Cureus
Part of SPRINGER NATURE

Open Access Case Report

Postmenopausal Invasive Mole Complicated by Spontaneous Uterine Rupture and Massive Haemorrhage: A Case Report

Marina S. Gomes [1], Mariana V. Tavares [1], Vanda Patrício [1], Mónica C. Pires [1]

1. Department of Gynecology, Instituto Português de Oncologia do Porto Francisco Gentil, EPE, Porto, PRT

Corresponding author: Marina S. Gomes, marinarsgomes@gmail.com

Review began 10/21/2026
Review ended 11/15/2026
Published 11/17/2026

© **Copyright** 2026
Gomes et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119819

Abstract

Gestational trophoblastic disease (GTD) is exceptionally uncommon after menopause. Invasive mole may penetrate the myometrium and uterine vasculature and, rarely, cause uterine rupture.

A 51-year-old woman, gravida 1 para 1 (term delivery 33 years earlier) and amenorrhoeic since the age of 46, presented with vaginal bleeding. Ultrasound showed a 15.1 × 12.4 × 10.0 cm multicystic intrauterine mass, serum beta-human chorionic gonadotropin (β-hCG) was 365,806 mIU/mL, and endometrial biopsy was reported as an early complete hydatidiform mole. On the eve of planned surgery, she developed acute abdominal pain, with haemoglobin of 5.3 g/dL and platelets of 86 × 10^9/L. Emergency laparotomy revealed spontaneous complete uterine rupture with haemoperitoneum and an intraoperative blood loss of 4,400 mL; haemostasis required total hysterectomy with bilateral salpingo-oophorectomy and right hypogastric artery ligation. Histology showed invasive mole with extensive lymphovascular invasion, parametrial extension, and a positive anterior parametrial margin (pT2 Nx; FIGO stage II). The postoperative course was complicated by pelvic abscess and haematoma requiring two re-laparotomies and intensive care. β-hCG remained elevated; the disease was classified as low-risk, methotrexate/folinic acid was started without response, and second-line actinomycin D led to normalisation, with no recurrence for more than six years.

Invasive mole should be considered in postmenopausal women with abnormal bleeding and markedly elevated β-hCG. In this patient, a large molar uterus with a very high β-hCG ruptured spontaneously and caused life-threatening haemorrhage; reliable predictors of rupture remain undefined. Management requires early surgical planning, specialist GTD risk assessment, and prolonged β-hCG surveillance.

Categories: Obstetrics/Gynecology, Oncology
Keywords: gestational trophoblastic disease (gtd), invasive mole, postmenopausal bleeding, spontaneous haemoperitoneum, spontaneous uterine rupture

Introduction

Gestational trophoblastic disease (GTD) comprises hydatidiform moles and gestational trophoblastic neoplasia (GTN), which includes invasive mole, choriocarcinoma, placental site trophoblastic tumour, and epithelioid trophoblastic tumour. Invasive mole is characterised by hydropic chorionic villi and trophoblastic proliferation that invades the myometrium and/or uterine vasculature and may cause persistent beta-human chorionic gonadotropin (β-hCG) production, tissue destruction, and severe haemorrhage [1,2]. Although these disorders are usually diagnosed during reproductive life, they are exceptionally rare after menopause, where they are described mainly in isolated case reports, and population-based epidemiological data are lacking [3,4]. Diagnosis of gestational trophoblastic disease after menopause is challenging. Postmenopausal bleeding is usually attributed to endometrial pathology, a large pelvic mass in this age group suggests a non-gestational malignancy, and a raised β-hCG may be attributed to pituitary hCG, so a gestational origin is rarely considered. Uterine rupture or perforation due to invasive mole is described mainly in case reports, spontaneous rupture after menopause has rarely been reported, and its surgical management is seldom detailed. We describe a postmenopausal woman with a complete mole and invasive disease who developed spontaneous complete uterine rupture with massive haemoperitoneum on the eve of planned surgery, in whom haemostasis required hysterectomy and right hypogastric artery ligation. We focus on the rupture and its surgical control, and review the published cases of invasive mole with uterine rupture or perforation.

Case Presentation

Presentation and workup

A 51-year-old woman, gravida 1 para 1, presented to the emergency department with moderate vaginal bleeding and hypogastric pain. For one month, she had also noticed generalised oedema, abdominal distension, and breast enlargement. She had reached menopause at 46 years and used hormone therapy for six months only. Her only pregnancy had ended in an uncomplicated vaginal delivery at 18 years of age.

Medical history included hypertension, dyslipidaemia, subclinical hyperthyroidism, and depression. Transvaginal ultrasound showed a well-defined heterogeneous mass of 15.1 × 12.4 × 10.0 cm arising from the uterine corpus and fundus, with a multicystic internal pattern and continuity with the endometrial line; the adnexa were normal, and there was no free fluid. An endometrial biopsy was performed and was interpreted as a complete hydatidiform mole. Serum β-hCG was 365,806 mIU/mL. She was referred to a gynaecological oncology centre for further management.

Staging showed a 4-mm non-specific nodule in the right upper lobe on chest CT and a normal brain CT. Pelvic MRI showed a uterine mass of about 20 cm in its longest axis with heterogeneous enhancement, extending inferiorly to the right side of the cervical region with a 5-cm component. Right parametrial involvement could not be excluded. There was a small amount of free fluid and periuterine nodes smaller than 1 cm, and the ovaries were not visualised. The size of the mass, its extension towards the cervical region, and the possible parametrial involvement made a uterus-sparing approach or chemotherapy alone unsuitable, so the multidisciplinary meeting recommended primary hysterectomy with bilateral salpingo-oophorectomy. The MRI showed only a small amount of free fluid, with no sign of rupture, and the patient was haemodynamically stable, so surgery was planned as an elective procedure.

On the eve of the planned surgery, she developed acute abdominopelvic pain with hypogastric tenderness and rebound. Temperature was 37.5°C, blood pressure 100/52 mmHg, heart rate 123 beats/min, and oxygen saturation 95%. Haemoglobin had fallen to 5.3 g/dL and platelets to 86 × 10^9/L (Table *1*). Uterine rupture was suspected clinically because of the sudden severe abdominal pain, an acute abdomen, and a fall in haemoglobin, and was confirmed at laparotomy. Two units of packed red blood cells were transfused before surgery, and she underwent emergency laparotomy.

Parameter	Result	Reference range
Haemoglobin (g/dL)	5.3	11.5-16.5
Haematocrit (L/L)	0.156	0.370-0.470
Platelet count (×10^9/L)	86	150-450
Prothrombin time (s)	15.6	12.4-15.2
Activated partial thromboplastin time (s)	37.8	24.6-31.8

TABLE 1: Relevant laboratory findings

Surgery and postoperative course

An emergency laparotomy was performed. The uterus was enlarged to the umbilicus. There was haemoperitoneum of approximately 1,100 mL and a spontaneous laceration of about 7 cm at the isthmus on the right, extending almost to the right cornu (Figure *1*). Total hysterectomy with bilateral salpingo-oophorectomy was performed, and because of profuse haemorrhage, right hypogastric artery ligation was also required to achieve haemostasis (Figure *2*). The total intraoperative blood loss was 4,400 mL, whereas the haemoperitoneum found on opening the abdomen was approximately 1,100 mL; the difference reflects the bleeding during dissection and haemostasis. The patient received two units of packed red blood cells, two units of plasma, and two units of platelets intraoperatively.

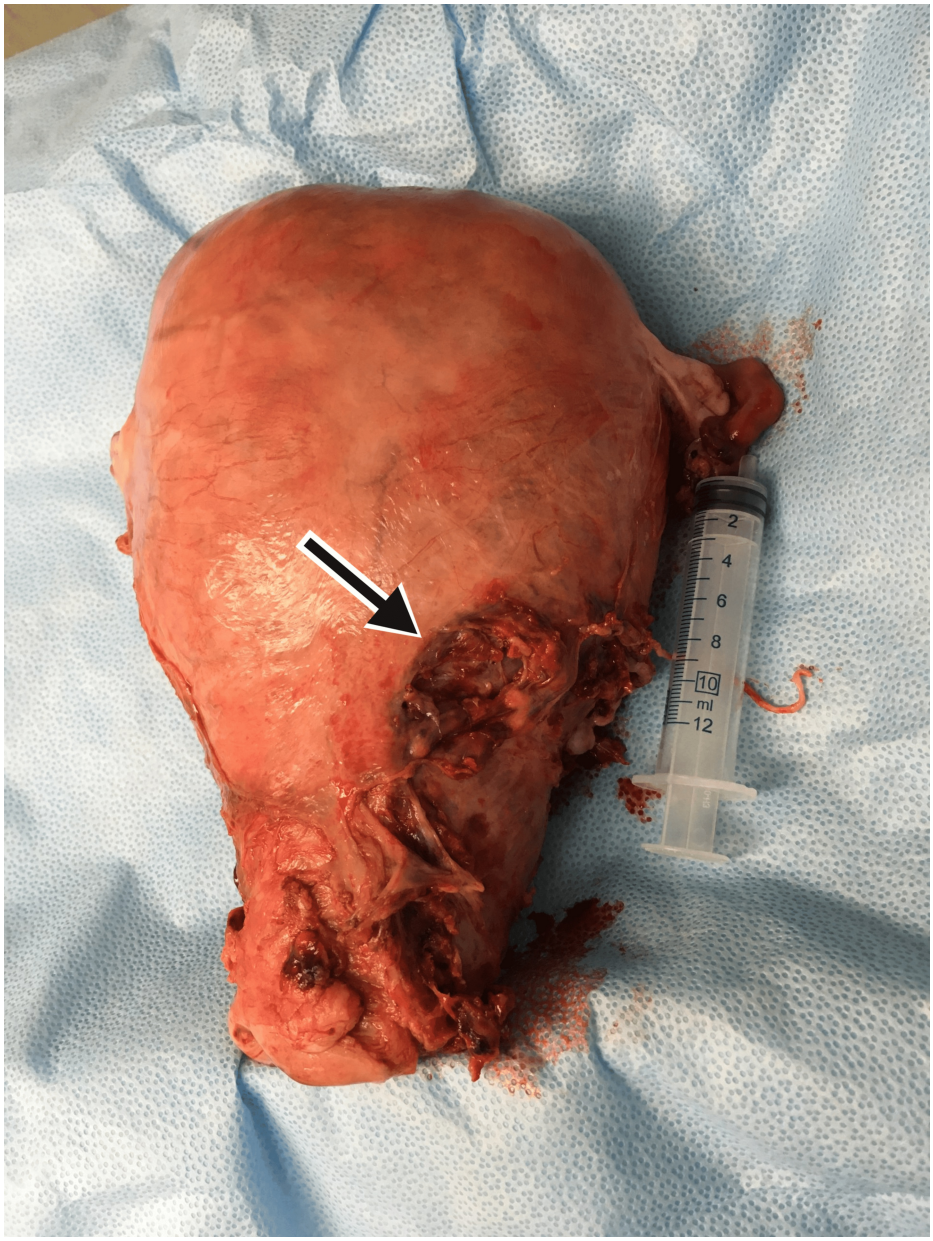


FIGURE 1: Gross appearance of the hysterectomy specimen showing the site of spontaneous uterine rupture (arrow)

A 12-mL syringe is shown for scale.

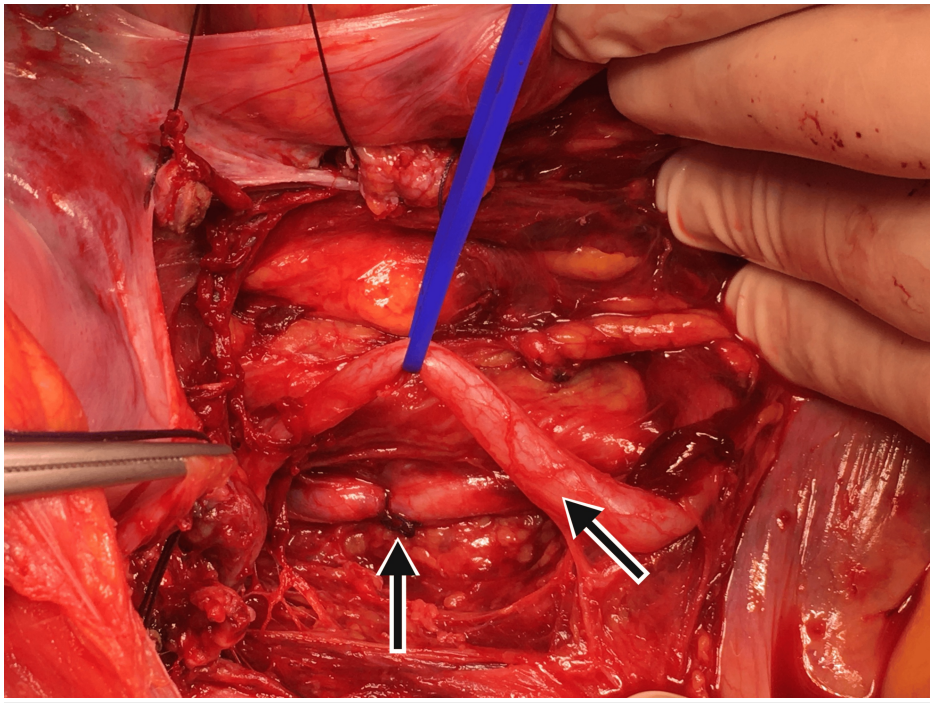


FIGURE 2: Intraoperative view of the right pelvic sidewall after right hypogastric (internal iliac) artery ligation

The arrow at the bottom indicates the ligature on the artery; the arrow on the right indicates the ureter.

The postoperative course was complicated by a pelvic abscess and a pelvic haematoma. On postoperative day 7, she underwent a second laparotomy, with drainage and abundant peritoneal lavage, for suspected pelvic abscess; a large abdominal wall and pelvic abscess were found, and cultures grew *Escherichia coli* and *Streptococcus agalactiae*. She was treated with piperacillin-tazobactam, a central venous catheter was placed, and she received total parenteral nutrition, albumin, and further transfusion of packed red blood cells. On postoperative day 14, a third laparotomy was performed for suspected pelvic haematoma, with evacuation of the haematoma and peritoneal lavage. She was admitted to the intensive care unit with sepsis of abdominal origin and respiratory failure, and antibiotic therapy was escalated to vancomycin and meropenem. She left the intensive care unit after five days with clinical and laboratory improvement. She was later readmitted with anaemia, received a transfusion of packed red blood cells, and was discharged with a haemoglobin of 11.2 g/dL.

Pathology

The specimen weighed 1,471 g, and the uterus measured 20.0 × 17.0 × 5.5 cm, with a maximum myometrial thickness of 2 cm. The cavity was filled by a 20 × 17 cm multivesicular lesion with yellowish softened areas and haemorrhage near the cervix. Vesicles were visible within the lumina of isthmic and myometrial vessels. A rupture with exposed tumour was described in the parametrial region, together with an extensive irregular area with adherent clot involving the lateral surface and corpus. The cervix and adnexa were macroscopically unremarkable.

Microscopy showed large chorionic villi with cistern formation, frequent villous trophoblastic hyperplasia, and extensive necrosis. There was an exuberant implantation site with intermediate trophoblast permeating the myometrium and extensive lymphovascular invasion in the myometrium and parametrium (Figure *3*). The tumour involved the parametrium in continuity with the broad ligament and peritoneal reflection, and the anterior parametrial margin was involved. The endocervix and adnexa were free; the endometrium showed decidual change. The diagnosis was invasive hydatidiform mole, stage pT2 Nx (AJCC 8th edition) and FIGO stage II (2009). Immunohistochemistry showed loss of p57 expression in villous stromal cells and cytotrophoblast. The molecular study concluded chorionic villi with an allelic profile compatible with a diandric diploid genotype.

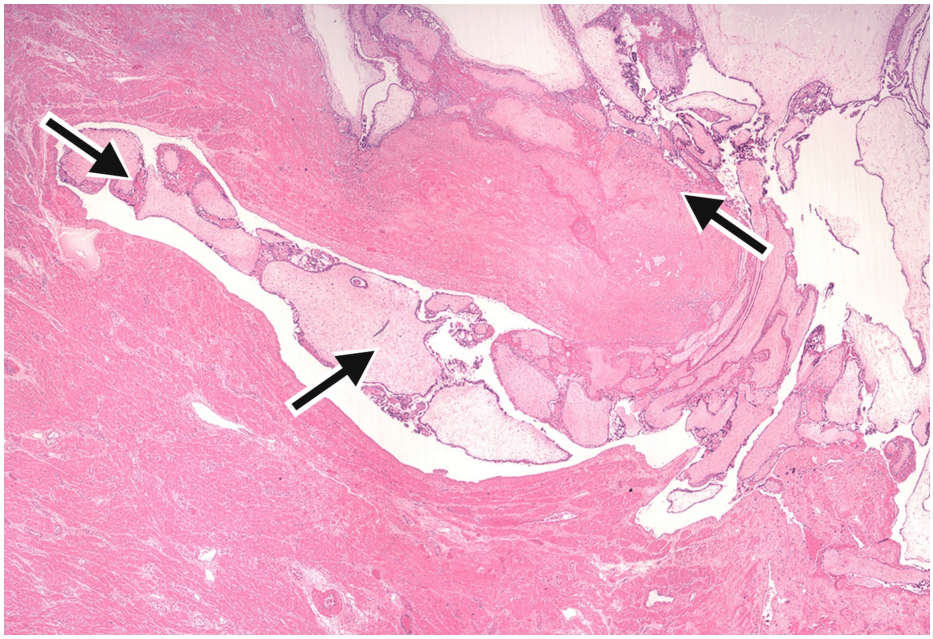


FIGURE 3: Low-power photomicrograph of the hysterectomy specimen (haematoxylin and eosin, ×40) showing large hydropic chorionic villi with oedematous stroma and cistern formation (arrows) within the myometrium, consistent with invasive hydatidiform mole

Persistent β-hCG and chemotherapy

β-hCG did not normalise after surgery: it was 613 mIU/mL two weeks and 475 mIU/mL five weeks after surgery, and then showed a plateau and a small rise (429 and 508 mIU/mL) before chemotherapy. Postoperative imaging (chest, abdominal, and pelvic CT, brain CT) showed no metastases. At the multidisciplinary meeting about six weeks after surgery, the group recommended adjuvant chemotherapy in view of the persistent β-hCG, the parametrial involvement, and the positive parametrial margin. The disease was classified as low-risk gestational trophoblastic neoplasia (FIGO stage II, prognostic score 3). Single-agent methotrexate with folinic acid rescue, the standard first-line treatment for low-risk disease [1,2], was started about eight weeks after surgery, with cycles every two weeks. At the start of methotrexate, β-hCG was 729 mIU/mL; it fell to 296, 103, and 44.6 mIU/mL after the first three cycles, reaching a nadir of 36.1 mIU/mL, and then rose to 44.3 and 92.1 mIU/mL after the fifth cycle. Methotrexate resistance was defined as a plateau (less than 10% decrease over two courses) or a rise in β-hCG (at least two values over two weeks) after an initial response, and this was fulfilled. Restaging with pelvic MRI and CT of the thorax, abdomen, and pelvis showed no measurable disease. Second-line actinomycin D, given as a five-day course repeated every two weeks, was started five months after surgery, when β-hCG was 97.6 mIU/mL. It fell to 29.9, 12.5, and 3.33 mIU/mL, and then to 0.991 mIU/mL, about two months after starting actinomycin D. She received six cycles in total, four of them as consolidation after normalisation, and chemotherapy was concluded about eight months after surgery. Since then, β-hCG has remained negative, with no clinical or imaging evidence of recurrence.

Discussion

This case illustrates spontaneous uterine rupture with massive haemoperitoneum in a postmenopausal woman with invasive mole, in whom haemostasis required hysterectomy and right hypogastric artery ligation. Persistent disease after surgery was resistant to methotrexate and was cured with actinomycin D. Invasive mole is distinguished from choriocarcinoma by the presence of chorionic villi within the myometrium and/or vessels, as seen here [1,2]. The diagnosis of a complete mole is supported by p57 loss and a diandric diploid genotype.

Spontaneous rupture most likely resulted from extensive myometrial and vascular invasion, together with a 20-cm, 1,471-g molar uterus with only about 2 cm of myometrium. We cannot exclude that the endometrial biopsy contributed to the weakening of the wall, although the interval of almost four weeks makes this unlikely. The initial multidisciplinary plan was elective surgery. Whether a large molar uterus and a very high β-hCG predict rupture cannot be concluded from a single case and a small, heterogeneous literature. Reliable predictors of rupture remain undefined. We therefore suggest only that these features should raise clinical suspicion and prompt early specialist planning, rather than serve as a validated risk criterion. The rupture declared itself on the eve of planned surgery as an acute abdomen with a haemoglobin of 5.3 g/dL,

and haemostasis was achieved only after hysterectomy combined with right hypogastric artery ligation, a last-resort measure for uncontrolled pelvic haemorrhage.

The pathogenesis of gestational trophoblastic disease presenting after menopause remains uncertain, and the antecedent gestational event cannot be established in the present case. A complete mole requires fertilisation of an oocyte, and our patient had been amenorrhoeic for five years, with a single term delivery 33 years earlier that cannot be the antecedent event. Previous reports have proposed an unrecognised perimenopausal conception or prolonged persistence of molar tissue [3,4].

The principal clinical implication is that a markedly elevated β-hCG in a postmenopausal woman with abnormal uterine bleeding should not automatically be attributed to a non-gestational source, particularly when imaging and pathology demonstrate molar tissue.

Using the FIGO 2000 prognostic scoring system, the patient scored 3 points, which is low risk (≤6): age 51 (1), antecedent pregnancy a mole (0), tumour larger than 5 cm (2), β-hCG of 508 mIU/mL before chemotherapy (0), no metastases (0), and no previous chemotherapy (0). The β-hCG used was the value measured just before the start of chemotherapy, in line with the recommendation that the FIGO score be calculated from the hCG immediately before treatment. The multidisciplinary group considered the disease gestational, given the chorionic villi in the specimen, and did not take the term delivery 33 years earlier as the antecedent pregnancy. The patient was therefore managed as low risk, with single-agent chemotherapy. We note that this classification depends on two choices: using the β-hCG before chemotherapy instead of the preoperative value of 365,806 mIU/mL, which would give a score of 7, and the fact that the interval from the index pregnancy cannot be defined in a postmenopausal woman. The later resistance to methotrexate shows the limits of the score in this unusual case. Anatomical staging was FIGO stage II.

Single-agent chemotherapy is the standard first-line treatment for low-risk GTN. The patient had no metastases on CT and MRI, the β-hCG before chemotherapy was low after hysterectomy (508 mIU/mL), and she was classified as low risk. Methotrexate with folinic acid was therefore chosen as first-line treatment. The disease invaded the myometrium and the right parametrium, but this local extent is not part of the FIGO risk score. In retrospect, the plateau and rise in β-hCG after five cycles, and the need for second-line actinomycin D, suggest that local invasion with a positive margin may predict resistance. This is a hypothesis from a single case.

The positive parametrial margin showed that the disease extended beyond the uterus and that hysterectomy alone was not curative, which explains the persistently elevated β-hCG after surgery (475, 429 and 508 mIU/mL). It established FIGO stage II and justified adjuvant chemotherapy, instead of surveillance alone. Its prognostic value for chemotherapy resistance is unknown, and we cannot say whether it contributed to the resistance seen here.

Review of published cases

We searched PubMed/MEDLINE in September 2026 using (“Hydatidiform Mole, Invasive” OR “invasive mole” OR “chorioadenoma destruens”) AND (“Uterine Rupture” OR “uterine rupture” OR perforat*) AND (hemoperitoneum OR haemoperitoneum OR hemorrhag* OR haemorrhag*) (Table *2*).

Report	Age	Menopausal status	Antecedent pregnancy	Complication	Haemoperitoneum	Surgery	Chemotherapy	Outcome
Atala et al. (1992) [5]	NR	NR	Abortion	Rupture after curettage	Yes	Hysterectomy	Yes (regimen NR)	Died of complications
Lewis et al. (1994) [6]	NR	NR	NR	Perforation	Yes	Total abdominal hysterectomy	NR	NR
Kittur and Venktesh (2013) [7]	21	Premenopausal	Complete mole, evacuated	Silent perforation	3.5-4 L	Emergency laparotomy, hysterectomy	EMA-CO ×6	β-hCG normal at 6 months
Sinha et al. (2014) [8]	20	Premenopausal	NR	Fundal rupture	About 2 L	Emergency laparotomy, hysterectomy	NR	NR
Grin et al. (2017) [9]	24	Premenopausal	NR	Posterior perforation	About 2.5 L	Laparoscopic resection, uterus preserved	Methotrexate	NR
Aminimoghaddam and Maghsoudnia (2017) [10]	41	Premenopausal	Complete mole, curettage twice	Left posterior wall perforation	Yes (volume NR)	Subtotal hysterectomy, left salpingo-oophorectomy, omentectomy	EMA-CO ×4, then 3 consolidation courses	Disease-free at 5 years
Dhieb et al. (2021) [11]	48	Perimenopausal	No known molar pregnancy	Rupture	Yes	Subtotal hysterectomy, bilateral adnexectomy	Yes (regimen NR)	NR
Zamani et al. (2021) [12]	21	Premenopausal	Complete mole, evacuated	Rupture	About 2 L	Resection, uterus preserved	Methotrexate, then actinomycin D	β-hCG declined
Wu et al. (2022) [13]	31	Premenopausal	Abortion	Rupture	2.6 L	Hysterectomy	EMA-CO ×6	β-hCG normal
Xu et al. (2022) [14]	22	Premenopausal	Complete mole, two evacuations	Rupture	About 2 L	Uterine artery embolisation, uterus preserved	Intra-arterial methotrexate, EMA-CO	Disease-free at 13 months
Lakra et al. (2025) [15]	30	Premenopausal	Oral abortifacient at about 2 months' gestation	Posterior wall perforation	Yes (volume NR)	Obstetric hysterectomy, bilateral salpingo-oophorectomy	None	β-hCG undetectable at 6 months
Houssaini et al. (2026) [16]	55	Postmenopausal	Complete mole evacuated 6 months earlier	Spontaneous perforation	Massive	Total hysterectomy	EMA-CO ×6	Complete remission
Present case	51	Postmenopausal	No known antecedent; complete mole	Spontaneous complete rupture	About 1.1 L	Hysterectomy, bilateral salpingo-oophorectomy, hypogastric artery ligation	Methotrexate (resistant), then actinomycin D	Disease-free at more than 6 years

TABLE 2: Published cases of invasive mole complicated by uterine rupture or perforation

Dhieb et al. [11] and Aminimoghaddam et al. [10] describe metastatic disease. The total blood loss in the present case was 4.4 L. "Chorioadenoma destruens", the term used by Sinha et al. [8], is an older synonym for invasive mole. Details for Atala et al. [5] are taken from its abstract only.

β-hCG: beta-human chorionic gonadotropin, NR: not reported in the available source

In the targeted search, one case meeting the specific combination of invasive mole, spontaneous uterine rupture or perforation, and postmenopausal status was identified: the 2026 report by Houssaini et al. [16].

The management of gestational trophoblastic neoplasia is primarily determined by disease extent, clinical presentation, and prognostic assessment, with chemotherapy forming the cornerstone of treatment [1,2]. In the present case, emergency hysterectomy was necessary because of uncontrolled haemorrhage, complete uterine rupture, and extensive parametrial disease. Right hypogastric artery ligation was additionally required to obtain haemostasis. In contrast, selected younger patients with a strong desire for fertility preservation have been managed with uterine artery embolisation, local resection, or uterine repair, followed by chemotherapy [9,12,14]. These conservative strategies are not directly applicable to the present patient because fertility preservation was not a treatment objective and the extent of uterine and parametrial destruction necessitated definitive surgery.

In peri- and postmenopausal women, Dhieb et al. described a 48-year-old woman with a ruptured metastatic invasive mole [11]. The present case differs from the only other postmenopausal report by

Houssaini et al. [16] by the absence of a documented preceding molar pregnancy, the parametrial involvement with a positive margin, and the methotrexate resistance. Furthermore, in this patient, hysterectomy alone did not secure haemostasis, which was obtained after right hypogastric (internal iliac) artery ligation, performed on the side of the dominant laceration. Hypogastric artery ligation is an established measure for life-threatening pelvic haemorrhage that is not controlled by other means [17]. Among the cases in Table *2*, we found no other description of hypogastric artery ligation. In haemodynamically stable patients, embolisation can control haemorrhage and preserve the uterus [14]. Our patient, with a ruptured uterus, tachycardia of 123 beats/min, blood pressure of 100/52 mmHg, and a haemoglobin of 5.3 g/dL, needed emergency laparotomy. No complication specific to the ligation was recorded; the postoperative abscess and pelvic haematoma are more likely related to emergency surgery with massive haemorrhage, although a contribution of the ligation cannot be excluded.

Limitations

The main limitation of this report is its single-case nature and the inability to determine the antecedent gestational event responsible for the trophoblastic disease. In addition, the rarity of the condition and the heterogeneous terminology used in older reports limit meaningful comparison of outcomes across published cases. The literature review is exploratory and not systematic: it used one database (PubMed), and non-indexed or non-English literature may have been missed. It was not designed to be complete and should be read as context for the present case, not as a comprehensive account of the literature.

Conclusions

Invasive mole should be considered in postmenopausal women with abnormal bleeding, a large multicystic uterine mass, and markedly elevated β-hCG, even when pregnancy would not ordinarily be considered. In this patient, a large molar uterus with a very high β-hCG ruptured spontaneously, producing an acute haemoperitoneum that required emergency surgery to control haemorrhage. This is a single observation, and reliable predictors of rupture remain undefined. Such features should raise clinical suspicion and prompt early planning with blood products and experienced pelvic surgical support. Careful GTN risk assessment, awareness that a positive parametrial margin may require systemic therapy, and prolonged β-hCG surveillance in a specialist centre are essential.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Marina S. Gomes

Acquisition, analysis, or interpretation of data: Marina S. Gomes, Mariana V. Tavares, Vanda Patrício, Mónica C. Pires

Drafting of the manuscript: Marina S. Gomes

Critical review of the manuscript for important intellectual content: Mariana V. Tavares, Vanda Patrício, Mónica C. Pires

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Ngan HY, Seckl MJ, Berkowitz RS, et al.: Diagnosis and management of gestational trophoblastic disease: 2025 update. Int J Gynaecol Obstet. 2025, 171 Suppl 1:78-86. 10.1002/ijgo.70275
2. Soper JT: Gestational trophoblastic disease: current evaluation and management . Obstet Gynecol. 2021, 137:355-70. 10.1097/AOG.0000000000004240
3. Garcia M, Romaguera RL, Gomez-Fernandez C: A hydatidiform mole in a postmenopausal woman. A case report and review of the literature. Arch Pathol Lab Med. 2004, 128:1039-42. 10.5858/2004-128-1039-AHMIAP
4. Taşkin S, Cengiz B, Ortaç F: Invasive mole in a postmenopausal woman . Int J Gynaecol Obstet. 2006, 93:156-

7. 10.1016/j.ijgo.2006.01.010
5. Atala C, Riedemann R, Biotti M, Ramírez F, Paublo M: Invasive mole with uterine rupture . Rev Chil Obstet Ginecol. 1992, 57:356-8.
6. Lewis PL, Mendelovici R, Scotti RJ: A case report of invasive mole with uterine rupture . J Gynecol Surg. 1994, 10:89-91. 10.1089/gyn.1994.10.89
7. Kittur S, Venktesh: A rare case of invasive mole with silent uterine perforation . Int J Reprod Contracept Obstet Gynecol. 2016, 2:109-10. 10.5455/2320-1770.ijrcog20130222
8. Sinha M, Kaur R, Gupta R, Rani R, Aggarwal A: Rare presentation of chorioadenoma destruens as acute haemoperitoneum mimicking ruptured ectopic pregnancy. Kathmandu Univ Med J (KUMJ). 2014, 12:288-91. 10.3126/kumj.v12i4.13736
9. Grin L, Namazov A, Volodarsky M, Anteby E, Lavie O, Gemer O: Laparoscopic management of an invasive mole perforating the uterus. J Minim Invasive Gynecol. 2017, 24:199-200. 10.1016/j.jmig.2016.08.814
10. Aminimoghaddam S, Maghsoudnia A: Unusual presentation of invasive mole: a case report . J Reprod Infertil. 2017, 18:205-9.
11. Dhieb F, Boumediene M, Saoussem A, et al.: Case report: acute abdomen presentation revealing a metastatic invasive mole with uterine rupture. F1000Res. 2021, 10:1115. 10.12688/f1000research.74051.1
12. Zamani M, Alizadeh S, Mollabashi M: Fertility-sparing uterine lesion resection in a woman with hemoperitoneum due to invasive mole: a rare case report. Int J Surg Case Rep. 2021, 84:106117. 10.1016/j.ijscr.2021.106117
13. Wu A, Zhu Q, Tan C, Chen L, Tao Y: Invasive mole resulting in uterine rupture: a case report . Front Surg. 2021, 8:798640. 10.3389/fsurg.2021.798640
14. Xu L, Feng M, Cai J, Li H: Case report: conservative treatment for fertility preservation in a woman with hemoperitoneum due to an invasive mole. Front Oncol. 2022, 12:1019082. 10.3389/fonc.2022.1019082
15. Lakra S, Mallick S, Singh S: Perforating invasive hydatidiform mole a catastrophe: a case report . Bioinformation. 2025, 21:1140-3. 10.6026/973206300211140
16. Houssaini KI, Essaoudy I, Bassir A, et al.: Spontaneous uterine perforation revealing an invasive mole: a case report. Pan Afr Med J. 2026, 55:44. 10.11604/pamj.2026.55.44.49092
17. Shrestha R, Shrestha S, Sitaula S, Basnet P: Anatomy of internal iliac artery and its ligation to control pelvic hemorrhage. JNMA J Nepal Med Assoc. 2020, 58:826-30. 10.31729/jnma.4958