

Peritoneal Metastasis of Malignant Melanoma: A Case Report and Literature Review

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Case Report

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Abstract

Malignant melanoma (MM) is a highly aggressive tumor originating from melanocytes in the skin and mucosa. Although peritoneal metastasis is uncommon clinically, it confers an extremely poor prognosis. This paper reports a female patient presenting with abdominal distension and anorexia, who was preliminarily diagnosed with ovarian malignant tumor before surgery. Intraoperative frozen-section pathology indicated poorly differentiated carcinoma and she underwent surgery combined with hyperthermic intraperitoneal chemotherapy (HIPEC). Postoperative pathology confirmed MM, and the final diagnosis was multiple metastases from subcutaneous MM. The patient received targeted therapy postoperatively and is currently alive with more than one year of follow-up. Based on a literature review, this article analyzed the clinical features, diagnosis, and comprehensive treatment strategies of this condition, so as to provide a reference for the early diagnosis and individualized treatment of MM with peritoneal metastasis.

Introduction

Malignant melanoma (MM) is a highly aggressive neoplasm originating from melanocytes in the skin and mucosa. Its incidence varies considerably across regions, with the highest rate in Australia and the lowest in Central Asia [1]. Although melanoma accounts for less than 5% of all skin cancers, it is responsible for 65% of skin cancer-related deaths due to its high malignancy and strong metastatic tendency [2].

This article reports a female patient admitted to the Department of Gynecology, Fourth Hospital of Hebei Medical University, presenting with abdominal distension and anorexia. Preoperative examination indicated a potential ovarian malignant tumor. Intraoperative frozen-section pathology indicated poorly differentiated carcinoma, and therefore cytoreductive surgery and hyperthermic intraperitoneal chemotherapy (HIPEC) were performed. Postoperative pathology and immunohistochemistry (IHC) confirmed the diagnosis of malignant MM. Combined with a prior history of melanoma in the buttocks and postoperative PET/CT, multiple metastases in the lung, bone, and peritoneum were confirmed. Genetic testing showed BRAF V600 mutation, and thus postoperative treatment included dual-target therapy with dabrafenib and trametinib, as well as denosumab for bone protection. The patient was followed up for one year with stable disease. This article analyzed the clinical characteristics, diagnostic, and treatment methods of multiple metastases of MM.

Clinical data

The patient, a 45-year-old female, was admitted with a chief complaint of abdominal distension and anorexia for over one month. Main manifestations included abdominal distension, poor appetite, occasional shortness of breath, without nausea, vomiting, abdominal pain, abnormal vaginal discharge or bleeding.

Gynecological ultrasound revealed a cystic-solid mass in the right adnexal region, possibly of ovarian origin; multiple nodules on the uterine surface and pelvic peritoneal surface; and ascites. Contrast-enhanced chest CT showed multiple pulmonary nodules and masses of varying sizes in both lungs (metastasis not excluded), enlarged mediastinal lymph nodes, slight elevation of the right hemidiaphragm, focal atelectasis of the right middle and lower lobes and left lung, multiple cord-like opacities in both lungs, and bilateral small pleural effusions with adjacent lung atelectasis. Contrast-enhanced abdominopelvic CT indicated fullness in the right adnexal region with suspicious hypodensity (pelvic MRI recommended), flocculent and nodular thickening of the omentum (metastasis not excluded), and abdominopelvic fluid. Pelvic MRI revealed a malignant mass in the right adnexal region (cystadenocarcinoma not excluded), peritoneal thickening, blurred fat planes in the anterior pelvis (all considered metastatic), and pelvic fluid. Tumor markers showed HCG 0.1 mIU/mL, AFP 1.90 ng/mL, CEA 0.52 ng/mL, CA19-9 8.10 U/mL, CA125 100 U/mL, CA72-4 1.12 U/mL, HE4 65.2 pmol/L. Preoperative ascitic fluid punctures were performed four times, and pathology showed no tumor cells and no aneuploid cells.

To establish a diagnosis, laparoscopic exploration was performed. Intraoperative findings as following: the uterus was normal in size, with miliary nodules on its surface, measuring approximately 4 cm × 5 cm (Fig. 1A); the left adnexa was absent; the right fallopian tube was thickened with a diameter of approximately 3 cm and had a smooth surface (Fig. 1B); the right ovary contained a 2 cm corpus hemorrhagicum with a smooth surface (Fig. 1B); the omentum was covered with rust-colored miliary tumor nodules (Fig. 1C); patchy miliary lesions were noted on the pelvic floor peritoneum, and scattered miliary nodules were present on parts of the mesentery and diaphragmatic peritoneum; no enlarged lymph nodes were noted in the pelvis or para-aortic region; the surfaces of the liver, stomach, spleen, and appendix were smooth. Intraoperative frozen sections revealed poorly differentiated carcinoma nodules in the fallopian tube and omentum (Fig. 2). Consequently, we performed laparoscopic total hysterectomy, right adnexectomy, omentectomy, pelvic peritoneum resection and placement of an intraperitoneal hyperthermic perfusion catheter. Hyperthermic perfusion with intraperitoneal cisplatin (60 mg) was performed later.

Postoperative pathological findings were as follows: atypical cell clusters were observed on the ovarian surface, fallopian tube, and omentum. IHC results showed AE1/AE3 (-), Vimentin (+), Syn (-), Ki67 (positive in 30% of cells), S100 (+), HMB45 (+), MART-1 (+), Calretinin (-), Desmin (-), CD34 (vascular +), CD117 (regional +) and SF-1 (-). The pathological diagnosis was consistent with MM. Tumor infiltration was observed on the uterine serosa, parts of the omentum, and pelvic peritoneum. Peritoneal lavage fluid revealed atypical cells and aneuploid cells.

Postoperative PET/CT imaging showed: ☐ Multiple nodular high-density shadows of varying sizes were identified in both lungs, with abnormal hypermetabolism of glucose on PET; ☐ Lymph nodes were noted in the mediastinum (regions 2R, 4R, and 7) and the right supradiaphragmatic region, some of which were enlarged, and abnormal hypermetabolism of glucose was observed in PET; ☐ Circular density shadows was seen in the right scapula, sternum, upper segment of the right humerus, lumbar I vertebrae, iliac

surface and sacral surface of the left sacroiliac joint, left ischium, and medullary cavity of the left iliac wing. Local cortical bone thinning can be observed, and abnormal hypermetabolism of glucose can be seen in PET; ¶ In the pelvic cavity, there were strip-shaped and nodular hypermetabolism of glucose beside the intestine. Abnormal hypermetabolism of glucose still was seen in the local area by delayed imaging. CT showed an increase in soft tissue shadows in the corresponding area, which couldn't be ruled out that there were residual tumor in the local area.

Further history inquiry revealed that the patient had undergone excision of a gluteal mass 6 years prior. Postoperative pathology (right gluteal groove) showed atypical melanocytic nevus with malignant change; margins and base were negative for tumor. IHC showed HMB45 (+), Melan-A (+), S-100 (+), PNL-2 (+), P53 (+), P16 (-), P63 (-) and Ki-67 (+, 20%). Pathological consultation at the fourth hospital indicated a compound nevus with active growth and malignant potential, with uninvolved surgical margins.

Based on the patient's prior history, intraoperative findings, postoperative pathology, and imaging results, the diagnosis was multiple metastases from MM. Postoperative genetic testing revealed a BRAF V600 mutation. Targeted therapy with a BRAF inhibitor (dabrafenib) combined with a MEK inhibitor (trametinib) was initiated. Due to concurrent bone metastases, denosumab injection was administered for bone-modifying maintenance therapy. The patient was regularly followed up, and the disease remained stable, with no progressive metastatic lesions observed on one year, which indicated treatment effective.

Discussion

Melanoma is characterized by high invasiveness and a propensity for systemic metastasis [3]. Distant metastasis occurs in more than 60% of patients with advanced (stage IV) disease, and the 5-year survival rate is only 15–20% [4]. Acral and mucosal melanomas have a higher risk of early metastasis compared to cutaneous melanoma [5].

Peritoneal metastasis is a rare distant metastasis from melanoma. A population-based study reported that melanoma accounts for less than 0.5% of all cases of peritoneal metastatic carcinoma [6]. Flanagan et al. further demonstrated that the median overall survival of patients with stage IV melanoma and peritoneal metastasis was only 1.8 months, compared to 12.3 months for those without peritoneal metastasis, indicating that it is an independent poor prognostic factor [7].

Intra-abdominal metastasis of melanoma involves liver and pelvic lymph nodes most commonly, while involvement of the peritoneum, ovaries, and adrenal glands is even rarer. Trout AT and colleagues studied abdominopelvic metastases from melanoma, enrolling 102 patients with abdominopelvic cavity metastasis from cutaneous melanoma; they found that the most common sites of abdominopelvic metastasis were the liver (47% of patients) and pelvic lymph nodes (27% of patients). Although the ovary was not separately analyzed as a primary focus, it was regarded as an "atypical metastatic site". The study also found a strong correlation between metastatic pattern and primary tumor location: lower extremity primaries had the highest metastatic rate (52%) and were prone to metastasize to the pelvis;

ocular and head/neck primaries showed a strong predilection for liver metastases (70% and 63%, respectively) [8]. In this case, the melanoma originated in the right buttock with extensive metastasis, involving the uterine surface, ovarian surface, omentum, peritoneum, bone and lung, without liver or pelvic lymph node involvement.

The time interval from primary cutaneous melanoma resection to peritoneal metastasis is highly heterogeneous, ranging from as short as 10 months (e.g., T1a scalp melanoma [9]) to over 10 years. A case reported in 2025 described a 53-year-old female who developed peritoneal metastasis more than 10 years after initial treatment for stage IIB nodular melanoma, with a clinical presentation resembling advanced ovarian cancer [10]. Rarely, metastasis may occur 30 years after primary resection [11]. This temporal heterogeneity reflects the biological capability of melanoma cells to undergo dormancy and late reactivation. The present case, with peritoneal metastasis was discovered more than six years after cutaneous melanoma resection, corroborating this feature.

Peritoneal metastasis from melanoma presents with symptoms similar to primary peritoneal carcinoma (PPC) and ovarian cancer (OC), requiring careful differential diagnosis. Medical history and tumor markers are the key points for initial differentiation. Most patients with peritoneal melanoma metastasis have a prior history of melanoma; approximately 10% have an occult primary. Serum S100 and lactate dehydrogenase (LDH) are the core elevated markers, while CA125 and HE4 are usually normal. CA125 may only be secondarily elevated when there is extensive peritoneal infiltration accompanied by massive ascites. Imaging modalities (CT, MRI, PET-CT) are essential for lesion detection and extent assessment, but lack specificity [12]. Diagnosis is particularly challenging in patients without a known cutaneous primary [13]. Notably, ascitic fluid cytology in patients with peritoneal melanoma metastasis is often negative. In one case of peritoneal metastasis confirmed by laparoscopic biopsy, cytological examination revealed no malignant cells in ascites done by ultrasound-guided paracentesis [14].

Pathology and IHC represent the gold standard for differential diagnosis. Morphologically, both primary OC and PPC originate from Müllerian epithelium, and serous carcinoma is the most common type. The cellular atypia is limited to the characteristics of epithelial-derived tumors [15]. In contrast, metastatic melanoma often shows epithelioid or spindle cell morphology and typically lacks glandular differentiation. It may show pigmentation in some cases, and the amelanotic type is easily confused with poorly differentiated carcinoma [16]. IHC is crucial for distinguishing among these entities. Melanoma characteristically expresses SOX10, S-100, HMB-45, and Melan-A, but is negative for PAX8 and WT1. Primary OC and PPC are characterized by positive expression of markers PAX8, WT1, and CK7 [15, 16]. A panel combining these markers significantly improves diagnostic accuracy and helps avoid clinical misdiagnosis.

Regarding treatment, according to the NCCN Clinical Practice Guidelines in Oncology for Cutaneous Melanoma [17] and the CSCO Clinical Practice Guidelines for Melanoma [18], a comprehensive treatment system has been established for advanced multiple metastatic melanoma, based on molecular subtyping, with systemic therapy as the mainstay and local therapy as a supplement.

Systemic therapy is the core treatment for multiple metastatic melanoma. Guidelines recommend molecular profiling followed by stratified therapy. For patients with BRAF V600 mutations, both NCCN and CSCO give a Category I recommendation for the dual-target regimen of a BRAF inhibitor (dabrafenib, vemurafenib) combined with a MEK inhibitor (trametinib, cobimetinib). This regimen serves as the first-line option for patients with high tumor burden and rapidly progressive disease. For BRAF wild-type patients, treatment primarily involves immune checkpoint inhibitors: PD-1 inhibitor monotherapy (e.g., nivolumab or pembrolizumab) serves as the backbone. For patients with high tumor burden and good performance status, dual immunotherapy with a PD-1 inhibitor (e.g., nivolumab or pembrolizumab) plus a CTLA-4 inhibitor (e.g., ipilimumab) may be used. For NRAS-mutant patients, MEK inhibitors (e.g., trametinib or cobimetinib) may be considered after immunotherapy failure. This stratified approach represents the standard treatment pathway for advanced melanoma [17, 18]. The present patient harbored a BRAF V600 mutation and received dabrafenib plus trametinib dual-target therapy, which was successful, with stable disease at one-year follow-up.

Local therapy plays a pivotal role in the comprehensive treatment strategy for multiple metastases. Surgical resection of solitary or oligometastatic lesions, stereotactic body radiotherapy (SBRT), and other modalities can effectively control local progression and alleviate symptoms. For bone metastases (including spinal and vertebral metastases), NCCN and CSCO guidelines recommend routine use of bone-modifying agents (e.g., denosumab) in addition to systemic therapy. For bone metastases in weight-bearing sites such as thoracic and lumbar spine, radiotherapy or SBRT may be selected based on bone stability and symptoms. For patients with vertebral instability, pathological fracture, or risk of spinal cord compression, timely orthopedic surgery is required to restore spinal stability and relieve neural compression. For special-site metastases such as peritoneal, hepatic or cerebral metastases, cytoreductive surgery, local radiotherapy or interventional/ablation procedures can reduce tumor burden and alleviate patient discomfort. Both NCCN and CSCO guidelines recommend that, under the premise of effective systemic therapy, local intervention for oligometastatic lesions can further prolong disease control duration [17, 18].

In summary, peritoneal metastasis from melanoma is clinically extremely rare. Without a clear history of cutaneous melanoma, clinical misdiagnosis or omission is common. For such cases, detailed history taking and review of postoperative pathology are essential. In terms of treatment, the present patient underwent surgical resection of the lesion combined with HIPEC, followed by maintenance targeted therapy and denosumab for bone management. The comprehensive treatment was effective, and the patient remained stable at one-year follow-up.

Abbreviations

MM, Malignant melanoma

HIPEC, Hyperthermic intraperitoneal chemotherapy

IHC, Immunohistochemistry

PPC, Primary peritoneal carcinoma

OC, Ovarian cancer

LDH, Lactate dehydrogenase

SBRT, Stereotactic body radiotherapy

Declarations

Ethics approval and consent to participate

This single case report is a retrospective clinical case observation without experimental intervention, so formal ethics committee approval was not required. Written informed consent was obtained from the patient.

Consent for publication

Written informed consent was obtained from the patient for the publication of relevant clinical data and images in this case report.

Availability of data and materials

Not applicable.

Competing Interests

All the authors declare that the authors have no competing interests as defined by BMC, or other interests that might be perceived to influence the results and/or discussion reported in this paper.

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Authors' contributions

J.L. drafted the full manuscript, and L.L. revised and polished the entire article. All authors read and approved the final version of the manuscript.

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Figures

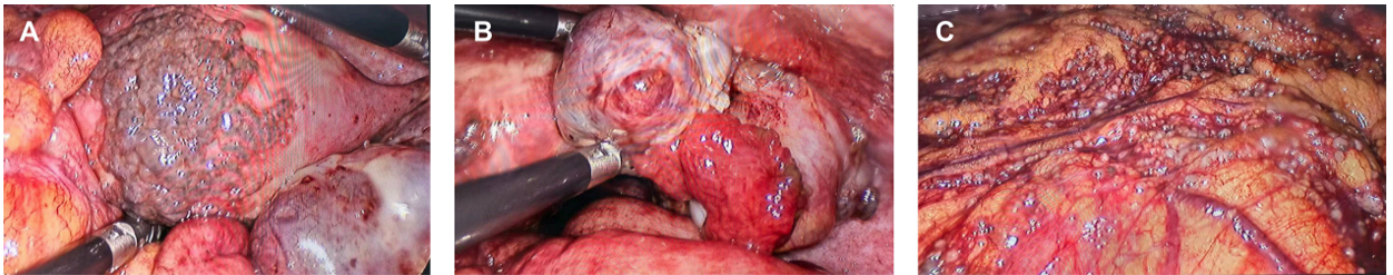


Figure 1

Laparoscopic findings. A: the uterus was normal in size, with miliary nodules on its surface, measuring approximately 4 cm × 5 cm; B: the right fallopian tube was thickened with a diameter of approximately 3 cm and had a smooth surface; the right ovary contained a 2 cm corpus hemorrhagicum with a smooth surface; C: the omentum was covered with rust-colored miliary tumor nodules.

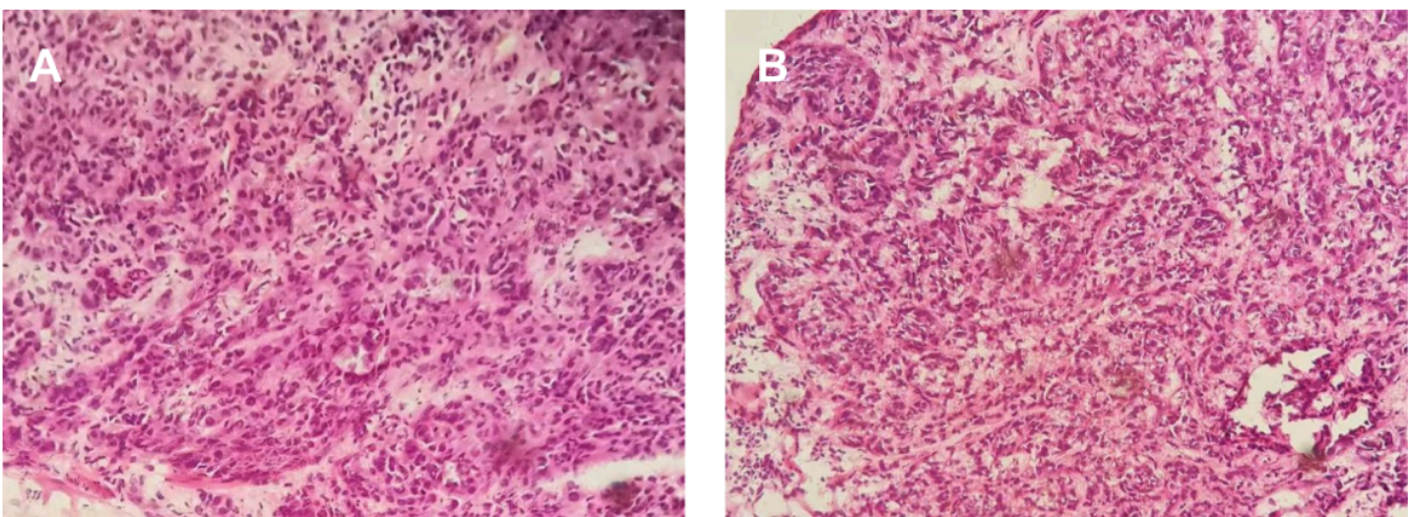


Figure 2

Intraoperative frozen sections. A: Poorly differentiated malignancy in fallopian tube; B: Poorly differentiated malignancy in omentum. (both with HE staining, ×200)