Diffuse peritoneal mesothelioma presenting with the appearance of mesenteric panniculitis: A case report

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Abstract. Diffuse peritoneal mesothelioma (DPM) is a rare and aggressive malignancy of the peritoneum. Early symptoms are non-specific and the condition is liable to misdiagnosis. In the present case, a 54-year-old man was misdiagnosed between April 2020 and October 2021 with mesenteric panniculitis. The patient later presented with a 2-month history of persistent upper-middle abdominal pain and weight loss in April 2022. A laparoscopic biopsy was performed and the sample was positive for Wilms tumor protein, Hector Battifora mesothelial-1 and podoplanin. Therefore, a diagnosis of DPM was made. A total of six cycles of pemetrexed-cisplatin chemotherapy were given and elicited a partial response. The disease progressed and intraperitoneal bevacizumab-cisplatin therapy was administered, which was ineffective. The patient died 13 months post-diagnosis. DPM often has a similar presentation to benign conditions, such as mesenteric panniculitis. Correct diagnosis is facilitated by an awareness of occupational exposure risk, such as that of asphalt exposure for the present case, and early histopathological evaluation. The prognosis for DPM remains poor despite multimodal therapy, and novel therapeutic strategies are needed.

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Introduction

Diffuse peritoneal mesothelioma (DPM) is a rare and highly aggressive primary tumor, characterized by its invasive nature, frequent misdiagnosis and poor prognosis (1-3). DPM accounts for 7-30% of all mesothelioma cases, with marked geographic variation in incidence. In the USA, ~15,000 new cases are reported annually, while in China, the incidence increased from 2.14 to 3.14 per 10^6 between 2000 and 2013 (4,5). The etiology of DPM is primarily associated with asbestos exposure; however, only 33-50% of patients have a clear history of exposure. Other contributing factors include physicochemical carcinogens, chronic peritonitis, and genetic susceptibility, such as BRCA1-associated protein 1 (BAP1) mutations (6,7). As asbestos has not been completely banned in China, the incidence of DPM may continue to increase in the future.

Most DPM is asymptomatic or non-specific in the early stages, with insidious onset, and is diagnosed in the mid- to late-stage, with a median time from symptom onset to diagnosis of ~4 months (8,9). The diversity of clinical manifestations depends largely on the extent of tumor spread in the abdominal cavity. The main clinical manifestations include abdominal distension (41-86%) and abdominal pain (31-87%), and may also include weight loss, abdominal masses, and, in some cases, paraneoplastic syndromes. A definitive diagnosis of DPM requires a histopathological examination. DPM etiology and pathogenesis remain to be fully elucidated and current management involves cytoreductive surgery (CRS) combined with hyperthermic intraperitoneal chemotherapy (HIPEC) and systemic chemotherapy. Patients treated with this therapy have improved 5-year overall survival and progression-free survival (PFS) rates compared with untreated patients (10-12). The present study reports a case of DPM misdiagnosed as mesenteric panniculitis, detailing its clinical course, highlighting the

diagnostic challenges, presenting the management outcomes and discussing the potential association with occupational asphalt exposure.

Case report

Clinical presentation. A 54-year-old male highway maintenance worker with a 28-year history of occupational exposure to asphalt (1994-2022) presented with a 2-month history of persistent upper-middle abdominal pain and weight loss, and was admitted (The First People's Hospital of Xiaoshan District, Hangzhou, China) in April 2022. The patient was responsible for repairing potholes and paving road surfaces, and had daily contact with hot-mix (170±5˚C) modified asphalt. The patient did not wear workplace respiratory protective equipment or protective gloves, resulting in the inhalation of asphalt fumes via the respiratory tract and skin contact with hot asphalt. The duration of exposure was long-term, high-dose and unprotected, lasting for 4-6 h per day and ≥22 days/month. No asbestos testing was conducted in the patient's workplace. The patient had no history of smoking or alcohol consumption and no family history of hereditary cancer. The medical history included a laparoscopic appendectomy in November 2019. The patient sought medical attention multiple times for apparent mesenteric panniculitis at The First People's Hospital of Xiaoshan District, The Third People's Hospital of Xiaoshan District, Jinhua Town Community Health Service Center of Xiaoshan District, as well as the Second Affiliated Hospital of Zhejiang University School of Medicine (all Hangzhou, China) between April 2020 and October 2021, and this condition was diagnosed on the basis of abdominal computed tomography (CT) scan (Fig. 1A-C).

A physical examination on admission revealed no jaundice or palpable enlargement of superficial lymph nodes. The thyroid was normal upon palpation and auscultation of the heart and lungs showed no abnormalities. The abdomen was soft with no palpable masses and mild tenderness in the lower middle abdomen, without rebound tenderness or guarding.

Laboratory findings included a normal white blood cell count ($9.62x10^9$/l; normal range: 3.5-$9.5x10^9$/l) and an elevated absolute neutrophil count ($7.80x10^9$/l; normal range: 1.8-$6.3x10^9$/l), giving a neutrophil percentage of 81.0% (normal range: 40-75%) and a lymphocyte percentage of 13.4% (normal range: 20-50%). Serum albumin was decreased (30.8 g/l; normal range: 35.0-55.0 g/l). Tumor markers indicated elevated carbohydrate antigen 125 (CA125) levels (160.60 U/ml; normal range: 0-35 U/ml) and elevated cytokeratin (CK) 19 fragment (Cyfra 21-1) levels (16.86 ng/ml; normal range: 0-3.3 ng/ml). Enhanced abdominal CT imaging showed changes to the omentum, multiple intra-abdominal masses, ascites, pelvic effusion and a mass at the cardiophrenic angle (Fig. 1D-F). Gastro-duodenoscopy showed chronic non-atrophic gastritis, but no notable abnormalities were seen on colonoscopy.

Treatment and follow-up. The patient was diagnosed with diffuse malignant peritoneal mesothelioma in April 2022. From May to December 2022, the patient received six cycles of systemic chemotherapy: Pemetrexed (800 mg on day 1 plus cisplatin 120 mg (40 mg daily for 3 days, total 75 mg/m^2)/cycle. In March 2023, disease progression was confirmed, and treatment was switched to intraperitoneal perfusion therapy with cisplatin (30 mg + bevacizumab 300 mg. In May 2023, one additional cycle of the original pemetrexed-cisplatin regimen was administered. The patient died in September 2023 (Table I).

Laparoscopic surgery under general anesthesia with resection of intra-abdominal lesions, lysis of intestinal adhesions, omental biopsy and intra-abdominal exploration was performed in April 2022. A notable quantity of yellow-white purulent fluid was found in the abdomen, with adhesions in the right lower abdomen and widespread abdominal wall and omental nodules, ranging in size from 0.2 to 2 cm, with scattered nodular masses on the small intestinal surface (Fig. 2). Histopathological examination of abdominal wall and omental biopsies showed morphological features consistent with malignant epithelioid-type mesothelioma (Fig. 3A and B). Immunohistochemical staining was performed on 4-μm-thick sections obtained from 4% neutral buffered formalin-fixed (25˚C, 24 h), paraffin-embedded tissue specimens. All specimens were obtained from the same laparoscopic biopsy of abdominal wall and omental lesions. Antigen retrieval was performed by heating in EDTA antigen retrieval buffer (pH 9.0) at 100˚C for 20 min. Endogenous peroxidase activity was blocked with 3% hydrogen peroxide at room temperature for 10 min and non-specific binding was blocked with 5% BSA (Beijing Solarbio Science & Technology Co., Ltd.) at room temperature for 30 min. Sections were incubated with primary antibodies at room temperature for 40 min:

Cytokeratin (CK), ready-to-use, cat. no. MAB-0093, Fuzhou Maixin Biotech
Cam5.2, ready-to-use, cat. no. MAB-0209, Fuzhou Maixin Biotech
Ki-67, 1:200, cat. no. ZM-0166, OriGene Technologies
CK7, ready-to-use, cat. no. KIT-0021, Fuzhou Maixin Biotech
CK20, ready-to-use, cat. no. ZA-0574, OriGene Technologies
Villin, ready-to-use, cat. no. MAB-0540, Fuzhou Maixin Biotech
CDX2, ready-to-use, cat. no. ZA-0520, OriGene Technologies
Carcinoembryonic antigen (CEA), ready-to-use, cat. no. MAB-0010, Fuzhou Maixin Biotech
MUC2, ready-to-use, cat. no. MAB-1101, Fuzhou Maixin Biotech
MUC4, ready-to-use, cat. no. MAB-1102, Fuzhou Maixin Biotech
MUC5AC, ready-to-use, cat. no. MAB-1103, Fuzhou Maixin Biotech
MUC6 (all ready-to-use, cat. no. MAB-1104, Fuzhou Maixin Biotech
Calretinin (1:100, cat. no. MAB-0064, Fuzhou Maixin Biotech; Wilms tumor protein (WT-1), ready-to-use, cat. no. MAB-0037, Fuzhou Maixin Biotech); Hector Battifora mesothelial-1 (HBME-1), ready-to-use, cat. no. MAB-0211, Fuzhou Maixin Biotech); podoplanin (D2-40), ready-to-use, cat. no. MAB-0049, Fuzhou Maixin Biotech; Progesterone receptor (PR), ready-to-use, cat. no. MAB-0158, Fuzhou Maixin Biotech; PAX8, ready-to-use, cat. no. MAB-0865,

Table I. Treatment timeline.

Postoperative time	Date	Event	Efficacy/physical status assessment
Month 1	May 2022	Initiation of the 1st cycle of chemotherapy (pemetrexed + cisplatin)	-
Month 3	July 2022	2nd cycle of chemotherapy	The sum of the largest diameters of peritoneal masses was reduced by 25% compared with baseline, achieving a PR; CA125 increased to 556.2 U/ml
Month 4	August 2022	3rd cycle of chemotherapy	No notable adverse reactions; serum albumin stabilized at 31.2 g/l
Month 5	September 2022	4th cycle of chemotherapy	Peritoneal masses continued to shrink; ascites remained minimal
Month 6	October 2022	5th cycle of chemotherapy	CA125 decreased to 286.3 U/ml; body weight remained stable
Month 8	December 2022	Completion of the 6th cycle of chemotherapy	The sum of the largest diameters of peritoneal masses was reduced by 55% compared with baseline; CA125 decreased to 156.5 U/ml; PR was maintained
Month 11	March 2023	Disease progression assessed; initiation of intraperitoneal perfusion therapy (cisplatin + bevacizumab)	Masses increased by 26%; moderate ascites; CA125 increased to 923.3 U/ml; ECOG-PS score 2
Month 13	May 2023	Intraperitoneal perfusion therapy ineffective; additional cycle of chemotherapy administered	Cachexia worsened; serum albumin 22.1 g/l; total parenteral nutrition required
Month 17	September 2023	Patient death	Cachexia was the main cause of mortality

PR, partial response; CA125, cancer antigen 125; ECOG-PS, Eastern Cooperative Oncology Group-Performance Status.

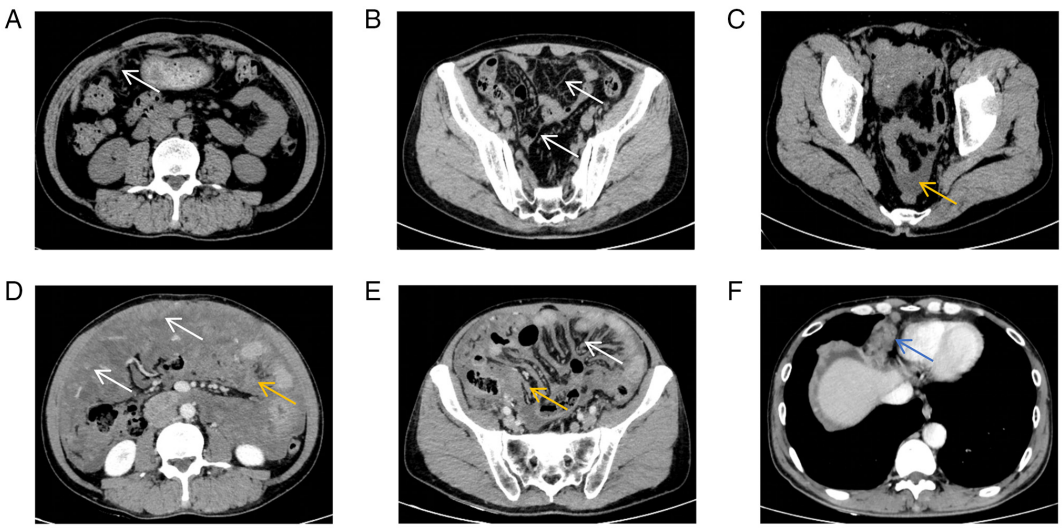

Figure 1. Contrast-enhanced axial abdominal computed tomography scans. (A) Image from July 2020 showed mild striated thickening of the omentum in the upper middle abdomen with minimal surrounding infiltration (white arrows). (B) Image from July 2020 shows striated and nodular thickening of the omentum in the lower abdomen (white arrows). (C) Image from July 2020 shows a small amount of peritoneal fluid in the pelvic cavity (yellow arrow). (D) Image from April 2022 shows marked diffuse thickening of the omentum in the upper middle abdomen (white arrows) and a heterogeneous mass within the omentum (yellow arrow). (E) Image from April 2022 shows irregular sheet like and striated thickening of the omentum with fat stranding in the lower abdomen (white arrows) and a heterogeneous mass within the omentum (yellow arrow). (F) Image from April 2022 shows an enlarged metastatic lymph node at the cardiophrenic angle (blue arrow).

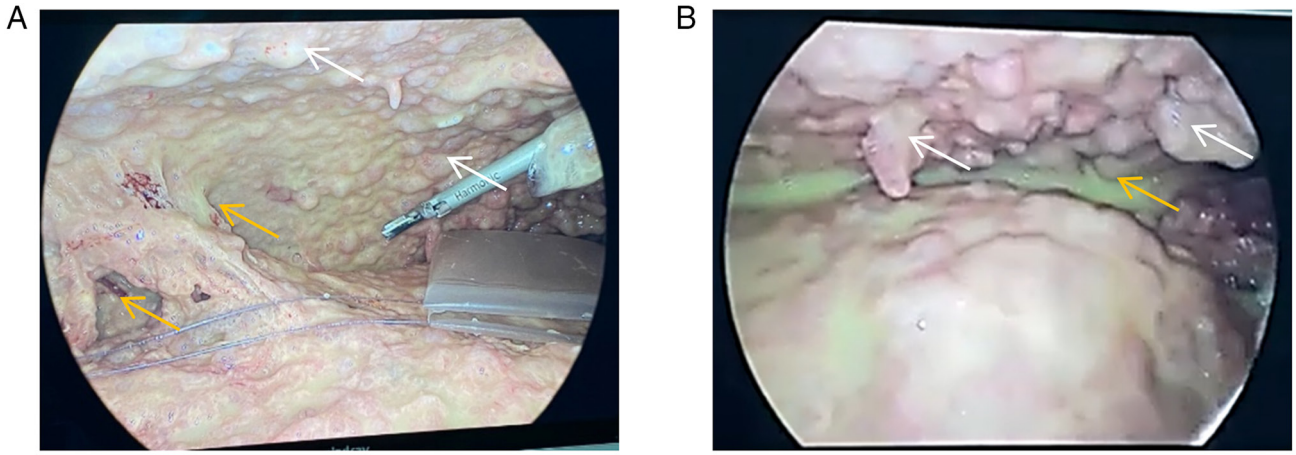

Figure 2. Images from the laparoscopic examination. (A) Laparoscopic image obtained in April 2022 showing widely distributed nodules and masses covering the entire abdominal wall and peritoneal surface (white arrows), accompanied by local intra-abdominal adhesions and purulent effusion (yellow arrow). (B) Magnified laparoscopic view showing variably sized nodules and masses on the peritoneal surface (white arrows) and yellowish viscous purulent effusion (yellow arrows).

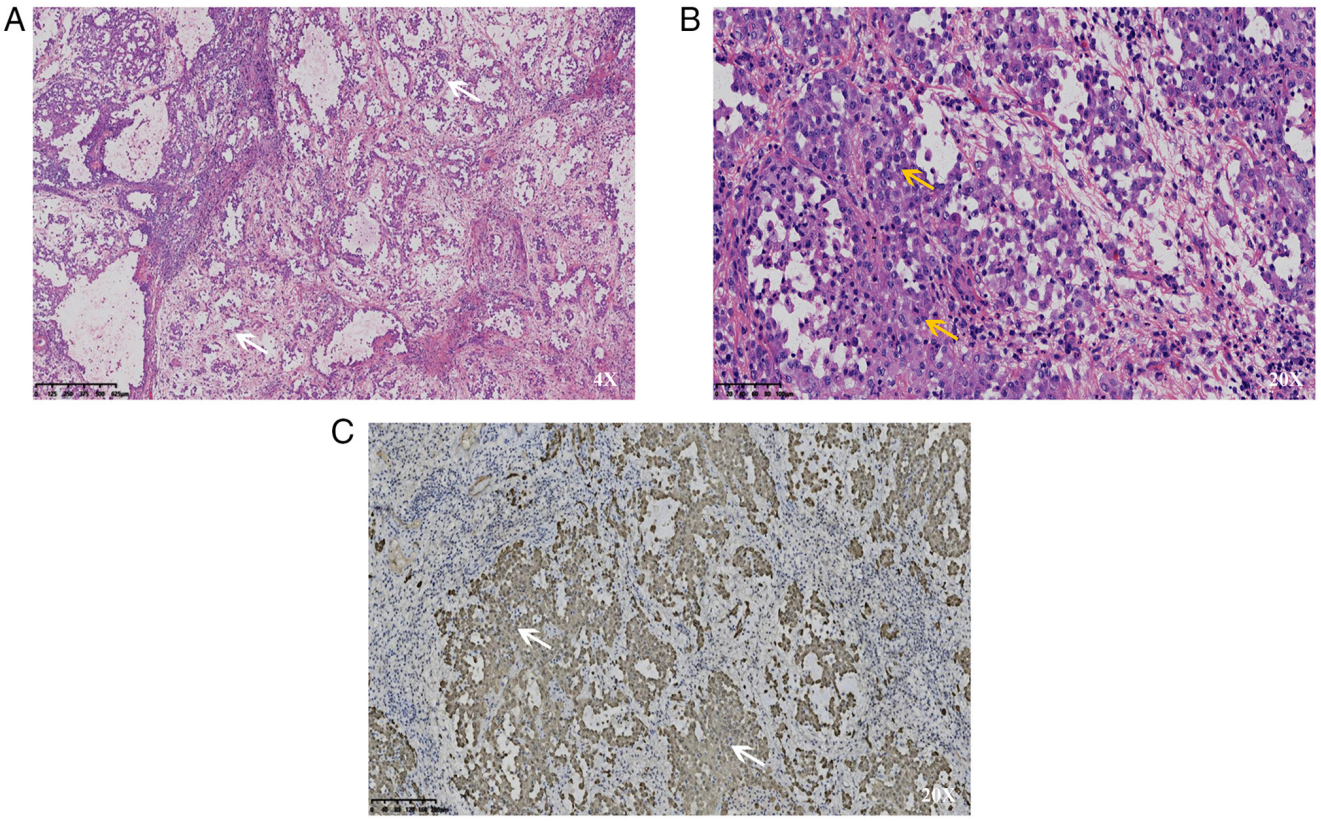

Figure 3. Pathological sections of tumor tissue with hematoxylin and eosin, and immunohistochemical staining. (A) Hematoxylin and eosin staining shows the tumor tissue growing in tubular, fissured, microcystic and patchy shapes under the microscope (white arrows) (magnification, x4). (B) Hematoxylin and eosin staining shows that the tumor cell cytosol is eosinophilic, and the nuclei are round or oval and vacuolated with an obvious nucleolus (yellow arrows) (magnification, x20). (C) Immunohistochemical staining of calretinin in tumor tissue (magnification, x20) shows positive staining of the tumor cytosol and nucleus (white arrows).

Fuzhou Maixin Biotech; p53, ready-to-use, cat. no. KIT-0010, Fuzhou Maixin Biotech

p16, 1:150, cat. no. CF500036, OriGene Technologies. HRP-conjugated goat anti-mouse/rabbit IgG polymer (ready-to-use, cat. no. PV8000D, Beijing Zhongshan Jinqiao Biotechnology Co., Ltd.), incubated at room temperature for 15 min. Detection was performed using a DAB chromogenic kit (cat. no. kit-0014, Fuzhou Maixin Biotech). Slides were counterstained at 25˚C with hematoxylin for 3 min and eosin for 2 min, dehydrated, cleared, and mounted, then observed and imaged using a light microscope. For Ki-67 quantification, the Ki-67 index was determined as the percentage of positively

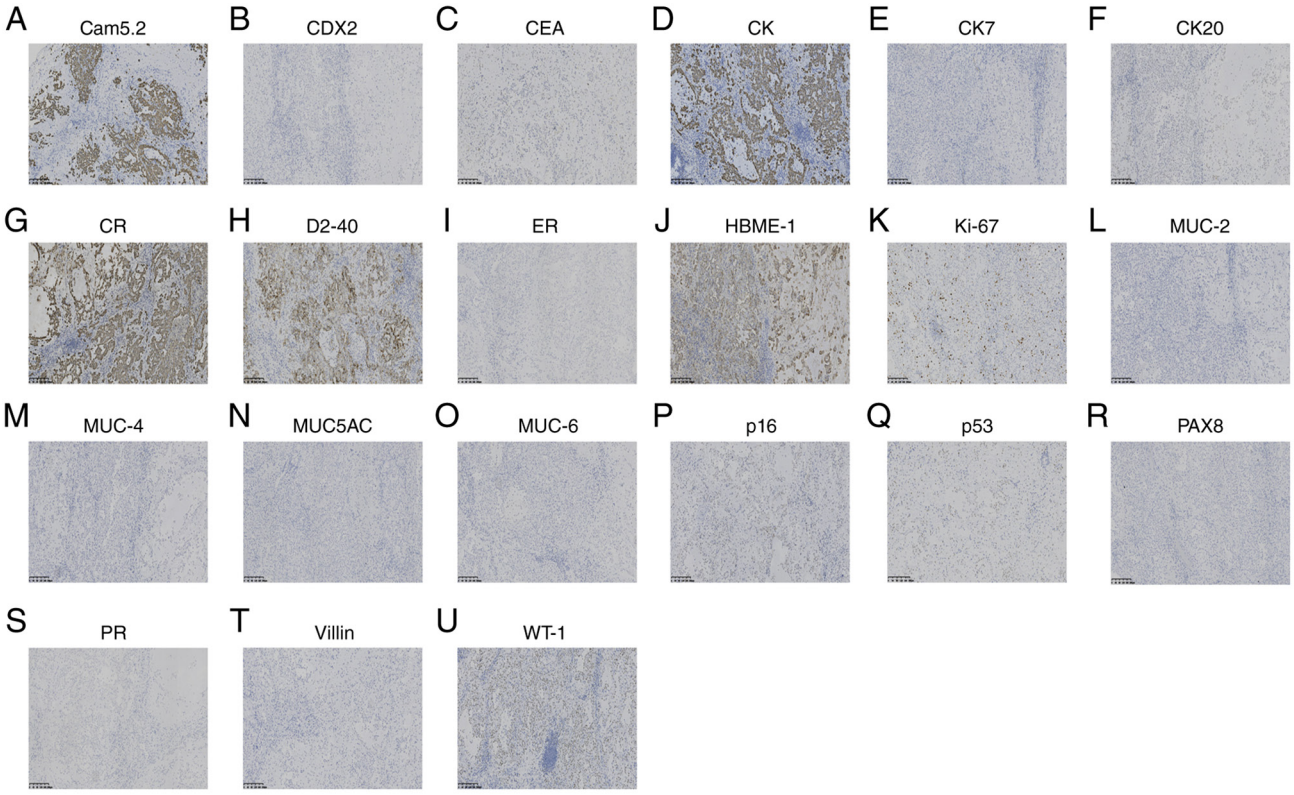

Figure 4. Immunohistochemical results. Images of staining for (A) Cam5.2, (B) CDX2, (C) CEA, (D) CK, (E) CK7, (F) CK20, (G) CR, (H) D2-40, (I) ER, (J) HBME-1, (K) Ki-67, (L) MUC-2, (M) MUC-4, (N) MUC5AC, (O) MUC-6, (P) p16, (Q) p53, (R) PAX8, (S) PR, (T) villin and (U) WT-1. CDX2, homeobox protein CDX2; CEA, carcinoembryonic antigen; CK, cytokeratin; CR, calretinin; D2-40, podoplanin; ER, estrogen receptor; HBME-1, Hector Battifora mesothelial-1; MUC, mucin; PAX8, paired box gene 8; PR, progesterone receptor; WT-1, Wilms tumor protein; CK, cytokeratin. Scale: 200 μm.

stained tumor cells in ≥1,000 cells counted in five randomly selected fields of view using ImageJ software (v1.53k, National Institutes of Health, Bethesda, MD, USA).

Immunohistochemical analysis showed that tumor cells exhibited the following expression results: Cytokeratin (CK) (+), Cam5.2(+), a Ki-67 index of 50% (indicating high proliferative activity), CK7(-), CK20(-), villin(-), homeobox protein CDX-2 (CDX-2)(-), carcinoembryonic antigen (CEA)(-) and mucin (MUC) family (for MUC-2, MUC-4, MUC5A, MUC-6) (-). The results demonstrated positive expression of calretinin and the mesothelioma-specific markers Wilms tumor protein (WT-1), Hector Battifora mesothelial-1 (HBME-1) and podoplanin (D2-40) (Fig. 3C). Representative immunohistochemical images for all markers are available in Fig. 4. These findings were consistent with a diagnosis of peritoneal mesothelioma.

The Eastern Cooperative Oncology Group-Performance Status (ECOG-PS) was 1 (13), with serum albumin at 30.8 g/l (mild hypoalbuminemia) at diagnosis in April 2022. During chemotherapy (May-December 2022), grade II nausea and vomiting occurred according to the National Cancer Institute Common Terminology Criteria for Adverse Events version 5.0 (14), resolving after symptomatic treatment. Albumin levels remained stable at 29.5-32.1 g/l and body weight was stable (no marked decline). Following disease progression (March 2023), the ECOG-PS was 2, the albumin level dropped to 26.3 g/l and mild cachexia developed (8% weight loss over 6 months). In August 2023, the ECOG-PS was 4, the albumin level was 22.1 g/l and the severe cachexia required total parenteral nutritional support.

The patient underwent targeted sequencing using a 120-gene pan-cancer panel (Shanghai Yijian Intelligent Manufacturing Life Technology Co., Ltd.). Genomic DNA was extracted from formalin-fixed paraffin-embedded clinical specimens and circulating cell-free DNA. Sample quality and integrity were validated using Invitrogen Qubit 4 Fluorometer (Thermo Fisher Scientific, Inc.) and the Agilent 2100 Bioanalyzer (Agilent Technologies) for library fragment distribution assessment, according to the manufacturer's protocols. Library preparation was performed with the Human FFPE DNA 120 Gene Mutation Detection or the Human Circulating Free DNA 120 Gene Mutation Detection Kit (both Shanghai Yijian Intelligent Manufacturing Life Technology Co., Ltd.). Sequencing was performed on the YJSeq 300 Dx Genetic Sequencer (Shanghai Yijian Intelligent Manufacturing Life Technology Co., Ltd.) using the Sequencing Reaction Universal Kit (Shanghai Yijian Intelligent Manufacturing Life Technology Co., Ltd.), in paired-end 2X 150 cycles mode (300 cycles total read length). The final library concentration was measured using the Invitrogen Qubit 4 Fluorometer, and the average library fragment size (bp) was determined using the Agilent 2100 Bioanalyzer; libraries were loaded at a final concentration of 1-20 pM. Raw sequencing data were processed and analyzed using YJ-Tumor Tumor Gene Mutation Detection Analysis Software v1.0.0 (Shanghai Yijian Intelligent Manufacturing Life Technology Co., Ltd.), which performs data preprocessing, sequence alignment, quality control, variant calling, microsatellite instability (MSI) analysis, and variant annotation. The panel covers genes associated with therapeutic

targets for mesothelioma and common solid tumors, including EGFR, anaplastic lymphoma kinase, ROS proto-oncogene 1, receptor tyrosine kinase, RAF, neurotrophic tropomyosin receptor kinase 1/2/3, programmed death ligand 1, CDK inhibitor 2A, TP53, neurofibromatosis type 1 (NF1), BRCA-1 associated protein 1 and NF2. Following bioinformatics annotation and filtering, no pathogenic single nucleotide variants, insertions/deletions, copy number variations or gene fusions were detected, and no clear targets for targeted therapy were identified. Accordingly, neither immunotherapy nor targeted therapy could be recommended for this patient.

Response evaluation criteria in solid tumors 1.1 (15) for tumor response assessment were applied throughout the present study. Following the initial pemetrexed-cisplatin chemotherapy regimen, follow-up contrast-enhanced CT revealed a 25% reduction in the sum of the largest diameters of peritoneal masses compared with baseline (decreasing from 6.0 to 4.5 cm), along with a marked decrease in ascites (from 'massive ascites' prior to treatment to 'minimal ascites'). This met the criteria for a partial response (PR) but tumor markers remained elevated (CA125 increased from 160.60 to 556.2 U/ml). During the second to sixth chemotherapy cycles, follow-up contrast-enhanced CT showed continued reduction in the sum of the largest diameters of peritoneal masses, decreasing by 55% from baseline (from 6.0 to 2.7 cm). The minimal ascites remained stable and tumor markers continued to decline (CA125 decreased from 556.20 to 156.5 U/ml), meeting the criteria for a PR. Disease progression parameters at 11 months (using the value before peritoneal perfusion therapy as the new baseline) showed that the sum of the largest diameters of peritoneal masses increased by 26% compared with the PR assessment (from 2.7 to 3.4 cm), ascites volume increased again (moderate amount), CA125 rose to 923.3 U/ml, and the patient experienced worsening abdominal pain and distension along with further weight loss (5 kg over 2 months). This met the criteria for disease progression.

During a laparoscopy, the surgical team in collaboration with the Department of Radiology assessed the patient's peritoneal cancer index (PCI) (16) with a total score of points. A PCI score >15 indicates a high tumor burden with a low probability (<30%) of achieving complete cytoreductive surgery, according to Peritoneal Surface Oncology Group International (PSOGI) (17) guidelines. The patient refused surgery and a 'non-surgical treatment' approach was selected. The patient received six cycles of chemotherapy (21-day) between May and December 2022 with 500 mg/m^2 (actual dose, 800 mg) pemetrexed on day 1 and 75 mg/m^2 cisplatin administered on days 1, 2, and 3 (40 mg daily).. The body surface area of the patient was calculated as 1.68 m^2 using the Mosteller formula (18) (height, 170 cm; weight, 60 kg). According to RECIST 1.1 (15), pemetrexed-cisplatin chemotherapy achieved a PR after six cycles, with a 55% decrease in the sum of the largest diameters of peritoneal masses (from 6.0 to 2.7 cm), but was accompanied by severe adverse events, mainly grade III myelosuppression and grade II gastrointestinal reactions. Treatment was adjusted to intraperitoneal perfusion with 30 mg cisplatin plus 300 mg bevacizumab administered on day 1 every 3 weeks in the 11th post-operative month, a compassionate-use protocol which was submitted to the hospital's Ethics Committee for special review and was approved (approval no. 2024-07-01). The patient and his family were informed of the lack of clear evidence-based support, potential risks and benefits, and then provided written informed consent. This process complies with the relevant provisions regarding compassionate use in the Declaration of Helsinki and China's Drug Administration Law. Disease progression was recorded and one additional cycle of pemetrexed and cisplatin was given in the 13th post-operative month. The patient died in September 2023 with cachexia noted as a contributing condition.

Discussion

To the best of our knowledge, the present case is the first reported instance worldwide of a road maintenance worker with long-term exposure to asphalt who developed DPM and was misdiagnosed with mesenteric panniculitis. A previous study reported the misdiagnosis of DPM as ovarian cancer or abdominal tuberculosis (3). To the best of our knowledge, the present case is the first to define the characteristic imaging evolution over 18 months during which diffuse peritoneal mesothelioma was initially misdiagnosed as mesenteric panniculitis (presenting as mild omental thickening and minimal effusion) and demonstrated typical features of DPM including diffuse omental thickening, intra-abdominal masses, and cardiophrenic angle lymph node metastasis. This progression gives imaging indications for the early clinical differentiation between the two conditions. A rare calretinin-negative immunophenotype of epithelioid DPM was identified, which accounts for 8.7% of epithelioid DPM cases (12). A triple-marker panel of WT-1, HBME-1 and D2-40 was used to avoid misconceptions based on calretinin-only testing and to refine the DPM immunohistochemical diagnostic system. Clinical evidence for the non-surgical treatment of patients with DPM with a high PCI score (27 points) who refuse surgery is provided in the study. Pemetrexed + cisplatin chemotherapy achieved a 55% decrease in the sum of the longest diameters of peritoneal masses (from 6.0 to 2.7 cm), as calculated per RECIST 1.1 criteria (15), representing tumor regression although standard doses were associated with grade III myelosuppression. A dose adjustment regimen of 600-700 mg/m^2 was proposed, filling the clinical gap of a lack of standardized treatment protocols for patients with diffuse peritoneal mesothelioma, PCI >15), and who refuse cytoreductive surgery.

A potential association between asphalt exposure and DPM was identified. An epidemiological meta-analysis by Partanen and Boffetta (19) reported a 2.3-fold increased risk of developing malignant tumors in the abdominal cavity among individuals exposed to asphalt and showed a dose-response relationship. The present patient had a 28-year history of unprotected exposure to asphalt, constituting high-risk exposure. Asphalt contains carcinogens, such as polycyclic aromatic hydrocarbons (PAHs) and benzo(a)pyrene, which cause DNA damage in peritoneal mesothelial cells by generating reactive oxygen species, a similar mechanism to that of asbestos (20). A global population study by Jin *et al* (21) showed that DPM incidence did not decrease markedly following the ban or restriction of asbestos, suggesting a potential role for non-asbestos carcinogens. The present case gives the first clinical evidence to support this conclusion.

Asbestos exposure is associated with DPM, although with a weaker association than for the more common pleural mesothelioma (17,21). The length of asbestos fibers, which may exceed the capacity for phagocytosis, causing release of reactive oxygen species (ROS) and an inflammatory and mutagenic environment, seems a likely explanation for mesothelioma occurrence at both the pleural and peritoneal sites (20,22). Less is known about the impact of genetic susceptibility at the two sites, although some predisposing polymorphisms in genes including BAP1, TP53, GSTT1 and XRCC1 have been linked to the pleural form of the disease (23). DPM is characterized by extensive proliferation along peritoneal surfaces, often adhering to organs. Typical radiological findings include diffuse peritoneal and omental thickening, abdominal masses, ascites and lymph node metastases (24).

The present patient had no history of asbestos exposure but had long been engaged in highway maintenance work. The base asphalt + modifier products commonly used in highway maintenance do not contain natural asbestos but, when heated to 160-180˚C, they release PAHs, benzo(a)pyrene and volatile organic compounds. The asphalt aggregate contains additional heavy metal impurities, such as chromium and nickel, which are classified as Group 1 or Group 2A carcinogens by the International Agency for Research on Cancer (25). Asphalt carcinogens induce malignant transformation of peritoneal mesothelial cells through two pathways (20,26). First, PAHs are metabolized by cytochrome P450 to produce ROS, causing point mutations in tumor suppressor genes, such as p53 and CDKN2A, which disrupt cell cycle regulation. Second, long-term accumulation of asphalt microparticles and heavy metals in the peritoneal cavity may trigger chronic aseptic inflammation, activate the NF-κB pathway and create a tumor-promoting microenvironment, leading to abnormal proliferation and invasion of mesothelial cells. Occupational exposure to potential carcinogens, such as asphalt, may have occurred in the present case (19,21,27) and contributed to the disease. However, causal evidence is lacking and further epidemiological and mechanistic studies are needed to validate the association. Mesothelioma may involve the pleura, peritoneum and pericardium with pleural mesothelioma being the most common (accounting for 80-85%), followed by peritoneal mesothelioma (15%) and pericardial mesothelioma being rare (<1%) (21,28). The present case involved peritoneal involvement without manifestations of pleural or pericardial involvement (chest CT showed no pleural thickening or pericardial effusion), consistent with the typical site characteristics of DPM.

An accurate diagnosis of DPM relies on histopathological examination, including morphological observation of biopsy specimens and immunohistochemical verification. Tumor cell morphological features, such as a tubular arrangement of epithelioid cells and spindle-shaped morphology of sarcomatoid cells, must be examined microscopically and combined with positivity for at least two mesothelioma-specific markers (such as WT-1, D2-40 and HBME-1) and two negative markers (such as CEA and thyroid transcription factor 1) while excluding metastatic carcinoma or other peritoneal tumors (29,30). The following tests for markers associated with metastatic cancer of gastrointestinal and female reproductive tract origin were performed for the present case with the following results: CK7(-), CK20(-), CDX2(-), p16(-), paired box gene 8 (PAX8)(-), p53 (wild-type, positivity rate <10%), estrogen receptor (ER)(-) and progesterone receptor (PR)(-). Combined with the previous negative results for CEA and MUC family, common malignant tumors with peritoneal metastasis, such as gastrointestinal adenocarcinoma, may be excluded, meeting the core requirement of 'exclusion of metastatic cancer' in the 2023 IMIG Mesothelioma Diagnostic Guidelines (29). The annual age-adjusted incidence of secondary peritoneal metastases is 99.00/1,000,000 persons, compared with only 4.36 per 1,000,000 persons for primary peritoneal malignancies including mesothelioma (31). The combination of the aforementioned markers is key to the differential diagnosis, since tumors of gastrointestinal origin are often CK7/CK20/CDX2-positive and tumors of female reproductive tract origin are frequently associated with abnormal expression of PAX8, ER/PR or p16 (30). All of these were negative for the present patient, and the mesothelioma-specific phenotype of WT-1, HBME-1 and D2-40 confirmed the accuracy of the DPM diagnosis. Tumor, lymph node involvement and metastasis staging must factor in the PCI, which evaluates the size and distribution of peritoneal tumors (32). The American Cancer Society has reported that patients have markedly improved prognosis in the earlier stages, with 5-year survival rates being 87% at stage I, 53% at stage II and 29% at stage III (33,34). Laparoscopic biopsy allowed a definitive diagnosis for the present case. A nodular lesion was identified at the site of the laparoscopic port in the right lower quadrant of the abdominal wall at 1 month post-surgery, indicating the occurrence of implantation metastasis. This observation indicates the aggressive nature of this type of tumor and may suggest a particularly poor prognosis for the patient (35).

Standardized treatment protocols have yet to be established for DPM. CRS + HIPEC is recommended by the PSOGI, with the aim of removing visible tumors and separate adhesions to improve HIPEC efficiency (21). This therapeutic strategy carries a notable rate of grade 3-4 complications; however, the 5-year survival rate has been extended to 42% from the baseline of <15% for patients treated with non-CRS + HIPEC regimens (36). Systemic chemotherapy protocols for DPM have their basis in those established for pleural mesothelioma, and pemetrexed in combination with cisplatin, carboplatin or gemcitabine is generally preferred, especially for patients who are not candidates for CRS + HIPEC. Intraperitoneal chemotherapy increases local drug concentrations, giving more efficacious treatment of peritoneal surface malignancies and reducing systemic side effects (37). Promising results were reported for the treatment of 26 patients with unresectable DPM treated with pressurized intraperitoneal aerosol chemotherapy. The treatment achieved clinical symptom improvement in 32% of patients and ascites control in 46%; 54% of patients underwent successful CRS + HIPEC, with a median progression-free survival of 33.5 months in resected patients vs. 7.4 months in unresected patients (38). Although the present patient initially achieved a PR with pemetrexed at a close to standard dose, grade III myelosuppression developed. Therefore, patients with DPM who have a high PCI score and refuse surgery are recommended a dose-adjusted regimen (such as pemetrexed 600-700 mg/m^2) to balance efficacy and safety.

No targets suitable for immunotherapy were identified in the present case by genetic testing, rendering immunotherapy inappropriate. The patient received six cycles of chemotherapy with pemetrexed (800 mg on day 1) combined with cisplatin (40 mg on days 1-3). Although a PR was achieved (35% reduction in peritoneal mass; CA125 decreased from 160.6 to 156.5 U/ml), Grade III bone marrow suppression (lowest neutrophil count $0.8x10^9$/l) and grade II gastrointestinal reactions developed. This indicates that while the regimen is effective for low-grade epithelioid DPM, enhanced toxicity management is required (for example, prophylactic use of granulocyte colony-stimulating factor and 5-HT3 receptor antagonists). The reasons for the ineffectiveness of intraperitoneal perfusion therapy could be due to the regimen being adjusted to intraperitoneal cisplatin (30 mg) + bevacizumab (300 mg) at 11 months while disease progression occurred within 1 month. This treatment regimen constitutes off-label compassionate use. The treatment decision was based on two factors. First, the patient's disease progressed after six cycles of pemetrexed plus cisplatin chemotherapy and no standard second-line regimen was available. Second, the anti-angiogenic mechanism of bevacizumab may have a synergistic effect on malignant peritoneal tumors and a small number of studies (39,40) on intraperitoneal administration for ovarian cancer and colorectal cancer with peritoneal metastases have demonstrated a potential for local tumor control (38). The tumor may have extensively invaded extraperitoneal tissues (such as peritoneal wall puncture site metastasis), rendering local drug concentrations insufficient to effectively cover the lesions, and there exists a lack of medical evidence for bevacizumab in the intraperitoneal treatment of DPM (38). This suggests that careful evaluation of the extent of tumor invasion should be made when selecting intraperitoneal perfusion regimens. The present case suggests that patients with DPM with high PCI scores (>15) who refuse surgery should have initial chemotherapy that prioritizes 'dose-adjusted regimens with manageable toxicity' (for example, reducing pemetrexed to 600-700 mg/m^2) while monitoring tumor markers (CA125 and Cyfra 21-1) and imaging changes to identify disease progression early and adjust treatment strategies.

There were several limitations to the present study. Current international guidelines recommend CRS + HIPEC as the first-line treatment for resectable DPM with patients receiving this regimen achieving a 5-year survival rate of 42% (36). By contrast, the present case received only systemic chemotherapy (pemetrexed + cisplatin) and intraperitoneal perfusion therapy (cisplatin + bevacizumab), falling within the second-line treatment category for 'unresectable/ surgery refusal'. The treatment efficacy (13-month survival) reflects outcomes only for this patient population and cannot be extrapolated to patients with DPM eligible for CRS + HIPEC. In addition, due to the absence of efficacy data from a CRS + HIPEC treatment group (such as tumor burden reduction and PFS), the outcome in this case of PR to pemetrexed-cisplatin chemotherapy followed by progression does not validate the superiority of 'non-surgical treatment vs. surgery combined with HIPEC' and acts as a reference only for patients similarly unable to undergo CRS + HIPEC. There are also ethical review limitations: The intraperitoneal bevacizumab combined with cisplatin regimen has not undergone institutional review board approval. Informed consent was obtained from the patient and their family but strict adherence to ethical review procedures is still required in subsequent clinical practice to ensure the compliance of the treatment regimen. The purulent fluid found in the peritoneal cavity during laparoscopy was not subjected to cytological examination or microbiological culture and the possibility of a concurrent infection cannot be ruled out. The diagnosis of malignancy could not be supported by the cytological analysis of peritoneal fluid, which may have impacted clinical decision-making. The present PCI score of 27 points was determined from combined assessment by laparoscopic examination and radiologists (32). However, laparoscopic PCI assessment has limitations due to incomplete visualization of the abdominal cavity, particularly regarding tumor involvement in hidden areas such as subphrenic and retrohepatic regions, and may lead to underestimation of the true tumor burden (34). By contrast, open surgery allows for complete abdominal exploration and yields a more accurate PCI score (41). Furthermore, there is a lack of high-level (randomized controlled trials, systematic reviews, or meta-analyses) evidence supporting intraperitoneal bevacizumab therapy for DPM, and the lack of response observed in the present case underscores the uncertainty of this treatment regimen (37). The use off-label intraperitoneal cisplatin + bevacizumab was made to inform potential treatment options for patients with advanced disease and may serve as a reference for future studies.

In conclusion, the present study presents a case of DPM initially misdiagnosed as mesenteric panniculitis, illustrating the difficulties of the early diagnosis and treatment of DPM. More effective therapeutic interventions are needed which may be diversified by future developments in immunotherapy and genotyping techniques to inform future treatment strategies for DPM.

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Availability of data and materials

The data generated in the present study may be found in Figshare under accession number 10.6084/m9.figshare.32287683 at the following URL: https://doi.org/10.6084/m9.figshare.32287683.

Authors' contributions

YC conceived and designed the study, collected, analyzed and interpreted data, participated in clinical management and perioperative care, constructed the figures and tables and wrote the original manuscript. QF performed the histopathological and immunohistochemical examination. JX collected the clinical and follow-up data, supervised the clinical management, and edited the manuscript for important intellectual content. RW

performed and interpreted all computed tomography images, provided formal radiological analysis, and contributed to the imaging methodology. YM collected and analyzed data. YL designed the study, supervised the entire research process, and administered the project. YC and YL confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of The First People's Hospital of Xiaoshan District (Hangzhou, China) in accordance with the Declaration of Helsinki (approval no. 2024-07). Written informed consent was obtained from the patient's family.

Patient consent for publication

The patient provided written informed consent for the publication of the case report and associated images.

Competing interests

The authors declare that they have no competing interests.

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