

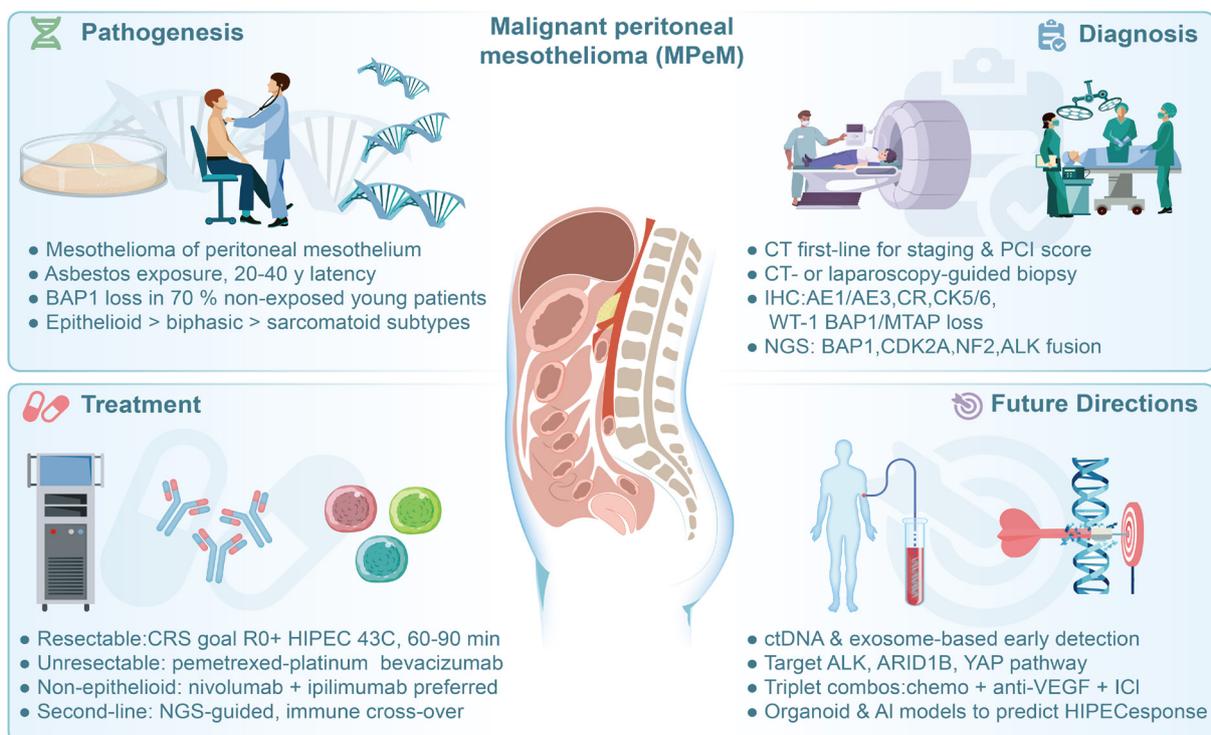
Expert consensus on the diagnosis and treatment of malignant peritoneal mesothelioma

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Received: November 19, 2025 Accepted: December 23, 2025
<https://dx.doi.org/10.65364/iomj.2026.01>

GRAPHICAL ABSTRACT



Public summary

- Malignant peritoneal mesothelioma is a rare cancer of the abdominal lining.
- Long-term asbestos exposure and certain gene changes (BAP1) are the main causes.
- Today, doctors combine surgery, heated chemotherapy, immunotherapy and precision medicine to treat it, and they are working on blood tests and AI to catch it earlier and tailor care for each patient.

Review

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How to cite this article: Li J, Hu J, Xu C, Ji Q, Wang W, Guo Z, Song Z, Li Z, Liu A, Yu J, Wang Z, Fang W, Zhang Y, Zhang S, Cai X, Liu A, Lv T, Miao L, Min L, Chen Y, Yuan J, Jiang Z, Huang L, Wang F, Pu X, Lin R, Rao C, Lv D, Ji Y, Liang X, Yu Z, Li X, Tang C, Zhou C, Zhang J, Ma X, Qiu L, Guo H, Chu Q, Meng R, Wu J, Fu J, Zhou J, Zhu Z, Zhang H, Piao Y, Chen X, Liu X, Yao Y, Zhang J, Pan W, Pang F, Wu F, Wei Q, Wang L, Ye S, Zhang Y, Lin X, Cai J, Feng J, Li J, Jiao X, Li K, Feng H, Wang L, Du Y, Song B, Shi X, Li W, Niu X, Huang J, Wang Y, Feng Y, Zhang Y, Sun P, Dai E, Wang H, Wang D, Hao Y, Wang Z, Wan B, Lv D, Yang S, Shi L, Li B, Zhang Z, Li Z, Liu Z, Yang N, Wu L, Li H, Wang L, Li M, Chen X, Wang G, Wang J, Lou F, Li Y, Fang M, Zhang Y, Zhu X, Wang K, Ma S, Song Y, Zhu Y, Lu Y, Wang Q, Chen T, Shen P. Expert consensus on the diagnosis and treatment of malignant peritoneal mesothelioma. *Int Open Med J.* 2026;1:7-31. <https://dx.doi.org/10.65364/iomj.2026.01>

Received: 19 Nov 2025 **Accepted:** 23 Dec 2025 **Published:** 16 Jan 2026

Academic Editor: Sinbad Xia **Copy Editor:** Huanliang Wu **Production Editor:** Huanliang Wu

Abstract

Malignant peritoneal mesothelioma (MPeM) is a malignant tumor originating from the peritoneum. In recent years, the incidence of MPeM has been increasing. Because MPeM is insidious in onset and of strong local invasiveness, and most patients are found in advanced stage, early screening and treatment of the population at the high risk of malignant mesothelioma are of vital importance. The main treatment methods for MPeM include cytoreductive surgery, hyperthermic intraperitoneal chemotherapy, systemic chemotherapy and immunotherapy. To promote the homogenization and normalization of the diagnosis and treatment of malignant peritoneal mesothelioma, based on existing clinical research evidence, the Chinese Alliance of Research for Mesothelioma (ChARM) formulated the National Expert Consensus on the Clinical Diagnosis and Treatment of Malignant Peritoneal Mesothelioma in combination with expert opinions nationwide. The contents of expert consensus cover epidemiology, diagnosis, treatment and prognosis follow-up.

Keywords: Malignant peritoneal mesothelioma, diagnosis, therapy, consensus

INTRODUCTION

Malignant peritoneal mesothelioma (MPeM) is a rare primary malignant tumor originating from peritoneal mesothelial cells, accounting for 7%-30% of all mesotheliomas. The peritoneum is the second most common site of mesothelioma after the pleura. MPeM can be categorized into two types: diffuse type and

localized type, of which the former is more common^[1]. MPeM accounts for about 10%-15% of all mesotheliomas, with an annual incidence of 1-2 per million. MPeM often invades abdominal organs, with the symptoms at advanced stage including abdominal distension, nausea, vomiting, intestinal obstruction, etc.^[2]. Previously, little was known about the pathogenesis, biological behavior, and natural history of malignant peritoneal mesothelioma. Most cases were diagnosed at advanced stage, limiting treatment options, resulting in poor efficacy and prognosis, and most patients died within one year after diagnosis^[3]. Malignant mesothelioma is closely related to occupational or environmental inhalation of asbestos fibers and other elongated mineral particles (EMPs), and the latency period may be as long as 20-40 years, with the longest reaching 71 years^[4].

The Chinese Alliance of Research for Mesothelioma (ChARM) began to be established in 2022 and was officially founded in 2023. It has 52 member institutions and is committed to studies on mesothelioma with Chinese characteristics. The database established at the beginning of the establishment of ChARM has laid a solid foundation for the research work. The retrospective database has included data on mesothelioma from 1990 to 2023, with information about 3,947 cases as of December 2023. In order to further deepen the study on mesothelioma, a prospective database was built and launched in 2023, covering pleural mesothelioma, peritoneal mesothelioma, mesothelioma of the tunica vaginalis testis and pericardial mesothelioma. As of December 2023, the information about a total of 2,452 cases has been entered. To date, more than 6 domestic and foreign articles have been published based on retrospective and prospective databases, and the Chinese expert consensus on the diagnosis and treatment of malignant pleural mesothelioma (2023), the Expert Consensus on the Diagnosis and Treatment of Malignant Mesothelioma of the Tunica Vaginalis Testis (2025) and the Expert Consensus on the Diagnosis and Treatment of Malignant Pericardial Mesothelioma (2025) have been issued^[5-7].

At present, there are several guidelines and consensus both at home and abroad that provide guidance for the standardized clinical diagnosis and treatment of malignant peritoneal mesothelioma^[8-11]. However, in clinical diagnosis and treatment practice, there are still many issues that need to be confirmed and standardized urgently, especially in the standardized application of new diagnostic techniques and treatment options. Therefore, based on the previous three mesothelioma expert consensus, the ChARM organized domestic experts in the field of peritoneal mesothelioma to conduct in-depth discussions and exchanges by referring to existing literature and guidelines, ultimately forming this expert consensus, with the aim of converting the new progress in the diagnosis and treatment of malignant peritoneal mesothelioma into tangible benefits in clinical application^[12].

EPIDEMIOLOGICAL DATA AND ETIOLOGY

Consensus 1: The definite diagnosis rate of malignant peritoneal mesothelioma is low, while its misdiagnosis rate is high. Screening for malignant peritoneal mesothelioma is not currently recommended (Recommended).

Epidemiological data

The latest global cancer burden data in 2022 released by the International Agency for Research on Cancer (IARC) of the World Health Organization showed that there were 30,618 new cases of malignant mesothelioma worldwide in 2022, accounting for 0.2% of the global new malignant tumors, and 25,372 deaths, accounting for 0.3% of the global deaths from malignant tumors^[13]. Peritoneal mesothelioma occurred more frequently in males, with a male-to-female incidence ratio of (4-5):1. An epidemiological study conducted by domestic scholars in MPeM patients in China from 2000 to 2013 found that the age-standardized incidence rate of MPeM in 2013 was 1.71 per million, with a yearly increasing trend in incidence^[14].

Etiology

At present, the carcinogenic factors associated with mesothelioma include chemical carcinogenic factors, such as asbestos and other mineral fibers, physical carcinogenic factors, such as chronic peritonitis and therapeutic radiation, and biological carcinogenic factors. Among them, asbestos is the main carcinogen, but only 33% ~ 50% of patients have a history of asbestos exposure^[15]. Both occupational asbestos exposure and non-occupational asbestos exposure are unsafe for humans, especially adolescents and children^[16].

The lifetime risk of developing mesothelioma among asbestos practitioners is as high as 10% and the latency period from asbestos exposure to the occurrence of mesothelioma is 20-40 years^[17,18]. However, the association between asbestos exposure and MPeM is weaker than that between asbestos exposure and pleural mesothelioma, particularly in women^[18,19]. The cumulative exposure required for asbestos-induced MPeM, compared with asbestos-induced pleural mesothelioma, is generally higher^[20,21]. As asbestos use has not been completely banned in China, and the latency period of the asbestos-induced disease is long, it is indicated that the incidence of mesothelioma may continue rising.

Although asbestos exposure is the leading clear risk factor, there have been case reports revealing the occurrence of MPeM within radiotherapy fields, only with a small total number of cases and unclear risk degree^[22,23]. An analysis to investigate the association between radiation exposure and mesothelioma found that cancer patients whose peritoneum was directly exposed to external radiation showed a slight increase in the risk of peritoneal mesothelioma, whereas patients exposed to scattered radiation due to radiotherapy at other sites did not show any increase^[24]. MPeM can also occur in the context of chronic peritonitis^[25].

BRCA-associated protein 1 (BAP1) mutations are common in MPeM patients, and this tumor suppressor gene has also been reported in pleural mesothelioma^[26]. Mice with BAP1 germline heterozygous mutation have been shown to have increased susceptibility to MPM^[27]. Based on this, it is speculated that individuals with BAP1 mutations are predisposed to malignant mesothelioma upon asbestos exposure. Some patients with mesothelioma carry both BAP1 mutations and other germline mutations that induce other cancers^[28]. The significance of identifying these patients lies in the fact that both the patients and their relatives may be prone to other cancers, and it is beneficial for them to conduct genetic counseling and cancer screening.

In China, Chuxiong, Yunnan and Ningbo, Zhejiang are two areas with a high incidence of malignant mesothelioma. Although released epidemiological data in Chuxiong, Yunnan were limited, on-site investigations indicated that the disease types and sexes of malignant mesothelioma in Chuxiong were highly consistent with foreign data, but Yuyao and Cixi, Ningbo, were the opposite of Chuxiong, with peritoneal mesothelioma being predominant^[29,30]. The in-depth investigation found that the predominance of peritoneal mesothelioma in Yuyao and Cixi was related to the occupational environment, and in Zhejiang, asbestos processing was mainly carried out in small workshops where both diet and daily life took place, with a significantly increased probability of accidental ingestion of asbestos, in addition to inhaling asbestos; in Yunnan, mainly relying on mining, diet and daily life took place at home and the mine field was just a workplace, with a high probability of inhalation and a low probability of accidental ingestion^[31].

DIAGNOSIS

Consensus 2: Contrast-enhanced abdominal computed tomography (CT) is currently the preferred imaging method for diagnosing malignant peritoneal mesothelioma. Imaging methods such as magnetic resonance imaging (MRI) and positron emission tomography (PET)/CT have their own characteristics and advantages and may be used when conditions permit (Strongly recommended).

Consensus 3: CT-guided biopsy is recommended as the standard operation, while ultrasound-guided biopsy or laparoscopy can serve as an effective supplementary method in specific situations (Strongly recommended).

Consensus 4: Histopathology is the gold standard for the definite diagnosis of malignant peritoneal mesothelioma, and the histological subtype and Ki-67 proliferation index should be included in the pathological report. These subtypes include epithelioid, sarcomatoid, and biphasic types. Pathological evaluation of peritoneal malignant mesothelioma should be performed based on adequate tissue samples, and cytopathological examination of ascitic fluid is not recommended for diagnosis. The common markers for mesothelioma origins include AE1/AE3, Calretinin, CK5/6, WT-1, and D2-40, and the markers for prognosis include BAP1, MTAP, P16 (CDKN2A), EZH2, etc. It is recommended that at least 3 or more mesothelioma markers be used simultaneously. Markers for differentiation (mainly include those for gastrointestinal tumors, kidney cancer, and primary peritoneal carcinoma) include Claudin-4, MOC-31, BG8, CEA, B72.3, CDX-2, and Ber-EP4; PAX-2, PAX-8, and RCC; CA125, and HE4 (Strongly recommended).

Consensus 5: Common genetic alterations in malignant peritoneal mesothelioma include BAP1, CDKN2A, LATS2, NF2, SETD2, and TP53. For young patients without any history of asbestos exposure and with a family aggregation of tumors, *BAP1* gene testing is recommended (strongly recommended); ALK rearrangement has been found in some MPeM patients and can be considered as a genetic testing item (Recommended).

Clinical diagnosis

MPeM is a rare invasive disease. Most MPeM patients have the manifestation of diffuse peritoneal involvement, while a minority have localized diseases, with distinct clinical manifestations and natural courses^[32]. Diffuse MPeM is highly invasive, and patients with localized MPeM usually have a favorable prognosis after total resection. MPeM patients have no specific symptoms or vital signs. Common symptoms include abdominal distention, abdominal pain, weight loss, dyspnea and chest pain. The average interval from symptom onset to diagnosis is approximately 5 months^[33]. The clinical manifestations of diffuse MPeM are associated with ascites or intra-abdominal tumor progression^[34]. Common complaints include abdominal distention and/or increased abdominal circumference, abdominal pain or discomfort, nausea, anorexia, and weight loss. Gastrointestinal complications, such as intestinal obstruction, typically occur in the advanced stage of the disease. Because symptoms complained of are non-specific, many patients are already in advanced stages at the time of diagnosis. Abdominal distention (increased abdominal circumference) is the most common initial symptom, occurring in 30% to 80% of patients^[35]. Pain is the second most common initial symptom, occurring in 27% to 58% of patients^[36]. In most patients, the pain is diffuse and non-specific. Localized MPeM presents as a focal, borderline mass that may locally invade and extend to adjacent organs, but generally does not spread diffusely throughout the peritoneal cavity. The patients may complain of localized abdominal pain or palpable abdominal/pelvic mass^[37].

MPeM may occasionally metastasize to lymph nodes in the abdominal and pelvic cavities. Lymph node metastases occur in 20% to 28% of patients undergoing cytoreductive surgery due to diffuse MPeM^[38]. Distant metastases are rare^[39]. In a multi-institutional registry data-based study, 294 diffuse MPeM patients were included, among whom only 12 (4%) developed extra-abdominal metastases^[40]. Patients with MPeM may present with a variety of paraneoplastic manifestations: pyrexia, thrombocytosis, malignancy-related thrombosis, and hypoglycemia^[41].

Imaging diagnosis

Correct staging of malignant peritoneal mesothelioma requires a combination of imaging examinations and invasive exploratory procedures, which can be categorized into non-invasive and invasive examinations. Non-invasive examinations include CT, MRI, ultrasonography, PET-CT, or PET-MRI; invasive examinations include peritoneal biopsy (CT-guided or ultrasound-guided) and laparoscopy.

Enhanced CT of the whole abdomen remains the preferred imaging method for diagnosis, staging, surgical assessment, treatment monitoring, follow-up and complication evaluation of MPeM^[42]. Other imaging techniques such as ultrasonography, MRI, PET-CT and PET-MRI also have their respective advantages and features. It should be noted that in clinical practice, a single imaging method sometimes cannot meet the needs for diagnosis and treatment of MPeM, and the combination of two or more methods can improve the accuracy of diagnosis and evaluation of MPeM. Therefore, methods may be recommended to be used on the basis of the patient's condition (strongly recommended, Level II evidence).

Ultrasonography is recommended for the extraction and localization of peritoneal fluid.

If conditions permit, it is recommended to choose to complete PET-CT or whole abdominal MRI [especially diffusion-weighted imaging (DWI) sequences], which is helpful for accurately assessing tumor burden and extent. From a health economics perspective, PET-CT is not recommended as a routine follow-up method for MPeM patients due to its high cost.

Imaging manifestations of MPeM

The imaging manifestations of MPeM vary depending on the severity of the disease. The relatively characteristic imaging signs of MPeM include the following: widely and unevenly thickened and strengthened peritoneum, accompanied by soft tissue masses or nodules in the peritoneum, omentum, and mesentery, with cases where lesions may fuse into patchy masses, invading surrounding tissues and organs, greater omental lesions may present as “cake-like” changes, and mesenteric lesions may present as “stellate-like” changes; most cases are accompanied by ascitic fluid; calcification is uncommon, although pleural plaques with calcification are common in pleural mesothelioma^[43-45]. MPeM includes three different morphologic subtypes: (1) Wet type, which is common, manifested as diffuse uneven thickening of peritoneum with scattered nodular shadows, mostly with moderate to large amount of ascitic fluid; (2) Dry type, which is rare, manifested as a large peritoneal mass, generally without ascitic fluid; (3) Mixed type, featuring a combination of the above two manifestations^[42]. In terms of imaging, MPeM should be differentiated from such lesions as metastatic peritoneal carcinoma, primary peritoneal carcinoma and peritoneal tuberculosis.

CT

Contrast-enhanced CT has the advantages such as easy operation, large coverage, low cost and easy interpretation. The primary objective of imaging examination is to assess the overall tumor burden of MPeM, and the peritoneal cancer index (PCI) score helps quantitatively assess the disease burden of peritoneal lesions^[43]. Contrast-enhanced abdominopelvic CT is the preferred initial method for the diagnosis and evaluation of MPeM. Contrast-enhanced abdominopelvic CT has important clinical significance in the detection, staging, determination of disease extent, resectability assessment and follow-up of MPeM lesions^[44], which can meet the clinical needs of most patients. However, contrast-enhanced CT also has limitations in the assessment of MPeM, particularly its low sensitivity for detecting small peritoneal lesions (e.g., those located on the surface of the small intestine, mesentery and peritoneum); in addition, in the absence of ascitic fluid, it has low sensitivity for displaying smaller metastases in the hepatic integument/diaphragm. Therefore, in clinical practice, contrast-enhanced whole-abdominal MRI (which must include DWI sequences) or PET-CT is recommended for comprehensive assessment.

MRI

MRI has advantages such as high soft-tissue resolution, multisequencing, and multi-parameter imaging, particularly higher sensitivity for detecting small tumors such as those on the diaphragm and intestinal tube surfaces in combination with DWI sequences, and can improve the accuracy of tumor staging, preoperative assessment, and postoperative recurrence monitoring^[46]. MRI also has higher accuracy in assessing the relationship between lesions and surrounding tissues and organs, and can precisely guide the formulation of surgical plans. However, MRI also has corresponding clinical application limitations, such as long examination time (difficult to be tolerated by patients in poor condition), susceptibility to motion artifacts (e.g., intestinal peristalsis and respiratory movement may affect the display of lesions in mesentery and diaphragm), and high difficulty in image interpretation.

PET/CT

Currently, studies on PET/CT in peritoneal mesothelioma are limited. Available studies showed that PET/CT sensitivity, specificity, and accuracy for diagnosing peritoneal mesothelioma were within 86%-92%, 83%-89%, and 87%-89%, respectively^[47,48]; most (approximately 91.7%) malignant peritoneal mesotheliomas presented with high FDG uptake, with SUVmax ranging from 0 to 16.77 with an average of 7.32 ± 4.05 ^[49]. As a whole-body metabolic imaging method, PET/CT can detect more extensive lesions than other imaging methods, be used to assess the activity of malignant peritoneal mesothelioma, and guide biopsy site selection to increase positive rates in biopsy.

Imaging assessment of MPeM resectability

There is still a lack of unified and standardized imaging assessment models for MPeM, and more studies are needed to achieve standardization. It is recommended to firstly perform clinical staging with contrast-enhanced CT of the chest and abdomen, especially preoperative CT, which can be used to assess systemic metastasis and peritoneal metastasis, and evaluate CT-PCI. The imaging information that needs to be focused on when a surgical plan is formulated includes: description of peritoneal mass (size, morphology, extent, and involvement of surrounding organs), PCI score, volume of ascitic fluid, abdominal wall involvement, small intestinal and mesenteric diseases, extra-abdominal metastases, and sites unsuitable for surgical resection. Preoperative clarification of CT imaging features is helpful for screening those suitable for complete cytoreductive surgery. The presence of lesions at the following sites indicates a low possibility of achieving satisfactory cytoreduction: including tumor involvement of the abdominal cavity, around the portal vein, beside the diaphragm, the pelvic lateral wall, the retroperitoneal lymph nodes, as well as the sacrum and pelvic lateral wall; tumor-induced biliary obstruction; and stellate-like mesentery involving most of the proximal small intestine^[43,50,51].

Pathological diagnosis

Malignant peritoneal mesothelioma is a group of heterogeneous tumors, and its characteristic histological manifestations are similar to those of pleural mesothelioma. According to the classification criteria for pleural tumors issued by World Health Organization (WHO) in 2021, the histological subtypes of malignant peritoneal mesothelioma mainly include epithelioid type, sarcomatoid type and biphasic type, of which the epithelioid type is the most common, and the diagnosis of biphasic MPM requires that both epithelioid and sarcomatoid components be $> 10\%$ ^[52]. Tubulopapillary (well differentiated) mesothelioma is a rare subtype, more common in the peritoneum (rather than the pleura). In all pathological diagnoses, the above-mentioned major pathological subtype diagnoses should be provided. The pathological detection techniques for malignant peritoneal mesothelioma mainly include immunohistochemistry, fluorescence in situ hybridization (FISH), and DNA + RNA next generation sequencing (NGS).

Complete clinical information of patients, such as occupational exposure history, imaging manifestations, tumor history and treatment history, should be provided when specimens are submitted. Incomplete clinical information will affect the initial judgment, specimen handling, sampling procedures, and subsequent ancillary analysis.

There are various types of samples used for diagnosis, including laparoscopic surgical specimens, open surgical specimens, CT-guided coarse needle biopsy specimens, ultrasound-guided coarse needle biopsy specimens, fine needle aspiration cytology specimens and ascitic fluid exfoliative cytology specimens. Peritoneal biopsy, usually performed by laparoscopy or CT- or ultrasound-guided percutaneous biopsy, is the primary method for specimen acquisition. For patients who may undergo surgery, single-port thoracoscopy at the potential incision is recommended.

MPeM often presents with peritoneal fluid at initial diagnosis. Ascitic fluid cytology is an initial diagnostic procedure that is easy to be performed, and is also one of the methods for early diagnosis of malignant pleural mesothelioma. However, due to the low sensitivity of cytology and the fact that sarcomatoid mesothelioma cells typically do not shed into the serous cavity, ascitic fluid cytology is not routinely recommended as a basis for definite diagnosis. However, for patients from whom peritoneal lesion tissues cannot be obtained, the diagnosis of MPeM can be made by preparing cell paraffin blocks for immunohistochemistry and FISH analysis, in combination with clinical, imaging and/or surgical examination, provided that the mesothelioma cells are sufficient in quantity and representative. The term “atypical mesothelial hyperplasia” may be used when the cellular morphology shows varying degrees of atypia (usually low grade) but the degree of malignancy cannot be determined. However, this is not sufficient to diagnose MPeM. Significant sampling bias may also occur in fine needle biopsy, often with false negative results and low accuracy, so fine needle biopsy is not routinely recommended as the basis for sample diagnosis^[53,54].

The immunohistochemical markers to be selected should at least include cell lineage origin markers, benign and malignant lesion judgment markers and differential diagnosis markers. The common first-line markers for judging the mesothelial origins include Pancytokeratin (AE1/AE3 and CAM 5.2), cytokeratin 5/6, Calretinin (CR), Podoplanin/D2-40, and Wilms tumor-1 (WT-1). It is essential to select, at a minimum, antibodies with a high specificity for mesothelial lineage in clinical application. Secondary markers include mesothelin (MSLN), thromboxane, caveolin, tenascin-X, and collagen III (COL3A1), but they are less frequently used due to their low specificity and sensitivity.

For the judgment of benign and malignant mesothelial hyperplasia lesions, although Glucose Transporter 1 (GLUT1), Insulin Like Growth Factor 2 mRNA Binding Protein 3 (IGF2BP3)/IGF-II mRNA Binding Protein 3 (IMP3), Desmin and Epithelial Membrane Antigen (EMA) can be used to assist in differential diagnosis, the latest markers are mainly the three protein molecules, BAP1, MTAP and P16 (CDKN2A), which are almost 100% lost in malignant MPeM, but almost all of them are positively expressed in benign reactive mesothelial hyperplasia^[55-57]. EZH2 can also be used as a reliable marker to differentiate benign mesothelial hyperplasia from malignant mesothelioma^[58].

It is recommended to use at least two mesothelioma markers and two carcinoma markers, and the diagnosis should be made by pathologists experienced in diagnosing MPeM^[59].

Sarcomatoid mesothelioma usually does not express any typical mesothelioma markers, but positive keratin may be helpful for the diagnosis of sarcomatoid mesothelioma. When other metastatic adenocarcinoma is

differentiated, in addition to selecting adenocarcinoma markers (MOC31, BerEP4, BG8, B72.3 and CEA), organ-specific markers, such as estrogen receptor (ER), progesterone receptor (PR), GCDFP15 and Mamaglobin for breast cancer; PAX8, PAX2, RCC and CD15 for kidney cancer or PAX8, ER, PR, CA125 and HE4 for ovarian cancer, should also be included. It should be noted that most epithelioid mesotheliomas express GATA3, and some may also show positive PAX8. In immunohistochemistry, BAP1 and EZH2 are reliable markers for differentiating benign mesothelial hyperplasia from malignant mesothelioma^[55,56,58].

Molecular diagnosis

Currently, there is a lack of omics big data on MPeM genome and others, but existing studies have shown that the gene mutation spectrum of MPeM is similar to that of MPM, only with some differences in mutation frequency. According to TCGA data, the common somatic mutation genes in MPM include *BAP1*, *NF2*, *CDKN2A*, *TP53*, *LATS1/2*, and *SETD2*^[60]. *BAP1* is the most common mutant gene in MPM. In patients with MPeM, no history of asbestos exposure, younger age, and a previous history of tumors, the *BAP1* gene mutation rate is approximately 70%. Therefore, *BAP1* genetic testing is firstly recommended in MPeM molecular testing^[61,62].

CDKN2A mutation is associated with a poor prognosis, and its positive rate is nearly 100% in sarcomatoid mesotheliomas^[63]. Although the specificity of *BAP1* and *CDKN2A* gene mutations for diagnosing malignant mesothelioma is not 100%, they are helpful for differentiating MPM from benign pleural lesions. Homozygous deletion of *CDKN2A* can be detected by p16 FISH^[64]. It is helpful for the diagnosis of mesothelioma in situ to detect the expression loss of *BAP1* and/or *MTAP* via immunohistochemistry, and to detect the homozygous deletion of *CDKN2A* via FISH^[65]. Approximately 50% of patients harbored heterozygous or homozygous deletion mutations of *NF2*^[57]. *NF2* inactivation has been found to be associated with a poor clinical prognosis of MPeM, and *YAP* hyperactivation was present in approximately 70% of MPM patients^[66].

It was found that *KRAS* mutation was present in 13.7% of MPM patients and was associated with shortened median survival time^[67].

TP53 gene mutations are relatively uncommon in MPeM, only accounting for about 14%, but more than 50% of MPM lesions showed *TP53* protein overexpression, which was primarily caused by post-transcriptional phosphorylation or acetylation modifications of *TP53* protein^[68].

Approximately 13% of MPeM lesions harbored large tumor suppressor kinase 2 (*LATS2*) mutation or deletion inactivation, and the inactivation of *LATS2* could promote the development of malignant mesothelioma^[69].

In addition, in diffuse epithelioid MPeM, copy number gains or amplifications of some genes such as *WT1*, *KDR*, *TNFAIP3*, *NFKBIZ*, *NTRK1* and *MDM4* were also found (ranging from 12% to 50%), while some fusion genes including *ALK* rearrangement or *EWSR1::ATF1* or *EWSR1/FUS::CREB* were found in tumor tissues from some young MPeM patients, but *EWSR1::YY1* fusion was rare, and *ALK* rearrangement was almost absent in pleural mesothelioma, suggesting that these fusion genes may be driver variants for MPeM. It was also found in a recent study that high-frequency *ARID1B* mutations (56.5%) were present in MPeM^[61].

Although immunohistochemistry and FISH have now become routine techniques for the effective auxiliary

diagnosis of MPeM, and next generation sequencing(NGS)-based sequencing techniques have not been widely used in the diagnosis of MPM, given the high heterogeneity and complexity in the morphology, gene profile and prognosis of MPeM, for certain morphological subtypes such as sarcomatoid and biphasic MPeM, the application of NGS techniques should be more helpful for the accurate identification of key driver variants and new targets of MPeM. Molecular pathological testing shall be conducted on a compliant and strictly quality-controlled platform, and the interpretation of complex results shall be discussed by the Molecular Tumor Board (MTB).

TREATMENT

Surgical treatment

Consensus 6: Cytoreductive Surgery (CRS) combined with Hyperthermic Intraperitoneal Chemotherapy (HIPEC) is the key treatment method for MPeM (Strongly recommended).

Consensus 7: It is recommended that the feasibility of surgery for MPeM patients be assessed through discussion by a Multidisciplinary Team (MDT) (Recommended).

Consensus 8: The decision on the feasibility of CRS + HIPEC for MPeM should be made in combination with pathological findings and tumor stage. Sarcomatoid MPeM is a contraindication for CRS (Recommended).

Consensus 9: Laparoscopic exploration can provide significant guiding value for the diagnosis, staging and surgical feasibility assessment of MPeM, and the puncture site should be selected along the medioventral line (Recommended).

Consensus 10: The goal of CRS for MPeM is to achieve R0 resection, or at least satisfactory cytoreduction - residual tumor lesion < 1 cm in diameter (Recommended).

Consensus 11: For MPeM patients, it is recommended to use a platinum-containing (cisplatin or carboplatin) combination regimen for HIPEC. For MPeM patients, the recommended temperature for HIPEC is 43 °C with the duration of 60-90 min, and should maintain stable during the entire perfusion process. For Chinese female MPeM patients receiving cisplatin for HIPEC, the maximum cisplatin dose should not exceed 85 mg/m². When cisplatin is used for HIPEC, sodium thiosulfate is recommended to mitigate the risk of renal injury (Recommended).

In 2011, Yan *et al.* proposed a TNM staging system based on abdominal tumor burden (T), abdominal lymph node metastasis (N) and distant metastasis (M) to standardize and guide the clinical treatment and prognosis assessment of MPM^[40], as shown in [Table 1](#). By scoring the severity of the disease in a total of 13 areas, namely, 9 abdominal quadrants and 4 mesenteric segments (LS-0: no visible tumor; LS-1: tumor nodule ≤ 0.5 cm; LS-2: 0.5 cm < tumor nodule ≤ 5 cm; LS-3: tumor nodule > 5 cm) and adding up of scores of all quadrants, the PCI could be calculated. Based on the PCI quartiles (1-10, 11-20, 21-30, > 30), T1 to T4 stages were determined. The 5-year survival rates were 87%, 53% and 29% for stage I, II and III patients, respectively.

Surgical decision-making for MpeM

MPeM is often asymptomatic in its early stage, and highly invasive, with limited treatment options. Therefore, the prognosis of patients is poor. CRS combined with HIPEC is the key treatment method for MPeM^[70]. Radical surgery combined with HIPEC can cure some patients, and the radicality of CRS is the

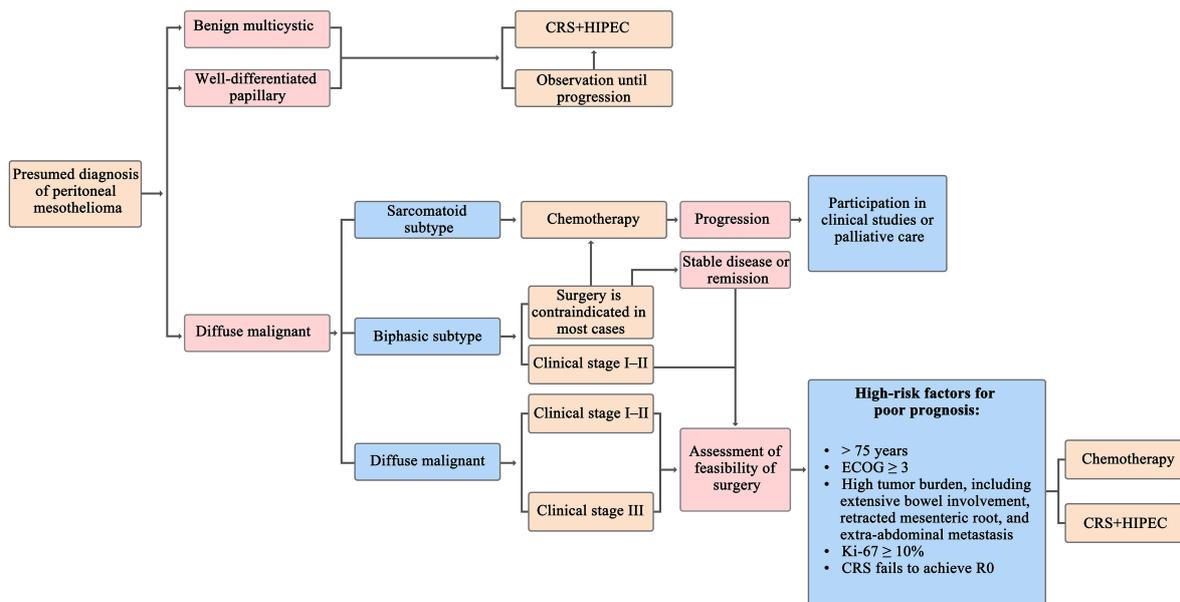


Figure 1. Decision-making for initial surgical treatment of MPeM. MPeM: Malignant peritoneal mesothelioma; CRS: cytoreductive surgery; HIPEC: hyperthermic intraperitoneal chemotherapy; ECOG: Eastern Cooperative Oncology Group.

Table 1. TNM staging system for MPeM

Stage	PCI	T	N	M
I	≤ 10	T ₁	N ₀	M ₀
II	11-30	T ₂₋₃	N ₀	M ₀
III	> 30	T ₄	N _{0/1}	M _{0/1}
		T ₁₋₄	N ₁	M _{0/1}
		T ₁₋₄	N _{0/1}	M ₁

T₁ includes PCI 1-10 points; T₂ includes PCI 11-20 points; T₃ includes PCI 21-30 points; T₄ included PCI 31-39 points; N₀: without lymph node metastasis; N₁: with lymph node metastasis; M₀: without extraperitoneal metastasis; N₁: with extraperitoneal metastasis; PCI: peritoneal cancer index; MpeM: malignant peritoneal mesothelioma; TNM: abdominal tumor burden (T), abdominal lymph node metastasis (N) and distant metastasis (M).

primary factor affecting the prognosis of peritoneal mesothelioma patients. Patient selection and surgeon experience are the key to determining the effect of CRS. For appropriately selected patients who undergo radical resection, their 5-year overall survival rate can be up to 68%^[71]. Refer to [Figure 1](#) for the patients' surgical decision-making process.

The following issues need to be considered for making clinical decisions about surgery.

Firstly, confirm the pathological diagnosis. MPeM includes three subtypes, namely, epithelioid, sarcomatoid and biphasic, among which the epithelioid subtype is the most common, which is mostly confined in the peritoneal cavity, with less frequent lymphatic and hematogenous metastases, and the patients with the subtype are suitable for surgical treatment. On the contrary, the sarcomatoid subtype is highly invasive, and prone to extraperitoneal metastasis, and most patients progress shortly after surgery even if CRS + HIPEC is completed, so it is a contraindication for surgical treatment. The proportion of patients with the biphasic subtype is similar to that of patients with the sarcomatoid subtype (20%). This subtype of MPeM contain both epithelioid MPeM and sarcomatoid MPeM. Some patients with localized tumors or those who remain stable or have achieved remission after chemotherapy can benefit from CRS +

HIPEC. It is crucial for accurate pathological classification to obtain adequate tissues. Although most peritoneal mesotheliomas are accompanied by ascitic fluid, the cytological diagnosis is very limited in accuracy rate, so it is not recommended as a diagnostic method. Laparoscopic or imaging-guided excisional biopsy can be used as a means to obtain tissue specimens surgically.

Secondly, determine the stage. The TNM staging system is the most commonly used for MPeM. It should be noted that TNM staging should be combined with the PCI^[72]. It is recommended that all patients complete CT scans to rule out thoracic metastasis, which is a contraindication for CRS + HIPEC, and lymph node metastasis, which has a very poor prognosis. PCI used for TMN staging needs to be determined at the time of exploratory laparotomy, and CT-assisted PCI can also guide surgery and prognosis. Compared with patients with PCI \geq 20, those with PCI $<$ 20 had a better prognosis (1-year survival: 59% vs. 22%). Although ascitic fluid was not involved in staging, when MPeM patients had ascitic fluid, the prognosis was poor, with a median overall survival of merely 7.0 months.

Thirdly, emphasize the application of diagnostic laparoscopy. Diagnostic laparoscopy plays an important guiding role in pathological diagnosis, staging and surgical feasibility assessment of peritoneal mesothelioma. It is recommended that laparoscopic assessment be performed by physicians proficient in surgical techniques for peritoneal tumors, which is helpful for completing the PCI scoring. During the assessment, the lesser omentum should be opened and the small intestine, especially the mesenteric root, should be assessed throughout. It should be noted that laparoscopic assessment should be performed in patients without surgical contraindications, and the puncture site should be located along the medioventral line to facilitate the excision of the tissues surrounding the puncture site during the subsequent laparotomy, thereby reducing the incidence of puncture site metastasis.

Surgical operation principles for MPeM

During CRS for MPeM, every effort should be made to resect all pelvic and abdominal tumor lesions. The surgical goal is R0 resection, or at least satisfactory cytoreduction - residual tumor lesion $<$ 1 cm in diameter. CRS is performed via a laparotomy, using a median abdominal incision extending from the xiphoid process to the pubic symphysis. All patients should undergo greater omentum resection. When mass on the intestinal surface is resected, especially for the small intestine, care must be taken to avoid damages to the serosal layer. In case of such damages, the layer should be promptly reinforced with absorbable sutures. When the involved area of the intestinal tube is relatively long, resection and anastomosis are recommended.

The recto-uterine pouch is the lowest site of the abdominal cavity in females when standing, which is highly susceptible to tumor implantation and metastasis. Posterior pelvic resection, including the uterus and rectosigmoid colon, is the most common surgical procedure. Because the lesion is confined within the abdominal cavity, transection of the rectum should be performed as close as possible to the peritoneal reflection of the recto-uterine pouch.

Principles of HIPEC treatment

For peritoneal mesothelioma where complete tumor resection is achievable, complete cytoreductive surgery combined with HIPEC is recommended. In a multicenter study, 401 patients with peritoneal mesothelioma were evaluated, of whom 46% achieved complete or near complete tumor cytoreduction and 92% received HIPEC. The median overall survival was up to 53 months, with 3- and 5-year survival rates of 60% and 47%, respectively. However, 127 (31%) patients experienced grade 3-4 complications and 9 patients died during the perioperative period^[73]. In a single-center study, 108 patients with peritoneal mesothelioma undergoing

CRS and HIPEC were enrolled, with Cisplatin and/or Doxorubicin or Mitomycin as hyperthermic perfusion agents, and the median overall survival was 63.2 months, with 19 patients surviving over 7 years^[74]. In another single-center study, 84 patients with peritoneal mesothelioma underwent CRS and HIPEC, of whom 66 patients achieved complete or near complete tumor cytoreduction, with a median overall survival of 38.4 months and a 5-year survival rate of 42%; 22 (26.2%) patients experienced grade 3-4 complications, and 3 patients died^[75]. A meta-analysis showed that 1,047 patients with peritoneal mesothelioma underwent CRS and HIPEC, of whom 67% achieved complete tumor cytoreduction, with a 1-year survival rate of 84%, 3-year survival rate of 59% and 5-year survival rate of 42%^[76]. These studies showed that, in patients with peritoneal mesothelioma who were eligible for surgery received HIPEC after surgery, nearly half of them survived for over 5 years, but the perioperative adverse event rate was high, so careful patient screening was required. For intraperitoneal chemotherapy regimens, the following may be selected: (1) Cisplatin/Doxorubicin; (2) Cisplatin; (3) Carboplatin; (4) Cisplatin/Mitomycin^[9,74,77,78].

HIPEC can be carried out either in an open or closed manner, and for patients requiring gastrointestinal anastomosis, there is no evidence suggesting that the timing of the anastomosis may affect the safety of the stoma. Therefore, the anastomosis can be performed either before or after HIPEC. In a study in 211 MPeM patients treated by CRS/HIPEC, Alexander *et al.* found that factors associated with survival included age, complete or near-complete mass resection, histologic grade of the tumor, and HIPEC (using cisplatin). Compared with Mitomycin C, Cisplatin was not only independently associated with improved survival in MPeM, but also was safer and more effective^[79].

The recommended temperature for HIPEC is 43 °C, with treatment duration of 60-90 min. Maintaining a stable temperature throughout the perfusion is crucial for ensuring both the efficacy and safety of HIPEC. The selection of the perfusion volume should follow the principles of adequate filling of abdominal cavity, patient tolerance and unobstructed circulation. The total volume of perfusion liquid should be within 3,000-5,000 mL, and the flow rate 300-600 mL/min. Realizing high-flow and high-rate perfusion under the condition that the patient can tolerate is an important way to improve the effectiveness of HIPEC.

As HIPEC is an intraperitoneal treatment and the ability for drugs to penetrate in the tumor is relatively limited, the benefits of HIPEC are predominantly observed in patients with a residual tumor ≤ 2.5 mm in diameter post-CRS. Previously, the maximum tolerated dose of cisplatin was determined to be 85 mg/m² in Chinese women undergoing cisplatin-based HIPEC in a phase I dose-finding study. Renal injury following cisplatin-based HIPEC is the most important event affecting the safety of treatment. There is evidence that sodium thiosulfate can reduce the risk of increased creatinine and acute and chronic kidney injury in patients undergoing cisplatin-based HIPEC^[80,81]. The recommended dosage regimen is sodium thiosulfate 9 g/m² + 0.9% NaCl 150 mL for the first dose, infusion within 40 min after transfer into the pump, and immediate administration after the start of HIPEC, followed by sodium thiosulfate 12 g/m² + 0.9% NaCl 1,000 mL, which is maintained for 6 h after the first dose of sodium thiosulfate into the pump. In addition, proper hydration and maintenance of effective blood volume are important measures to reduce the risk of renal injury.

For patients with malignant peritoneal carcinoma, there is a high incidence of CRS-HIPEC-related complications mainly resulting from the interaction between surgery, drugs, and HIPEC itself. From the available evidence, HIPEC does not increase the incidence of complications. Since HIPEC can enhance the cytotoxic effect of chemotherapeutic drugs, the monitoring of bone marrow and kidney toxicity reactions should be strengthened after treatment. It is suggested to combine CRS-HIPEC with enhanced recovery after surgery (ERAS) strategy, which can effectively reduce the risk of postoperative thrombosis and intestinal obstruction.

Medical treatment

Consensus 12: For patients with unresectable peritoneal mesothelioma, the chemotherapy regimen should be based on that for pleural mesothelioma, and pemetrexed plus platinum, bevacizumab plus pemetrexed and platinum, and nivolumab plus ipilimumab are recommended for first-line treatment. For patients with epithelioid subtype, chemotherapy ± bevacizumab is still the preferred regimen; for patients with non-epithelioid subtype, nivolumab plus ipilimumab, pembrolizumab plus chemotherapy, and bevacizumab plus atezolizumab and chemotherapy are optional therapeutic regimens (Recommended).

Consensus 13: Second-line treatment should be stratified according to the first-line treatment regimen for individualized diagnosis and treatment. NGS may be considered to achieve precise treatment. Or patients may participate in a clinical study. Patients who have not received immunotherapy can choose nivolumab + ipilimumab or nivolumab regimen. Patients who have received immunotherapy can choose bevacizumab combined with pemetrexed and platinum-based regimen. Pemetrexed, vinorelbine, and gemcitabine are optional regimens (Recommended).

Only a few clinical trials have explored the effectiveness of systemic therapy in patients with unresectable peritoneal mesothelioma. A study showed that among 98 patients with peritoneal mesothelioma who received pemetrexed as monotherapy or pemetrexed/cisplatin regimen, the objective response rate was 25% in first-line treatment patients and the median survival time was not reached^[82]. The expanded sample analysis results revealed that among 109 patients with peritoneal mesothelioma who received pemetrexed, pemetrexed/cisplatin, or pemetrexed/carboplatin as first- or second-line treatment, the 1-year survival rate was 57.4% for pemetrexed/cisplatin and 41.5% for pemetrexed alone, with a median survival time of 10.3 months^[83]. Other small-sample studies suggested that the median survival time of pemetrexed plus cisplatin as first-line treatment of peritoneal mesothelioma ranged from 15.4 to 16.6 months^[84,85]. There were also studies exploring gemcitabine plus cisplatin in the first-line treatment of peritoneal mesothelioma which showed some effect but led to serious adverse reactions^[86].

The pathological types and pathogenesis of pleural mesothelioma and peritoneal mesothelioma are basically similar. There are many phase III clinical studies of pleural mesothelioma with high level of evidence. Therefore, foreign guidelines recommend that systemic treatment of peritoneal mesothelioma can be based on clinical trials in patients with pleural mesothelioma. Pemetrexed + cisplatin or pemetrexed + cisplatin + bevacizumab is the preferred first-line treatment for pleural mesothelioma. A phase III randomized controlled study indicated that for patients with pleural mesothelioma who were not suitable for surgery, the median OS of patients treated with pemetrexed + cisplatin was prolonged by 2.8 months compared with cisplatin monotherapy (12.1 and 9.3 months, $P = 0.02$), which established the cornerstone of the combination of pemetrexed + cisplatin in the medical treatment of pleural mesothelioma^[87]. Several phase II clinical studies showed that pemetrexed + carboplatin was also effective in patients with pleural mesothelioma^[88-90]. A comparison of 1,704 patients with unresectable pleural mesothelioma showed that the median PFS and OS were similar for pemetrexed + cisplatin and pemetrexed + carboplatin^[91]. Therefore, pemetrexed + carboplatin is an option for patients with poor functional status who cannot tolerate cisplatin.

A randomized, multicenter, phase III study demonstrated that the combination of bevacizumab-based chemotherapy with pemetrexed + cisplatin followed by maintenance with bevacizumab prolonged median OS from 16.1 months to 18.8 months compared with pemetrexed + cisplatin (HR = 0.77, $P = 0.0167$)^[92]. This study established the status of pemetrexed + cisplatin + bevacizumab regimen in the first-line treatment of unresectable pleural mesothelioma. A phase II clinical study demonstrated that the pemetrexed + carboplatin + bevacizumab regimen achieved a median OS of 15.3 months and could also be considered as a treatment option for unresectable pleural mesothelioma^[93]. The results from several clinical trials showed

that gemcitabine + cisplatin, pemetrexed monotherapy, and vinorelbine monotherapy are all available as first-line options^[94-97].

The emergence of immunotherapy has revolutionized the landscape of oncotherapy. Several clinical studies of combination immunotherapy in pleural mesothelioma have shown a longer survival than the traditional treatment. CheckMate-743, an open-label, multicenter, randomized phase 3 clinical trial^[98], revealed that for nivolumab + ipilimumab and standard chemotherapy (pemetrexed + cisplatin or carboplatin), the median OS was 18.1 months and 14.1 months respectively (HR = 0.74, 96.6%CI: 0.60-0.91, $P = 0.0020$), with a 26% reduction in the risk of death in patients with unresectable pleural mesothelioma. The results of subgroup analyses showed a greater OS benefit in patients with non-epithelioid pleural mesothelioma (HR = 0.46) and programmed cell death-ligand 1 (PD-L1) $\geq 1\%$ (HR = 0.69). Based on the positive results from the CheckMate-743 study, the FDA approved nivolumab + ipilimumab for the first-line treatment of MPM on October 2, 2020, and the NMPA approved nivolumab plus ipilimumab for the treatment of and treatment-naive adult patients with unresectable non-epithelioid malignant pleural mesothelioma on June 8, 2021.

With the approval of dual-immunotherapy regimens for the indication of pleural mesothelioma, a number of phase III clinical studies of immunotherapy combined with chemotherapy have been conducted worldwide, including the Keynote-483 study, the BEAT-meso study and the DREAM3R study. The Keynote-483 study was an open-label, international, randomized multicenter phase III trial^[99] in which one arm received pembrolizumab plus pemetrexed and cisplatin/carboplatin and the other received only pemetrexed and cisplatin/carboplatin. The final results suggested an overall survival of 17.3 months and 16.1 months in the pembrolizumab combined with chemotherapy group versus the chemotherapy alone group, respectively (HR = 0.79, $P = 0.0324$). The 3-year overall survival was 25% in the pembrolizumab group and 17% in the chemotherapy alone group. The FDA has accelerated the approval of pembrolizumab for the indication of pleural mesothelioma. To evaluate whether the combination with immunotherapy based on bevacizumab + pemetrexed + carboplatin can further improve the efficacy in patients, BEAT-meso, an open-label, randomized, double-arm, multicenter phase III clinical trial^[100], was verbally presented at the ASCO meeting in 2024. The study was designed to evaluate the efficacy of atezolizumab + bevacizumab + pemetrexed + carboplatin (ABC) vs. bevacizumab + pemetrexed + carboplatin (BC) in the first-line treatment of patients with unresectable pleural mesothelioma. The results showed that ABC significantly prolonged PFS compared with BC, with a median PFS of 9.2 months vs. 7.6 months (HR = 0.72, $P = 0.0020$), but a median OS of 20.5 months vs. 18.1 months (HR = 0.84, $P = 0.14$), and a 2-year OS rate of 40% vs. 38%, respectively, which did not reach statistically significant difference. ABC regimen only resulted in a significant benefit in PFS, which did not translate into a significant benefit in OS, but subgroup analyses showed that patients with non-epithelioid subtype (HR = 0.50), PD-L1 TPS $\geq 1\%$ (HR = 0.66), and poor prognosis as reflected by EORTC score (HR = 0.60) had a significant OS benefit from ABC regimen. CheckMate 743, Keynote-483, and BEAT-meso all indicated that patients with non-epithelioid subtype benefited more from combination immunotherapy. Domestic and foreign guidelines recommend dual-immunotherapy combination regimen to be preferentially used in patients with non-epithelioid subtype, but further exploration is needed to determine which of the three treatment strategies (chemotherapy + immunotherapy, chemotherapy + bevacizumab + immunotherapy, and nivolumab + ipilimumab) will benefit which patients more. The phase 2 studies PrE0505^[101] and DREAM^[102], both of which used combination therapy of durvalumab + cisplatin + pemetrexed, demonstrated good efficacy of PDL1 monoclonal antibody combined with chemotherapy in pleural mesothelioma, and the phase 3 study DREAM3R is ongoing.

The studies of second-line treatment of peritoneal mesothelioma are mostly small-sample studies, and the data on second-line and subsequent treatments are relatively limited. A study evaluated 26 patients with

peritoneal mesothelioma^[85] who received gemcitabine, paclitaxel, nivolumab, and other agents, indicating a median overall survival of 16.9 months. A phase 2 study evaluated atezolizumab in combination with bevacizumab as a posterior line treatment in patients with peritoneal mesothelioma who had progressed after or were intolerant to first-line pemetrexed/platinum-based chemotherapy^[103]; 90% of the patients were epithelioid type, with a response rate of 40%, and 1-year overall survival rate of 85%. Another study evaluated the efficacy of immune checkpoint inhibitor as posterior line treatment in 29 patients with peritoneal mesothelioma^[104]; 69% of the patients received nivolumab/ipilimumab during posterior line treatment and 31% of patients received immune checkpoint inhibitor monotherapy, including nivolumab, pembrolizumab, and atezolizumab. The median survival time was 19 months and the 1-year overall survival rate was 68%.

For second-line treatment of peritoneal mesothelioma, also refer to the treatment of patients with recurrent pleural mesothelioma. For patients who did not receive pemetrexed as first-line treatment, it is recommended use it as second-line treatment. After failure of first-line treatment with pemetrexed-based therapy, pemetrexed may be re-administered to young patients with a favorable PS score and a long progression-free survival time after first-line treatment^[105,106]. Retrospective studies showed that gemcitabine and vinorelbine had some benefit^[96,107], and can be used when no other options are available. For patients who have progressed after chemotherapy, immunotherapy may be a posterior line treatment option. CONFIRM, a phase III randomized clinical trial^[108], evaluated 332 patients with MPM who had progressed after platinum-based chemotherapy, and treated with nivolumab and placebo respectively, and the results revealed that the median overall survival was 10.2 months for nivolumab-treated patients and 6.9 months for placebo-treated patients. Another randomized phase II study evaluating nivolumab with or without ipilimumab as subsequent therapy in patients with pleural mesothelioma^[109] showed that the median overall survival was 15.9 months in the nivolumab plus ipilimumab group and 11.9 months in the nivolumab alone group, but the adverse events were increased in the combination therapy group.

Although 31% of MPM patients had EGFR overexpression^[110], the efficacy of tyrosine kinase inhibitors (TKIs) for MPM was not satisfactory, and axitinib, sorafenib and imatinib have failed to benefit the patients^[111]. In a phase II clinical study^[112], the median PFS was 9.7 months in the nintedanib group compared with placebo (HR = 0.49, $P = 0.006$); the median OS was 20.6 months (HR = 0.70, $P = 0.197$), reflecting certain efficacy, and the phase III clinical study is ongoing. Recently, it has been reported in the literature that a patient with MePM had ALK translocations with STRN as fusion partner. This paper reported a 17-year-old female MPeM patient with *STRN-ALK* gene fusion and no history of asbestos exposure, who had tumor progression after standard chemotherapy and was subsequently enrolled in a clinical study using tyrosine kinase receptor inhibitor, alectinib^[113]. Another recently published trial of alectinib investigated its use in unresectable malignancies, and the author reported that alectinib may be effective in ALK-positive cancers^[114].

The results of a phase III randomized controlled trial in 448 patients with pleural mesothelioma indicated that the combination of bevacizumab with cisplatin + pemetrexed significantly prolonged OS compared with cisplatin + pemetrexed alone, and the difference was statistically significant (median OS 18.8 vs. 16.1 months, $P = 0.0167$), demonstrating the value of bevacizumab in the treatment of mesothelioma^[92]. A phase II clinical trial of ramucirumab as second-line treatment in patients with MPeM showed that ramucirumab + gemcitabine significantly prolonged OS with good safety; the median OS was 13.8 months (70%CI: 12.7-14.4 months) in the ramucirumab + gemcitabine group and 7.5 months (70%CI: 6.9-8.9 months) in the gemcitabine + placebo group, with a statistically significant difference ($P = 0.028$)^[115]. This suggests that anti-VEGF therapy is promising in patients with MPeM.

Radiotherapy

Consensus 14: The efficacy of radiotherapy in patients with MPeM is uncertain, and radiotherapy may be considered for whole-abdomen or local irradiation in patients with residual disease after cytoreductive surgery or patients who cannot undergo surgery (Recommended).

The efficacy of radiotherapy in patients with MPeM is uncertain, and radiotherapy is only used as an adjunct for other treatments to achieve more active and effective control of the condition in patients with MPeM, and radiotherapy may be considered for whole-abdomen or local irradiation in patients with residual disease after cytoreductive surgery or patients who cannot undergo surgery^[116]. Most of the current studies are small-sample case reports. A pilot study with a limited number of 10 patients showed an improvement in disease-free survival of 19 to 78 months after cytoreductive surgery, chemotherapy and whole-abdomen irradiation^[117]. It was reported that 27 patients with MPeM treated with sequential therapy of intraperitoneal drug injection, surgery, HIPEC and whole-abdomen radiotherapy had a 3-year survival rate of 67%^[118]. Adverse reactions such as low tolerated dose, low response rate and adhesion of some vital organs in the abdomen and intestinal obstruction are the main reasons for hindering the implementation of radiotherapy^[117].

Palliative supportive care

Consensus 15: Palliative and supportive treatment for malignant peritoneal mesothelioma focuses on pain management, as well as alleviation of gastrointestinal symptoms. For the management of cancer pain, refer to the principle of ladder medication for cancer pain management (Strongly Recommended).

Palliative and supportive treatment is an important part of the cancer prevention and control system, which focuses on relieving pain, other serious disease symptoms and emotional distress, so as to improve the quality of life. Drug therapy is the first choice for cancer pain management in patients with malignant peritoneal mesothelioma. For moderate pain, weak opioids, such as codeine or tramadol, are administered; for severe pain, strong opioids are administered.

The patients with malignant peritoneal mesothelioma usually have such clinical symptoms as abdominal pain, abdominal distension, intestinal obstruction, *etc.* Therefore, appropriate symptomatic treatment and nutritional support should be given during palliative and supportive treatment, and the adverse reactions due to hyperthermic intraperitoneal chemotherapy should be given corresponding drug therapy and clinical management.

Follow-up

Consensus 16: Patients with malignant peritoneal mesothelioma are recommended to be followed up every 3 months for the first 2 years after active surgical treatment, and a whole-abdomen CT reexamination should be performed (Strongly recommended). For patients with diffuse MPeM treated with CRS + HIPEC, follow-up may be extended to 15 years (Recommended).

At present, the follow-up model of malignant peritoneal mesothelioma is still lack of high-level evidence-based medical evidence. Considering the poor prognosis of malignant peritoneal mesothelioma, regular follow-up can help detect tumor progression at an early stage. In 2021, Professional Committee of Peritoneal Tumor of Chinese Anti-Cancer Association, Professional Committee of Tumor Hyperthermia of Chinese Anti-Cancer Association and Professional Committee of Tumor Hyperthermia of Beijing Cancer Prevention & Treatment Society jointly issued the Chinese Expert Consensus on the Diagnosis and Treatment of Diffuse Malignant Peritoneal Mesothelioma^[8]. In combination with clinical practice, it is recommended that follow-up items include physical examination, CT scan of the whole abdomen and

serum tumor markers. The frequency of follow-up is every 3 months for the first 2 years after surgery, every 6 months for 3 to 5 years after surgery, and every year after 5 years, and an extension to 15 years after surgery is possible. At present, PET-CT and MRI are not recommended as routine follow-up examinations; there is currently insufficient evidence to suggest that biomarkers can be used for follow-up of malignant peritoneal mesothelioma; the monitoring of disease progression should be guided by signs and symptoms that occur during clinical follow-up period.

DECLARATIONS

Authors' contributions

The methodological framework and intellectual architecture of this expert consensus were collaboratively developed: Shen P, Chen T, Wang Q, Lu Y

Responsible for conceptual refinement, cross-disciplinary coordination, and iterative drafting: Li J, Hu J, Xu C, Ji Q, Wang W, Guo Z

Final manuscript validation was achieved through critical appraisal and unanimous ratification by all contributing scholars.

Availability of data and materials

Not applicable.

Financial support and sponsorship

This research received funding from several sources, including the National Natural Science Foundation of China (82072577, 82503982). Additional support was provided by the China Postdoctoral Science Foundation (2022M723207) and the Medical Scientific Research Foundation of Zhejiang Province (2023KY666). Furthermore, funding was obtained from the Zhejiang Traditional Chinese Medicine Science Fund Project (2024ZL372), the Qiantang Cross Fund Project (2023-16), the National Natural Science Foundation of China for the Zhejiang Cancer Hospital Cultivation Project (PY2023006), and the Medical Scientific Research Foundation of Zhejiang Province (2024KY812). Lastly, support was also received from The Natural Science Foundation of Zhejiang Province (Q24H160110), International Medical Association and the Foundation of President of Nanfang Hospital, Southern Medical University (No. 2023A017)

Conflicts of interest

Chunwei Xu, Zhengbo Song, and Xingxiang Pu are Editorial Board members of *IOMJ* and co-authors of this article. They were excluded from editorial decision-making related to the acceptance of this article for publication in the journal.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

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REFERENCES

1. Magge D, Zenati MS, Austin F, et al. Malignant peritoneal mesothelioma: prognostic factors and oncologic outcome analysis. *Ann Surg Oncol.* 2014;21:1159-65. DOI PubMed PMC
2. Shavelle R, Vavra-Musser K, Lee J, Brooks J. Life expectancy in pleural and peritoneal mesothelioma. *Lung Cancer Int.* 2017;2017:2782590. DOI PubMed PMC
3. Mazurek JM, Syamlal G, Wood JM, Hendricks SA, Weston A. Malignant mesothelioma mortality - United States, 1999-2015. *MMWR Morb Mortal Wkly Rep.* 2017;66:214-8. DOI PubMed PMC
4. Beebe-Dimmer JL, Fryzek JP, Yee CL, et al. Mesothelioma in the United States: a surveillance, epidemiology, and end results (SEER)-medicare investigation of treatment patterns and overall survival. *Clin Epidemiol.* 2016;8:743-50. DOI
5. Ji Q, Xu C, Wang Q, et al. Expert consensus on the diagnosis and treatment of malignant pericardial mesothelioma. *Med Res.* 2025;1:122-30. DOI
6. Wang Q, Xu C, Wang W, et al. Chinese expert consensus on the diagnosis and treatment of malignant pleural mesothelioma. *Thorac Cancer.* 2023;14:2715-31. DOI
7. Cai Y, Xu C, Lin J, et al. Expert consensus on the diagnosis and treatment of malignant mesothelioma of the tunica vaginalis testis. *iMetaMed.* 2025;1:e70003. DOI
8. Chinese Anti Cancer Association Peritoneal Tumor Committee, Chinese Anti Cancer Association Tumor Thermal Therapy Committee, Beijing Cancer Prevention and Treatment Society Tumor Thermal Therapy Committee. Chinese expert consensus on the diagnosis and treatment of diffuse malignant peritoneal mesothelioma. *Natl Med J China.* 2021;101:2839-49. DOI
9. Chicago Consensus Working Group. The Chicago consensus on peritoneal surface malignancies: management of peritoneal mesothelioma. *Cancer.* 2020;126:2547-52. DOI
10. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®). Mesothelioma: Peritoneal Version 2.2025. Available from: <http://www.nccn.org/guidelines/guidelines-detail?category=1&id=1513>. [Last accessed on 4 Jan 2026].
11. Kusamura S, Kepenekian V, Villeneuve L, et al. ; PSOGI. Peritoneal mesothelioma: PSOGI/EURACAN clinical practice guidelines for diagnosis, treatment and follow-up. *Eur J Surg Oncol.* 2021;47:36-59. DOI
12. Xu C, Hao Y, Wang D, et al. ECLUNG consensus/guidelines development principles and methods (2024 edition). *Intelligent Pharmacy.* 2025;3:141-2. DOI
13. Bray F, Laversanne M, Sung H, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin.* 2024;74:229-63. DOI
14. Zhao J, Zuo T, Zheng R, et al. Epidemiology and trend analysis on malignant mesothelioma in China. *Chin J Cancer Res.* 2017;29:361-8. DOI PubMed PMC
15. Karpes JB, Shamavonian R, Dewhurst S, et al. Malignant peritoneal mesothelioma: an in-depth and up-to-date review of pathogenesis, diagnosis, management and future directions. *Cancers.* 2023;15:4704. DOI PubMed PMC
16. Singh R, Frank AL. Does the presence of asbestos-containing materials in buildings post-construction and before demolition have an impact on the exposure to occupants in non-occupational settings? *Cureus* 2023;15:e37305. DOI PubMed PMC
17. Frost G. The latency period of mesothelioma among a cohort of British asbestos workers (1978-2005). *Br J Cancer.* 2013;109:1965-73. DOI PubMed PMC
18. Abós-Herrándiz R, Rodríguez-Blanco T, García-Allas I, et al. Risk factors of mortality from all asbestos-related diseases: a competing risk analysis. *Can Respir J.* 2017;2017:9015914. DOI PubMed PMC
19. Kerrigan SA, Turnnir RT, Clement PB, Young RH, Churg A. Diffuse malignant epithelial mesotheliomas of the peritoneum in women: a clinicopathologic study of 25 patients. *Cancer.* 2002;94:378-85. DOI PubMed
20. Browne K. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann Occup Hyg.* 2001;45:327-9. DOI PubMed
21. Berry G, Reid A, Aboagye-Sarfo P, et al. Malignant mesotheliomas in former miners and millers of crocidolite at Wittenoom (Western Australia) after more than 50 years follow-up. *Br J Cancer.* 2012;106:1016-20. DOI PubMed PMC
22. Amin AM, Mason C, Rowe P. Diffuse malignant mesothelioma of the peritoneum following abdominal radiotherapy. *Eur J Surg Oncol.* 2001;27:214-5. DOI PubMed
23. Antman KH, Corson JM, Li FP, et al. Malignant mesothelioma following radiation exposure. *J Clin Oncol.* 1983;1:695-700. DOI
24. Farioli A, Ottone M, Morganti AG, et al. Radiation-induced mesothelioma among long-term solid cancer survivors: a longitudinal analysis of SEER database. *Cancer Med.* 2016;5:950-9. DOI
25. Mujahed T, Tazelaar HD, Sukov WR, et al. Malignant peritoneal mesothelioma arising in young adults with long-standing indwelling intra-abdominal shunt catheters. *Am J Surg Pathol.* 2021;45:255-62. DOI

26. Alakus H, Yost SE, Woo B, et al. BAP1 mutation is a frequent somatic event in peritoneal malignant mesothelioma. *J Transl Med.* 2015;13:122. [DOI PubMed PMC](#)
27. Napolitano A, Pellegrini L, Dey A, et al. Minimal asbestos exposure in germline BAP1 heterozygous mice is associated with deregulated inflammatory response and increased risk of mesothelioma. *Oncogene.* 2016;35:1996-2002. [DOI PubMed PMC](#)
28. Pastorino S, Yoshikawa Y, Pass HI, et al. A subset of mesotheliomas with improved survival occurring in carriers of BAP1 and other germline mutations. *J Clin Oncol.* 2018;36:JCO2018790352. [DOI](#)
29. Mao W, Zhang X, Guo Z, et al. Association of asbestos exposure with malignant mesothelioma incidence in Eastern China. *JAMA Oncol.* 2017;3:562-4. [DOI PubMed PMC](#)
30. Guo Z, Carbone M, Zhang X, et al. Improving the accuracy of mesothelioma diagnosis in China. *J Thorac Oncol.* 2017;12:714-23. [DOI](#)
31. Yang S, Hao Y, Wang K, et al. Epidemiology and clinical characteristics of mesothelioma in the Chinese population: a multicenter study. *Ther Adv Med Oncol.* 2025;17:17588359251378879. [DOI PubMed PMC](#)
32. Broeckx G, Pauwels P. Malignant peritoneal mesothelioma: a review. *Transl Lung Cancer Res.* 2018;7:537-42. [DOI PubMed PMC](#)
33. Kaya H, Sezgi C, Tanrikulu AC, et al. Prognostic factors influencing survival in 35 patients with malignant peritoneal mesothelioma. *Neoplasma.* 2014;61:433-8. [DOI](#)
34. Antman KH. Natural history and epidemiology of malignant mesothelioma. *Chest.* 1993;103:373S-6S. [DOI PubMed](#)
35. Acherman YI, Welch LS, Bromley CM, Sugarbaker PH. Clinical presentation of peritoneal mesothelioma. *Tumori.* 2003;89:269-73. [DOI PubMed](#)
36. Sugarbaker PH, Welch LS, Mohamed F, Glehen O. A review of peritoneal mesothelioma at the Washington Cancer Institute. *Surg Oncol Clin N Am.* 2003;12: 605-21, xi. [DOI PubMed](#)
37. Levy AD, Arnáiz J, Shaw JC, Sobin LH. From the archives of the AFIP: primary peritoneal tumors: imaging features with pathologic correlation. *Radiographics.* 2008;28:583-607. [DOI PubMed](#)
38. Baratti D, Kusamura S, Cabras AD, Laterza B, Balestra MR, Deraco M. Lymph node metastases in diffuse malignant peritoneal mesothelioma. *Ann Surg Oncol.* 2010;17:45-53. [DOI PubMed](#)
39. Pappa L, Machera M, Tsanou E, et al. Subcutaneous metastasis of peritoneal mesothelioma diagnosed by fine-needle aspiration. *Pathol Oncol Res.* 2006;12:247-50. [DOI](#)
40. Yan TD, Deraco M, Elias D, et al. ; Peritoneal Surface Oncology Group. A novel tumor-node-metastasis (TNM) staging system of diffuse malignant peritoneal mesothelioma using outcome analysis of a multi-institutional database*. *Cancer.* 2011;117:1855-63. [DOI](#)
41. Manzini VP, Recchia L, Cafferata M, et al. Malignant peritoneal mesothelioma: a multicenter study on 81 cases. *Ann Oncol.* 2010;21:348-53. [DOI](#)
42. Bonde A, Singh R, Prasad SR, et al. Mesotheliomas and benign mesothelial tumors: update on pathologic and imaging findings. *Radiographics.* 2023;43:e220128. [DOI](#)
43. Chandramohan A, Shah N, Thrower A, et al. Communicating imaging findings in peritoneal mesothelioma: the impact of 'PAUSE' on surgical decision-making. *Insights Imaging.* 2021;12:174. [DOI PubMed PMC](#)
44. Carlson B, Harmath C, Turaga K, Kindler HL, Armato SG 3rd, Straus C. The role of imaging in diagnosis and management of malignant peritoneal mesothelioma: a systematic review. *Abdom Radiol.* 2022;47:1725-40. [DOI PubMed](#)
45. Boussios S, Moschetta M, Karathanasi A, et al. Malignant peritoneal mesothelioma: clinical aspects, and therapeutic perspectives. *Ann Gastroenterol.* 2018;31:659-69. [DOI PubMed](#)
46. Lu J, Guo Q, Zhang Y, et al. A modified diffusion-weighted magnetic resonance imaging-based model from the radiologist's perspective: improved performance in determining the surgical resectability of advanced high-grade serous ovarian cancer. *Am J Obstet Gynecol.* 2024;231:117.e1-117.e17. [DOI](#)
47. Dubreuil J, Giammarile F, Rousset P, et al. The role of 18F-FDG-PET/ceCT in peritoneal mesothelioma. *Nucl Med Commun.* 2017;38:312-8. [DOI](#)
48. Domènech-Vilardell A, Rasiej MJ, Taub RN, Ichise M. Clinical utility of 18F-FDG positron emission tomography in malignant peritoneal mesothelioma. *Q J Nucl Med Mol Imaging.* 2014;60:54-61. [PubMed](#)
49. Kuribayashi K, Kitajima K, Minami T, Ikeda M, Yamakado K, Kijima T. Malignant peritoneal mesothelioma features shown by FDG-PET/CT. *Cancer Diagn Progn.* 2022;2:654-60. [DOI PubMed PMC](#)
50. Sugarbaker PH, Sardi A, Brown G, Dromain C, Rousset P, Jelinek JS. Concerning CT features used to select patients for treatment of peritoneal metastases, a pictorial essay. *Int J Hyperthermia.* 2017;33:497-504. [DOI PubMed](#)
51. Rivard JD, Temple WJ, McConnell YJ, Sultan H, Mack LA. Preoperative computed tomography does not predict resectability in peritoneal carcinomatosis. *Am J Surg.* 2014;207:760-5. [DOI](#)

52. Sauter JL, Dacic S, Galateau-Salle F, et al. The 2021 WHO classification of tumors of the pleura: advances since the 2015 classification. *J Thorac Oncol.* 2022;17:608-22. DOI
53. Paintal A, Raparia K, Zakowski MF, Nayar R. The diagnosis of malignant mesothelioma in effusion cytology: a reappraisal and results of a multi-institution survey. *Cancer Cytopathol.* 2013;121:703-7. DOI PubMed
54. Henderson DW, Reid G, Kao SC, van Zandwijk N, Klebe S. Challenges and controversies in the diagnosis of mesothelioma: Part 1. Cytology-only diagnosis, biopsies, immunohistochemistry, discrimination between mesothelioma and reactive mesothelial hyperplasia, and biomarkers. *J Clin Pathol.* 2013;66:847-53. DOI PubMed
55. Yoshimura M, Kinoshita Y, Hamasaki M, et al. Highly expressed EZH2 in combination with BAP1 and MTAP loss, as detected by immunohistochemistry, is useful for differentiating malignant pleural mesothelioma from reactive mesothelial hyperplasia. *Lung Cancer.* 2019;130:187-93. DOI
56. Hwang HC, Pyott S, Rodriguez S, et al. BAP1 Immunohistochemistry and p16 FISH in the diagnosis of sarcomatous and desmoplastic mesotheliomas. *Am J Surg Pathol.* 2016;40:714-8. DOI
57. Berg KB, Dacic S, Miller C, Cheung S, Churg A. Utility of methylthioadenosine phosphorylase compared with BAP1 immunohistochemistry, and CDKN2A and NF2 fluorescence in situ hybridization in separating reactive mesothelial proliferations from epithelioid malignant mesotheliomas. *Arch Pathol Lab Med.* 2018;142:1549-53. DOI PubMed
58. Ang PP, Tan GC, Karim N, Wong YP. Diagnostic value of the EZH2 immunomarker in malignant effusion cytology. *Acta Cytol.* 2020;64:248-55. DOI PubMed
59. Husain AN, Colby TV, Ordóñez NG, et al. Guidelines for pathologic diagnosis of malignant mesothelioma 2017 update of the consensus statement from the international mesothelioma interest group. *Arch Pathol Lab Med.* 2018;142:89-108. DOI
60. Panou V, Gadiraju M, Wolin A, et al. Frequency of germline mutations in cancer susceptibility genes in malignant mesothelioma. *J Clin Oncol.* 2018;36:2863-71. DOI
61. Hung YP, Dong F, Torre M, Crum CP, Bueno R, Chiriac LR. Molecular characterization of diffuse malignant peritoneal mesothelioma. *Mod Pathol.* 2020;33:2269-79. DOI PubMed
62. Betti M, Aspesi A, Ferrante D, et al. Sensitivity to asbestos is increased in patients with mesothelioma and pathogenic germline variants in BAP1 or other DNA repair genes. *Genes Chromosomes Cancer.* 2018;57:573-83. DOI
63. Wu D, Hiroshima K, Matsumoto S, et al. Diagnostic usefulness of p16/CDKN2A FISH in distinguishing between sarcomatoid mesothelioma and fibrous pleuritis. *Am J Clin Pathol.* 2013;139:39-46. DOI
64. Marshall K, Jackson S, Jones J, et al. Homozygous deletion of CDKN2A in malignant mesothelioma: diagnostic utility, patient characteristics and survival in a UK mesothelioma centre. *Lung Cancer.* 2020;150:195-200. DOI
65. Brcic L, Le Stang N, Gallob F, et al. A combination of MTAP and p16 immunohistochemistry can substitute for CDKN2A fluorescence in situ hybridization in diagnosis and prognosis of pleural mesotheliomas. *Arch Pathol Lab Med.* 2023;147:313-22. DOI
66. Marchevsky AM, LeStang N, Hiroshima K, et al. The differential diagnosis between pleural sarcomatoid mesothelioma and spindle cell/pleomorphic (sarcomatoid) carcinomas of the lung: evidence-based guidelines from the International Mesothelioma Panel and the MESOPATH National Reference Center. *Hum Pathol.* 2017;67:160-8. DOI
67. Vannucchi M, Pennati V, Mencaroni C, et al. KRAS mutations are associated with shortened survival in patients with epithelioid malignant pleural mesothelioma. *Cancers* 2023;15:2072. DOI PubMed PMC
68. Dietz MV, van Kooten JP, Paats MS, et al. Molecular alterations and potential actionable mutations in peritoneal mesothelioma: a scoping review of high-throughput sequencing studies. *ESMO Open.* 2023;8:101600. DOI PubMed PMC
69. Murakami H, Mizuno T, Taniguchi T, et al. LATS2 is a tumor suppressor gene of malignant mesothelioma. *Cancer Res.* 2011;71:873-83. DOI
70. Hübner M, van Der Speeten K, Govaerts K, et al. 2022 peritoneal surface oncology group international consensus on HIPEC regimens for peritoneal malignancies: colorectal cancer. *Ann Surg Oncol.* 2024;31:567-76. DOI PubMed PMC
71. Russell MC, Staley CA. Cytoreductive surgery and hyperthermic intraperitoneal chemotherapy for malignant peritoneal mesothelioma. *J Oncol Pract.* 2016;12:938-9. DOI PubMed
72. Jacquet P, Sugarbaker PH. Clinical research methodologies in diagnosis and staging of patients with peritoneal carcinomatosis. In: Sugarbaker PH, editor. *Peritoneal Carcinomatosis: Principles of Management.* Boston: Springer US; 1996. pp. 359-74. DOI
73. Yan TD, Deraco M, Baratti D, et al. Cytoreductive surgery and hyperthermic intraperitoneal chemotherapy for malignant peritoneal mesothelioma: multi-institutional experience. *J Clin Oncol.* 2009;27:6237-42. DOI
74. Baratti D, Kusamura S, Cabras AD, Bertulli R, Hutanu I, Deraco M. Diffuse malignant peritoneal mesothelioma: long-term survival with complete cytoreductive surgery followed by hyperthermic intraperitoneal chemotherapy (HIPEC). *Eur J Cancer.* 2013;49:3140-8. DOI PubMed

75. Acs M, Gerken M, Gajic I, Mayr M, Zustin J, Piso P. Ten-year single-center experience with treatment of primary diffuse malignant peritoneal mesothelioma (DMPM) by cytoreductive surgery (CRS) and hyperthermic intraperitoneal chemotherapy (HIPEC). *Langenbecks Arch Surg.* 2022;407:3057-67. DOI PubMed
76. Helm JH, Miura JT, Glenn JA, et al. Cytoreductive surgery and hyperthermic intraperitoneal chemotherapy for malignant peritoneal mesothelioma: a systematic review and meta-analysis. *Ann Surg Oncol.* 2015;22:1686-93. DOI
77. Shetty SJ, Bathla L, Govindarajan V, Thomas P, Loggie BW. Comparison of cytoreductive surgery and hyperthermic intraperitoneal chemotherapy with mitomycin or carboplatin for diffuse malignant peritoneal mesothelioma. *Am Surg.* 2014;80:348-52. DOI PubMed
78. Kepenekian V, Elias D, Passot G, et al. ; French Network for Rare Peritoneal Malignancies (RENAPE). Diffuse malignant peritoneal mesothelioma: Evaluation of systemic chemotherapy with comprehensive treatment through the RENAPE database: multi-institutional retrospective study. *Eur J Cancer.* 2016;65:69-79. DOI
79. Alexander HR Jr, Bartlett DL, Pingpank JF, et al. Treatment factors associated with long-term survival after cytoreductive surgery and regional chemotherapy for patients with malignant peritoneal mesothelioma. *Surgery.* 2013;153:779-86. DOI PubMed PMC
80. Laplace N, Kepenekian V, Friggeri A, et al. Sodium thiosulfate protects from renal impairment following hyperthermic intraperitoneal chemotherapy (HIPEC) with cisplatin. *Int J Hyperthermia.* 2020;37:897-902. DOI
81. Grillo-Marín C, Antón-Rodríguez C, Prieto L, Ortega-Pérez G, González-Moreno S. Nephrotoxicity associated with cytoreductive surgery combined with cisplatin-based hyperthermic intraperitoneal chemotherapy for peritoneal malignant disease: a systematic review and meta-analysis. *J Clin Med.* 2024;13:3793. DOI PubMed PMC
82. Jänne PA, Wozniak AJ, Belani CP, et al. Open-label study of pemetrexed alone or in combination with cisplatin for the treatment of patients with peritoneal mesothelioma: outcomes of an expanded access program. *Clin Lung Cancer.* 2005;7:40-6. DOI PubMed
83. Carteni G, Manegold C, Garcia GM, et al. Malignant peritoneal mesothelioma-results from the international expanded access program using pemetrexed alone or in combination with a platinum agent. *Lung Cancer.* 2009;64:211-8. DOI
84. Fujimoto E, Kijima T, Kuribayashi K, et al. First-line chemotherapy with pemetrexed plus cisplatin for malignant peritoneal mesothelioma. *Expert Rev Anticancer Ther.* 2017;17:865-72. DOI
85. Nagata Y, Sawada R, Takashima A, et al. Efficacy and safety of pemetrexed plus cisplatin as first-line chemotherapy in advanced malignant peritoneal mesothelioma. *Jpn J Clin Oncol.* 2019;49:1004-8. DOI
86. Simon GR, Verschraegen CF, Jänne PA, et al. Pemetrexed plus gemcitabine as first-line chemotherapy for patients with peritoneal mesothelioma: final report of a phase II trial. *J Clin Oncol.* 2008;26:3567-72. DOI
87. Vogelzang NJ, Rusthoven JJ, Symanowski J, et al. Phase III study of pemetrexed in combination with cisplatin versus cisplatin alone in patients with malignant pleural mesothelioma. *J Clin Oncol.* 2003;21:2636-44. DOI
88. Katirtzoglou N, Gkiozos I, Makrilia N, et al. Carboplatin plus pemetrexed as first-line treatment of patients with malignant pleural mesothelioma: a phase II study. *Clin Lung Cancer.* 2010;11:30-5. DOI
89. Ceresoli GL, Zucali PA, Favaretto AG, et al. Phase II study of pemetrexed plus carboplatin in malignant pleural mesothelioma. *J Clin Oncol.* 2006;24:1443-8. DOI
90. Castagneto B, Botta M, Aitini E, et al. Phase II study of pemetrexed in combination with carboplatin in patients with malignant pleural mesothelioma (MPM). *Ann Oncol.* 2008;19:370-3. DOI
91. Santoro A, O'Brien ME, Stahel RA, et al. Pemetrexed plus cisplatin or pemetrexed plus carboplatin for chemo-naïve patients with malignant pleural mesothelioma: results of the international expanded access program. *J Thorac Oncol.* 2008;3:756-63. DOI
92. Zalcman G, Mazieres J, Margery J, et al. ; French Cooperative Thoracic Intergroup (IFCT). Bevacizumab for newly diagnosed pleural mesothelioma in the mesothelioma avastin cisplatin pemetrexed study (MAPS): a randomised, controlled, open-label, phase 3 trial. *Lancet.* 2016;387:1405-14. DOI
93. Ceresoli GL, Zucali PA, Mencoboni M, et al. Phase II study of pemetrexed and carboplatin plus bevacizumab as first-line therapy in malignant pleural mesothelioma. *Br J Cancer.* 2013;109:552-8. DOI PubMed PMC
94. van Haarst JM, Baas P, Manegold Ch, et al. Multicentre phase II study of gemcitabine and cisplatin in malignant pleural mesothelioma. *Br J Cancer.* 2002;86:342-5. DOI PubMed PMC
95. Nowak AK, Byrne MJ, Williamson R, et al. A multicentre phase II study of cisplatin and gemcitabine for malignant mesothelioma. *Br J Cancer.* 2002;87:491-6. DOI PubMed PMC
96. Taylor P, Castagneto B, Dark G, et al. Single-agent pemetrexed for chemo-naïve and pretreated patients with malignant pleural mesothelioma: results of an international expanded access program. *J Thorac Oncol.* 2008;3:764-71. DOI
97. Muers MF, Stephens RJ, Fisher P, et al. ; MS01 Trial Management Group. Active symptom control with or without chemotherapy in the treatment of patients with malignant pleural mesothelioma (MS01): a multicentre randomised trial. *Lancet.* 2008;371:1685-94. DOI

98. Baas P, Scherpereel A, Nowak AK, et al. First-line nivolumab plus ipilimumab in unresectable malignant pleural mesothelioma (CheckMate 743): a multicentre, randomised, open-label, phase 3 trial. *Lancet.* 2021;397:375-86. DOI
99. Chu Q, Perrone F, Greillier L, et al. Pembrolizumab plus chemotherapy versus chemotherapy in untreated advanced pleural mesothelioma in Canada, Italy, and France: a phase 3, open-label, randomised controlled trial. *Lancet.* 2023;402:2295-306. DOI
100. Felip E, Popat S, Dafni U, et al. ; ETOP 13-18 BEAT-meso Collaborators. A randomised phase III study of bevacizumab and carboplatin-pemetrexed chemotherapy with or without atezolizumab as first-line treatment for advanced pleural mesothelioma: results of the ETOP 13-18 BEAT-meso trial. *Ann Oncol.* 2025;36:548-60. DOI
101. Forde PM, Anagnostou V, Sun Z, et al. Durvalumab with platinum-pemetrexed for unresectable pleural mesothelioma: survival, genomic and immunologic analyses from the phase 2 PrE0505 trial. *Nat Med.* 2021;27:1910-20. DOI PubMed PMC
102. Nowak AK, Lesterhuis WJ, Kok PS, et al. Durvalumab with first-line chemotherapy in previously untreated malignant pleural mesothelioma (DREAM): a multicentre, single-arm, phase 2 trial with a safety run-in. *Lancet Oncol.* 2020;21:1213-23. DOI
103. Raghav K, Liu S, Overman MJ, et al. Efficacy, safety, and biomarker analysis of combined PD-L1 (Atezolizumab) and VEGF (Bevacizumab) blockade in advanced malignant peritoneal mesothelioma. *Cancer Discov.* 2021;11:2738-47. DOI PubMed PMC
104. Raghav K, Liu S, Overman M, et al. Clinical efficacy of immune checkpoint inhibitors in patients with advanced malignant peritoneal mesothelioma. *JAMA Netw Open.* 2021;4:e2119934. DOI PubMed PMC
105. Abdel-Rahman O, Kelany M. Systemic therapy options for malignant pleural mesothelioma beyond first-line therapy: a systematic review. *Expert Rev Respir Med.* 2015;9:533-49. DOI PubMed
106. Zucali PA, Simonelli M, Michetti G, et al. Second-line chemotherapy in malignant pleural mesothelioma: results of a retrospective multicenter survey. *Lung Cancer.* 2012;75:360-7. DOI
107. Zauderer MG, Kass SL, Woo K, Sima CS, Ginsberg MS, Krug LM. Vinorelbine and gemcitabine as second- or third-line therapy for malignant pleural mesothelioma. *Lung Cancer.* 2014;84:271-4. DOI
108. Fennell DA, Ewings S, Ottensmeier C, et al. ; CONFIRM trial investigators. Nivolumab versus placebo in patients with relapsed malignant mesothelioma (CONFIRM): a multicentre, double-blind, randomised, phase 3 trial. *Lancet Oncol.* 2021;22:1530-40. DOI
109. Scherpereel A, Mazieres J, Greillier L, et al. ; French Cooperative Thoracic Intergroup. Nivolumab or nivolumab plus ipilimumab in patients with relapsed malignant pleural mesothelioma (IFCT-1501 MAPS2): a multicentre, open-label, randomised, non-comparative, phase 2 trial. *Lancet Oncol.* 2019;20:239-53. DOI
110. Foster JM, Radhakrishna U, Govindarajan V, et al. Clinical implications of novel activating EGFR mutations in malignant peritoneal mesothelioma. *World J Surg Oncol.* 2010;8:88. DOI PubMed PMC
111. Scherpereel A, Wallyn F, Albelda SM, Munck C. Novel therapies for malignant pleural mesothelioma. *Lancet Oncol.* 2018;19:e161-72. DOI PubMed
112. Grosso F, Steele N, Novello S, et al. Nintedanib plus pemetrexed/cisplatin in patients with malignant pleural mesothelioma: phase ii results from the randomized, placebo-controlled LUME-meso trial. *J Clin Oncol.* 2017;35:3591-600. DOI
113. Miyagawa C, Takaya H, Sakai K, et al. A novel malignant peritoneal mesothelioma with STRN exon 2 and ALK exon 20: a case report and literature review. *Oncologist.* 2021;26:356-61. DOI
114. Narita S, Goto Y, Okuma H, et al. TACKLE: a phase ii study of alectinib in patients with unresectable rare cancer harboring ALK alterations (NCCH1712). *Annals of Oncology.* 2019;30:vi137. DOI
115. Pinto C, Zucali PA, Pagano M, et al. Gemcitabine with or without ramucirumab as second-line treatment for malignant pleural mesothelioma (RAMES): a randomised, double-blind, placebo-controlled, phase 2 trial. *Lancet Oncol.* 2021;22:1438-47. DOI
116. Cao S, Jin S, Cao J, et al. Advances in malignant peritoneal mesothelioma. *Int J Colorectal Dis.* 2015;30:1-10. DOI
117. Lederman GS, Recht A, Herman T, Osteen R, Corson J, Antman KH. Long-term survival in peritoneal mesothelioma the role of radiotherapy and combined modality treatment. *Cancer.* 1987;59:1882-6. DOI PubMed
118. Hesdorffer ME, Chabot JA, Keohan ML, et al. Combined resection, intraperitoneal chemotherapy, and whole abdominal radiation for the treatment of malignant peritoneal mesothelioma. *Am J Clin Oncol.* 2008;31:49-54. DOI