mole. In our case, the patient presented with lung and vaginal metastases, marking an unusual manifestation of invasive mole. Notably, biopsy of metastases is generally avoided due to the risk of hemorrhage [10]. Invasive moles, which are often preceded by hydatidiform moles in about 95% of cases, are associated with several risk factors, including advanced reproductive age, spontaneous abortions, vitamin A deficiency, oral contraceptive use, and paternal and environmental factors [11]. The invasive hydatidiform mole presents with uncontrolled vaginal bleeding between 6 and 16 weeks of gestation in 80 to 90% of cases, uterine enlargement greater than expected for gestational age (28%), hyperemesis (8%), as well as a persistent elevation in b-HCG levels exceeding 100,000 mIU/mL following molar evacuation [12].

Ultrasonography is considered the first-line imaging modality for the initial diagnosis of hydatidiform moles and is also a reliable tool for monitoring disease progression in patients with elevated serum β-hCG levels. Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are recommended for disease staging and the detection of metastatic lesions, as demonstrated in our case [13].

Invasive hydatidiform moles are classified as low-risk and high-risk based on the prognostic scoring system established by FIGO. The treatment of low-risk gestational trophoblastic neoplasia (score ≤ 6) is relatively well established, with consensus favoring first-line monotherapy. Five different regimens have been studied: methotrexate alone, methotrexate combined with folinic acid, actinomycin D alone, sequential treatment with methotrexate and actinomycin D, and oral etoposide. Most of these therapies demonstrate an efficacy rate exceeding 70%. However, no randomized studies have directly compared the effectiveness of these different chemotherapy regimens. A complete response rate of up to 80% has been reported with weekly methotrexate monotherapy [14].

For high-risk invasive hydatidiform moles (FIGO score ≥ 7), the necessity of polychemotherapy is widely acknowledged. Several protocols have been proposed, among which the MAC and CHAMOCA regimens have been found to be both more toxic and less effective than EMA-CO [15]. The largest published series evaluating the EMA-CO regimen reported a complete response rate of 78%, with an acceptable tolerance profile [16].

Conclusion

Invasive hydatidiform mole is a rare but aggressive condition requiring early diagnosis and management. Persistent β-hCG elevation post-molar evacuation should raise suspicion, especially in cases with pulmonary or vaginal metastases. MRI is essential for assessing invasion, and chemotherapy remains the mainstay of treatment, with surgery reserved for complications. Early intervention ensures excellent outcomes.

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