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The Metachronous Co-Occurrence of Primary Prostate Adenocarcinoma and Malignant Peritoneal Mesothelioma: A Case Report

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Case Report

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Abstract

Background

The metachronous co-occurrence of prostate adenocarcinoma and malignant peritoneal mesothelioma is extremely rare, with few cases reported in the English literature. While malignant mesothelioma is mostly associated with the pleura and has a strong association with asbestos exposure, malignant peritoneal mesothelioma has less of this association. Some studies suggest a potential genetic linkage between prostate adenocarcinoma and malignant mesothelioma, primarily focusing on the better studied pleural variant. The most frequently studied mechanisms include BAP1, CDKN2A, NF2, SETD2, and TP53. While prostate cancer may be linked to germline mutations, the co-occurrence of prostate adenocarcinoma with malignant peritoneal mesothelioma has been rarely reported and cannot currently be explained by an overlap in molecular alterations.

Case Presentation

A 63-year-old Asian male, diagnosed with high-grade Stage IV prostate cancer with metastasis into the peritoneum, underwent radical prostatectomy followed by hormone therapy and chemotherapy, with favorable results and a prostate-specific antigen < 0.1. The patient continued to report recurrent ascites and subsequent computed tomography scans showed worsening of extensive bulky peritoneal and omental metastases, raising suspicion of an undifferentiated neuroendocrine cancer, necessitating biopsy. Pathology revealed a diagnosis of malignant epithelioid peritoneal mesothelioma, one year after diagnosis of prostate adenocarcinoma. Genetic testing revealed variants in BARD1 and CDKN2A. The patient began chemotherapy, later augmented with immunotherapy, due to the progression of peritoneal lesions. Evaluation for surgical debulking followed and surgery was performed 10 months after diagnosis. A completeness of cancer resection score of 2 was given post-peritonectomy and omentectomy, with residual disease on the bilateral iliac vessels, anterior bladder of the prostate, and right retroperitoneum.

Conclusions

A comprehensive review of the available English-language literature identified the rarity of published cases describing the concurrent presentation of prostate adenocarcinoma and malignant peritoneal mesothelioma within the same patient. This case may therefore represent one of the few reported instances of these two primary malignancies occurring metachronously. There lies an underlying challenge in simultaneously identifying these cancers, highlighting the significance of this case to provide awareness and the need for further research into any mechanisms that may describe a correlation between these two malignancies.

Background

Malignant mesothelioma is a rare cancer arising from mesothelial cells lining the pleura, peritoneum, pericardium, or tunica vaginalis of the testes, with the pleural subtype being most common and strongly associated with occupational asbestos exposure (1, 2). Malignant peritoneal mesothelioma (MPM) accounts for 7–30% of all mesotheliomas depending on the dataset and referral patterns (3). Compared to pleural mesothelioma, which predominantly affects older adults with a mean age at diagnosis of approximately 70–71 years, MPM presents in a slightly younger population with a median age of approximately 69 years, frequently manifesting as advanced disease at the time of diagnosis (1, 2, 4). Histologically, the epithelioid subtype is the most common form of MPM and carries a significantly better prognosis compared to the sarcomatoid or biphasic variants (5). Surgical series of patients undergoing cytoreductive surgery with hyperthermic intraperitoneal chemotherapy (CRS-HIPEC) tend to include younger, highly selected patients who are eligible for aggressive multimodal therapy.

At the molecular level, MPM is defined by recurrent genomic alterations in key tumor suppressor genes. BAP1 (BRCA1-associated protein 1) is among the most frequently altered genes, with somatic mutations and copy-number losses observed across multiple cohorts. Loss of BAP1 function disrupts chromatin remodeling and DNA damage response pathways, with emerging evidence of sensitivity to immune checkpoint inhibitors and DNA repair–targeted therapies (6). CDKN2A homozygous deletion, while more common in pleural mesothelioma, is seen in a subset of MPM cases and is associated with more aggressive disease and worse outcomes, particularly when co-occurring with NF2 (neurofibromin 2) alterations (7, 8). Alterations in epigenetic regulators such as SETD2 further highlight the importance of chromatin-modifying pathways in MPM biology (9). Notably, TP53 mutations, which are drivers in many other solid tumors, are relatively uncommon in MPM and are not considered a dominant alteration in this disease.

From a therapeutic standpoint, CRS-HIPEC has emerged as the standard of care for selected patients with resectable MPM, with large multicenter series and pooled analyses reporting 5-year overall survival rates of approximately 40–50% (10). Pathogenic or likely pathogenic (P/LP) germline variants of BAP1 are the most frequent and well-studied alteration in patients with mesothelioma who have increased cancer risk, and patients with the BAP1 P/LP variants have improved survival and better guided treatment decision-making with the use of platinum-based therapies (11). However, patients with unresectable disease or those managed with systemic therapy alone face substantially worse long-term outcomes (5).

The rare metachronous co-occurrence of MPM with other malignancies, such as prostate adenocarcinoma, is poorly understood, with only limited case reports describing such associations (12). Given the rarity of reported cases describing the metachronous occurrence of these two malignancies, this case highlights the importance of considering MPM as a distinct primary malignancy rather than metastatic disease and underscores the need for further investigation into shared molecular pathways that may contribute to the development of multiple primary cancers. This case report extends beyond the findings relating to the more commonly known pleural variant of malignant mesothelioma and provides awareness of the potential for malignant peritoneal mesothelioma to present as a secondary

malignancy rather than a metastasis of a primary malignancy. To that end, we present the case of a 63-year-old male with sequential diagnoses of prostate adenocarcinoma and malignant peritoneal mesothelioma, with the goal of raising awareness and prompting further investigation into the molecular mechanisms that may simultaneously predispose patients to these two primary malignancies.

Case Presentation

A 63-year-old Asian male with a past medical history of dyslipidemia and diabetes mellitus was diagnosed with high-grade Stage IV metastatic prostate cancer, Gleason score 4 + 5=9 with an initial prostate specific antigen (PSA) of 77 (normal value < 4.5 ng/mL). Family history of malignancy (reported by the patient) included lung cancer in his father at age 92, lung cancer in his paternal aunt in her 80s, uterine cancer in his paternal grandmother, and an unknown cancer in his maternal aunt at an unknown age. The patient was referred for genetic counseling to assess hereditary cancer risk. Findings revealed a likely pathogenic variant in the BARD1 gene and a variant of uncertain significance in the CDKN2A gene, both hypothesized as increased genetic susceptibility to cancer (to be discussed later). The patient underwent radical prostatectomy and was found to have peritoneal seeding and multiple pelvic lymph node involvement. A follow-up computed tomography (CT) scan showed several peritoneal implants, including an implant superior to the right liver lobe measuring 6.3 cm (Fig. 1), an implant in Morison's pouch measuring 6.1 cm axial, and a soft tissue density omental mass measuring 17.8 cm – interpreted as metastasis of prostate adenocarcinoma.

The patient then started triple therapy with leuprolide, abiraterone, and docetaxel in October 2023, which resulted in a nadir of PSA < 0.1 by December 2023. Chemotherapy for prostate adenocarcinoma was continued for a total of five cycles and concluded in February 2024, resulting in a favorable PSA response. However, follow-up CT scans showed interval worsening of extensive bulky peritoneal and omental metastases, including multiple new or increased lesions in the right hemidiaphragm and increased omental disease, measured at 22.8 cm in conglomerate on axial imaging, and previously estimated at 12.3 cm. There were also new implants and enlarged lymph nodes in the pelvis along the bilateral sidewall found in February 2024. At the time, the patient presented with recurring ascites and felt mild fatigue and fullness of the abdomen – thought to be side effects of chemotherapy. There was concern that the prostate adenocarcinoma had developed into an undifferentiated neuroendocrine cancer, so a biopsy was scheduled.

The percutaneous CT-guided biopsy of the left anterior omentum (March 2024) revealed low-grade epithelioid mesothelioma with a positive immunohistochemical stain of WT1 and calretinin. This is one year after the diagnosis of prostate adenocarcinoma. The patient had no known asbestos exposure, although a standardized asbestos exposure assessment was not performed. At the time, the patient presented with recurrent pleural effusions and atelectasis. He also experienced lumbar radiculopathy, but imaging revealed no evidence of pleural or bony metastatic disease. A follow-up CT scan post-diagnosis of epithelioid mesothelioma revealed increased peritoneal and omental masses compared to the prior scan. Examples of increased masses included: a right upper quadrant peritoneal nodularity with

mass effect on the right lobe of the liver that measured 3.6 cm in thickness, previously approximated as 3.1 cm; a mass along the dome of the liver that measured 4.2 cm craniocaudal, previously 3.5 cm; a large omental conglomerate that measured 25.4 cm in maximum axial extent, previously 22.8 cm; and a soft tissue mass interposed between the portal vein and inferior vena cava that measured 6.5 x 2.7 cm (previously 6.3 x 2.9 cm in March 2024). Inconsistencies in radiology reports posed challenges for comparing tumor growth and progression. MRIs of the brain and cervical spine were later performed and revealed no metastatic disease.

The patient began chemotherapy with cisplatin and pemetrexed with bevacizumab and later added immunotherapy with ipilimumab and nivolumab due to progression of the peritoneal lesions while on chemotherapy (See Table 1 for schedule). The patient's chemotherapy treatment plan was primarily influenced by his BARD1 mutation despite the presence of his CDKN2A mutation, which his care team described as a similar pathway to the more well-known BAP1 mutation. Additionally, the use of immunotherapy was extrapolated from the treatment pathways for pleural mesothelioma. Clinical and radiographic responses were noted. The patient completed a total of six cycles of chemotherapy and four cycles of immunotherapy. A CT scan showed improved peritoneal implants along the capsule of the liver and in the conglomerate mass in the left anterior lower abdomen, which measured 136 x 43 mm in August 2024, as compared to 169 x 50 mm in June 2024 (Fig. 2). The patient then had a laparoscopic evaluation for surgical debulking. CRS-HIPEC was performed 10 months after the diagnosis of malignant peritoneal mesothelioma (January 2025) and included a peritonectomy and omentectomy. There was a completeness of cancer resection score of 2 with residual disease on the bilateral iliac vessels, anterior bladder of the prostate, and right retroperitoneum. The peritoneal cancer index (PCI) was 15 and perfusion was performed with cisplatin for 90 minutes. Pathology showed small areas of poorly differentiated biphasic mesothelioma, with the remainder showing pathologic complete response. As of the submission of this case report, the patient is continuing clinical management.

Table 1
Schedule of Augmented Immunotherapy Due to Progression of Peritoneal Lesions While on Chemotherapy Alone.

Augmented Immunotherapy Schedule							
Nivolumab	6/4/24	7/10/24	8/1/24	8/21/24	9/11/24	10/3/24	11/13/24
Ipilimumab	6/4/24	-	8/1/24	-	9/11/24	-	11/13/24

Surgical Pathology Report Findings

The peritoneal seeding and pelvic lymph node involvement discovered on prostatectomy biopsy was initially interpreted as high-grade prostate adenocarcinoma with metastasis into the peritoneum. Hematoxylin and Eosin (H&E) slides of the prostate gland and omental biopsy were compared. Figure 3 shows the prostate biopsy at four different magnifications and Fig. 4 shows a side-by-side histologic comparison of the prostate tissue and omental biopsy (40x magnification).

The surgical pathology report from the prostatectomy conducted in June 2023 revealed a final pathologic diagnosis of metastatic prostate adenocarcinoma in the peritoneum. The report was later amended in March 2024 with a final pathologic diagnosis of low-grade epithelioid mesothelioma, highlighting the potential similarity of prostate adenocarcinoma and low-grade epithelioid mesothelioma on H&E stain (Fig. 4).

Discussion

The increasing incidence of second primary malignancies (SPM) is concerning, though not surprising, as individuals live longer. SPM can be synchronous – when the second cancer is diagnosed at the same time or within six months of the primary cancer – or metachronous – when the second cancer is diagnosed more than six months after the diagnosis of the primary cancer. The diagnostic criteria for SPM require that each malignancy present distinctly and that the probability of the secondary tumor originating from the primary tumor be excluded (12). Prostate cancer patients are at an increased risk of SPM, whether as the primary or secondary malignancy, as reported by Van Hemelrijck et al. and the Swedish National Prostate Cancer Registry (13). The study found that in 72,613 patients with prostate cancer, 17% of the patients had an additional cancer diagnosis within their lifetime. A more recent study by Bradley et al. found that among 237 patients who underwent CRS-HIPEC, 8 out of 17 patients with peritoneal mesothelioma were found to have a genitourinary malignancy. Most of the genitourinary malignancies were diagnosed prior to MPM (5 out 8), two were diagnosed after CRS-HIPEC, and one was diagnosed synchronously. Three patients had prior asbestos exposure, two had BAP1 mutations, and patients with both MPM and a GU malignancy had a higher PCI, findings that suggest a potential association between MPM and GU malignancy (14).

The Impact of Genetics

Alterations to BAP1, CDK2NA, NF2, SETD2, and TP53 have been associated with malignant peritoneal mesothelioma tumors, with overall survival worse when BAP1 mutations or deletions coexisted with loss on immunohistochemical (IHC) antibodies as compared to wild-type BAP1 or retained expression on IHC (4, 11). Moreover, BAP1 mutations or loss of expression had better overall survival independent of tumor histological subtype, age, and sex (4, 15, 16). Several studies have suggested that there is increased risk of pleural mesothelioma in patients with germline BAP1 mutations through modulating asbestos exposure or via gene-asbestos exposure interaction (12). These findings could suggest a link between prostate cancer and malignant mesothelioma, whether it is the pleural or peritoneal subtype.

In a recent study of 168 patients with mesothelioma, 28% of patients (45) had peritoneal mesothelioma and among the 39 patients with second malignant neoplasms, 8 had prostate cancer. 25 of 161 patients (16%) carried a P/LP germline variant, of which 36% (9 patients) had peritoneal mesothelioma. Asbestos exposure was not significantly different between P/LP variant carriers and those without germline variants. Most germline variants were in genes involved in DNA repair: BAP1 (29%), CHEK2 (21%), and ATM (11%), while other genes with a germline P/LP variant included ATR, DDX41, FANCM, HAX1,

MRE11A, MSH6, MUTYH, NF1, SAMD9L, and TMEM127. Each patient with multiple P/LP germline variants carried a P/LP BAP1 variant (17).

Genetic sequencing of this patient was performed to determine the hereditary cancer risk and inform treatment. Results revealed an autosomal dominant, heterozygous, pathogenic variant in BARD1 (BRCA1 Associated RING Domain 1), specifically c.2208T > A (p.Tyr736*) and a heterozygous, Variant of Uncertain Significance (VUS) in CDKN2A (p16INK4a), specifically c.343G > T (p.Val115Leu). There were no other known pathogenic mutations or VUS identified in any of the other genes analyzed. The cancer risk management options for BARD1 mutation carriers were discussed with the patient. The patient was notified that the impact of the VUS on prostate cancer is unknown and the limitations with a VUS were discussed. Literature shows that the BARD1 gene is commonly associated with an autosomal dominant predisposition to breast cancer and that the CDKN2A gene is associated with an autosomal dominant melanoma-pancreatic cancer syndrome and melanoma-neural system tumor syndrome (18).

BARD1 is a tumor-suppressor protein that forms a heterodimer with BRCA1; the complex acts as an E3 ubiquitin ligase that is involved in homologous recombination repair and the DNA damage response (19). Although the interaction between BRCA1/BARD1/BAP1 is not completely defined, experimental studies suggest that BAP1 can bind to the RING domain of BARD1, potentially influencing ubiquitination-related processes. However, whether this interaction directly disrupts the BARD1–BRCA1 heterodimer or its E3 ligase activity remains unclear (20, 21). Mutations in BARD1 are known to impair DNA repair pathways, including homologous recombination, and are associated with increased cancer risk, particularly in breast cancer. While BAP1 is also involved in DNA damage response, there is currently limited evidence supporting a shared mechanistic pathway or overlapping tumor spectrum mediated through a direct BARD1–BAP1 interaction. Further studies are needed to clarify the functional relevance of this potential interaction.

Unlike BARD1, CDKN2A has a more established role in MPM. Both homozygous and heterozygous deletions of CDKN2A have been observed, with the former more often reported (22). Homozygous CDKN2A deletions were identified in 29% of the 86 MPM patient cases reported by Singhi et al., with a 2-year overall survival rate of only 18% with a median of 8 months when patients had both homozygous CDKN2A deletions and hemizygous NF2 loss (8). When the CDKN2A alteration occurs in the germline, it is also associated with an increased propensity for hereditary cancer syndromes (17). Based on what is currently known about genetic variants in mesothelioma, CDKN2A has a clearer biological link to MPM and may be the more relevant genetic alteration in this patient's case.

Alterations in BRCA1, BRCA2, HOXB13, and mismatch repair genes are typically associated with a higher lifetime risk of prostate cancer, with patients who are carriers of the BRCA pathogenic variants typically having the most aggressive disease at time of diagnosis (12). Other than the most studied association between BARD1 and breast cancer, little else is currently known about the susceptibility of cancers in those with BARD1 mutations (23). However, since BARD1 directly interacts with BRCA1 and/or BAP1 in

the formation of a heterodimer, this raises the possibility of correlation between prostate cancer and malignant peritoneal mesothelioma in this specific patient case and warrants further investigation.

Conclusion

While genetic alterations in malignant mesothelioma have been studied, research has largely focused on the pleural variant with little investigation specific to MPM. The co-occurrence of prostate adenocarcinoma and MPM remains rarely reported in the literature, and the current lack of definitive linkage between these two malignancies risks missed or delayed diagnoses. To our knowledge, this is the first report suggesting a potential link between BARD1 and mesothelioma, and this case further underscores the diagnostic challenge posed by the histologic similarity between metastatic prostate adenocarcinoma and low-grade epithelioid MPM on H&E staining. Paired germline and somatic sequencing in prostate adenocarcinoma and malignant peritoneal mesothelioma represent a promising approach to elucidate shared molecular mechanisms, and validation in larger studies is needed to better characterize this rare co-occurrence and improve diagnostic accuracy.

Abbreviations

ATM
Ataxia telangiectasia mutated
ATR
Ataxia telangiectasia and Rad3 related
BAP1
BRCA1 associated protein 1
BARD1
BRCA1 associated RING domain 1
BRCA1
Breast cancer gene 1
BRCA2
Breast cancer gene 2
CDKN2A
Cyclin dependent kinase inhibitor 2A
CHEK2
Checkpoint kinase 2
CRS
HIPEC–Cytoreductive surgery with hyperthermic intraperitoneal chemotherapy
CT
Computed tomography
DDX41
DEAD–box helicase 41
DNA

Deoxyribonucleic acid
FANCM
Fanconi anemia complementation group M
H&E
Hematoxylin and eosin
HAX1
HCLS1 associated protein X–1
HOXB13
Homeobox B13
IHC
Immunohistochemistry
LP
Likely pathogenic
MRE11A
MRE11 homolog A
MPM
Malignant peritoneal mesothelioma
MRI
Magnetic resonance imaging
MSH6
MutS homolog 6
MUTYH
MutY DNA glycosylase
NF1
Neurofibromin 1
NF2
Neurofibromin 2
PCI
Peritoneal cancer index
P/LP
Pathogenic or likely pathogenic
PSA
Prostate–specific antigen
SETD2
SET domain containing 2
SPM
Second primary malignancy
TMEM127
Transmembrane protein 127
TP53

Tumor protein p53
VUS
Variant of uncertain significance
WT1
Wilms tumor 1

Declarations

Ethics approval and consent to participate –

Not applicable (case report). The patient gave informed consent to the publication of this data, the institutional consent form for research participant publication approval by Springer Nature was used to obtain consent.

Consent for publication -

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of this written consent is available for review by the Editor-in-Chief of this journal.

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Author Contribution

VW, EN: conceptualization. VW, EN: review of the literature. VW, TJ: digital image collation. VW, EN, VC, TJ: writing, editing, and review of manuscript. All authors read and approved the final manuscript.

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Availability of data and materials –

All cited literature and data was found on https://pubmed.ncbi.nlm.nih.gov/. Images and scans are available upon request addressed to the corresponding author.

Competing interests – The authors declare that they have no competing interests.

References

1. Strange CD, Marom EM, Ahuja J, Shroff GS, Gladish GW, Carter BW, et al. Imaging of Malignant Pleural, Pericardial, and Peritoneal Mesothelioma. Adv Anat Pathol. 2023;30(4):280–91.
2. Abdul Razzak S, Awan F, Ahmed S. Malignant peritoneal mesothelioma-a diagnostic challenge. J Surg Case Rep. 2022;2022(12):rjac555.
3. Ullah A, Waheed A, Khan J, Mishra A, Tareen B, Nama N, et al. Incidence, Survival Analysis and Future Perspective of Primary Peritoneal Mesothelioma (PPM): A Population-Based Study from SEER Database. Cancers (Basel). 2022;14(4).
4. Gregory SN, Sarvestani AL, Blakely AM. Malignant peritoneal mesothelioma literature review: past, present, and future. Dig Med Res. 2022;5.
5. Meyerhoff RR, Yang CF, Speicher PJ, Gulack BC, Hartwig MG, D'Amico TA, et al. Impact of mesothelioma histologic subtype on outcomes in the Surveillance, Epidemiology, and End Results database. J Surg Res. 2015;196(1):23–32.
6. Wadowski B, De Rienzo A, Bueno R. The Molecular Basis of Malignant Pleural Mesothelioma. Thorac Surg Clin. 2020;30(4):383–93.
7. Sekido Y, Sato T. NF2 alteration in mesothelioma. Front Toxicol. 2023;5:1161995.
8. Singhi AD, Krasinskas AM, Choudry HA, Bartlett DL, Pingpank JF, Zeh HJ, et al. The prognostic significance of BAP1, NF2, and CDKN2A in malignant peritoneal mesothelioma. Mod Pathol. 2016;29(1):14–24.
9. Quetel L, Meiller C, Assié JB, Blum Y, Imbeaud S, Montagne F, et al. Genetic alterations of malignant pleural mesothelioma: association with tumor heterogeneity and overall survival. Mol Oncol. 2020;14(6):1207–23.
10. Wang T, Li H, Ye B, Zhang D. Value of cytoreductive surgery combined with hyperthermic intraperitoneal chemotherapy to treat malignant peritoneal mesothelioma. Am J Transl Res. 2021;13(9):10712–20.
11. Offin M, Yang SR, Egger J, Jayakumaran G, Spencer RS, Lopardo J, et al. Molecular Characterization of Peritoneal Mesotheliomas. J Thorac Oncol. 2022;17(3):455–60.
12. Soni TP, Soni S, Somani N, Bhattacharya T, Kucha N. Prostate Carcinoma and Pleural Mesothelioma: An Extremely Rare Co-occurrence. Cureus. 2019;11(5):e4655.
13. Van Hemelrijck M, Drevin L, Holmberg L, Garmo H, Adolfsson J, Stattin P. Primary cancers before and after prostate cancer diagnosis. Cancer. 2012;118(24):6207–16.
14. Bradley EM, Rogers JL, Kuo MC, Magge D. Co-occurrence of peritoneal mesothelioma and genitourinary cancers: a case series with comparative outcomes. Pleura Peritoneum. 2025;10(4):177–84.
15. Carr NJ. New insights in the pathology of peritoneal surface malignancy. J Gastrointest Oncol. 2021;12(Suppl 1):S216-S29.

16. Leblay N, Lepretre F, Le Stang N, Gautier-Stein A, Villeneuve L, Isaac S, et al. BAP1 Is Altered by Copy Number Loss, Mutation, and/or Loss of Protein Expression in More Than 70% of Malignant Peritoneal Mesotheliomas. J Thorac Oncol. 2017;12(4):724–33.
17. Mitchell OD, Gilliam K, Del Gaudio D, McNeely KE, Smith S, Acevedo M, et al. Germline Variants Incidentally Detected via Tumor-Only Genomic Profiling of Patients With Mesothelioma. JAMA Netw Open. 2023;6(8):e2327351.
18. Jacobs M, Murad A, Cho N, al. e. CDKN2A Cancer Predisposition GeneReviews [Internet] [cited 2025 November 24]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK616232/.
19. Zhao W, Steinfeld JB, Liang F, Chen X, Maranon DG, Jian Ma C, et al. BRCA1-BARD1 promotes RAD51-mediated homologous DNA pairing. Nature. 2017;550(7676):360–5.
20. Nishikawa H, Wu W, Koike A, Kojima R, Gomi H, Fukuda M, et al. BRCA1-associated protein 1 interferes with BRCA1/BARD1 RING heterodimer activity. Cancer Res. 2009;69(1):111–9.
21. Yue X, Liu T, Wang X, Wu W, Wen G, Yi Y, et al. Pharmacological inhibition of BAP1 recruits HERC2 to competitively dissociate BRCA1-BARD1, suppresses DNA repair and sensitizes CRC to radiotherapy. Acta Pharm Sin B. 2023;13(8):3382–99.
22. Pinton G, Wang Z, Balzano C, Missaglia S, Tavian D, Boldorini R, et al. Determines Mesothelioma Cell Fate to EZH2 Inhibition. Front Oncol. 2021;11:678447.
23. Stempa K, Wokołorczyk D, Kluźniak W, Rogoża-Janiszewska E, Malińska K, Rudnicka H, et al. Do BARD1 Mutations Confer an Elevated Risk of Prostate Cancer? Cancers (Basel). 2021;13(21).

Figures

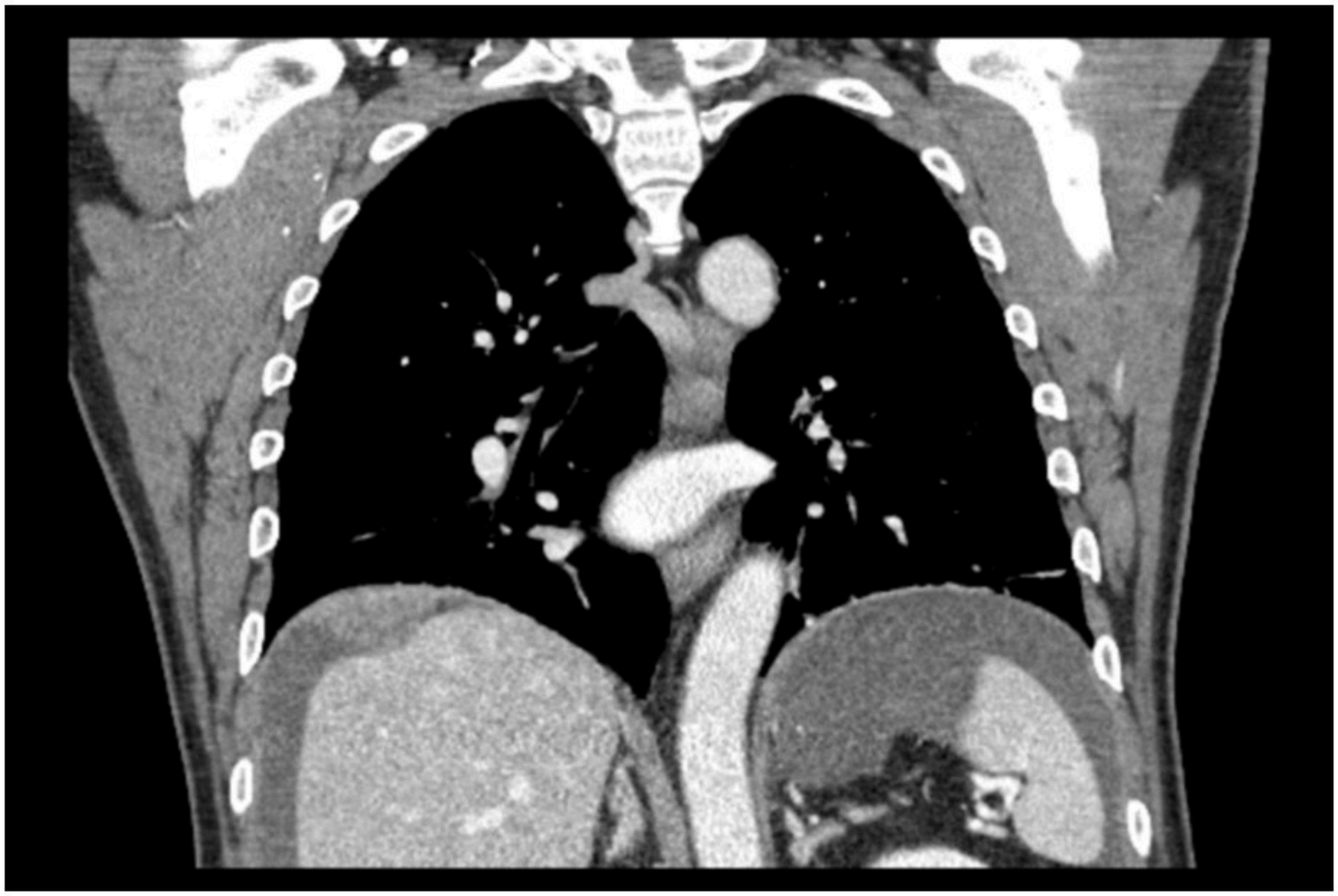


Figure 1

This image shows a peritoneal implant superior to the right liver lobe, measuring 6.3 cm. This image is part of a series of CT images of the chest, abdomen, and pelvis acquired with 150 mL Omnipaque 300 intravenous contrast in September 2023.

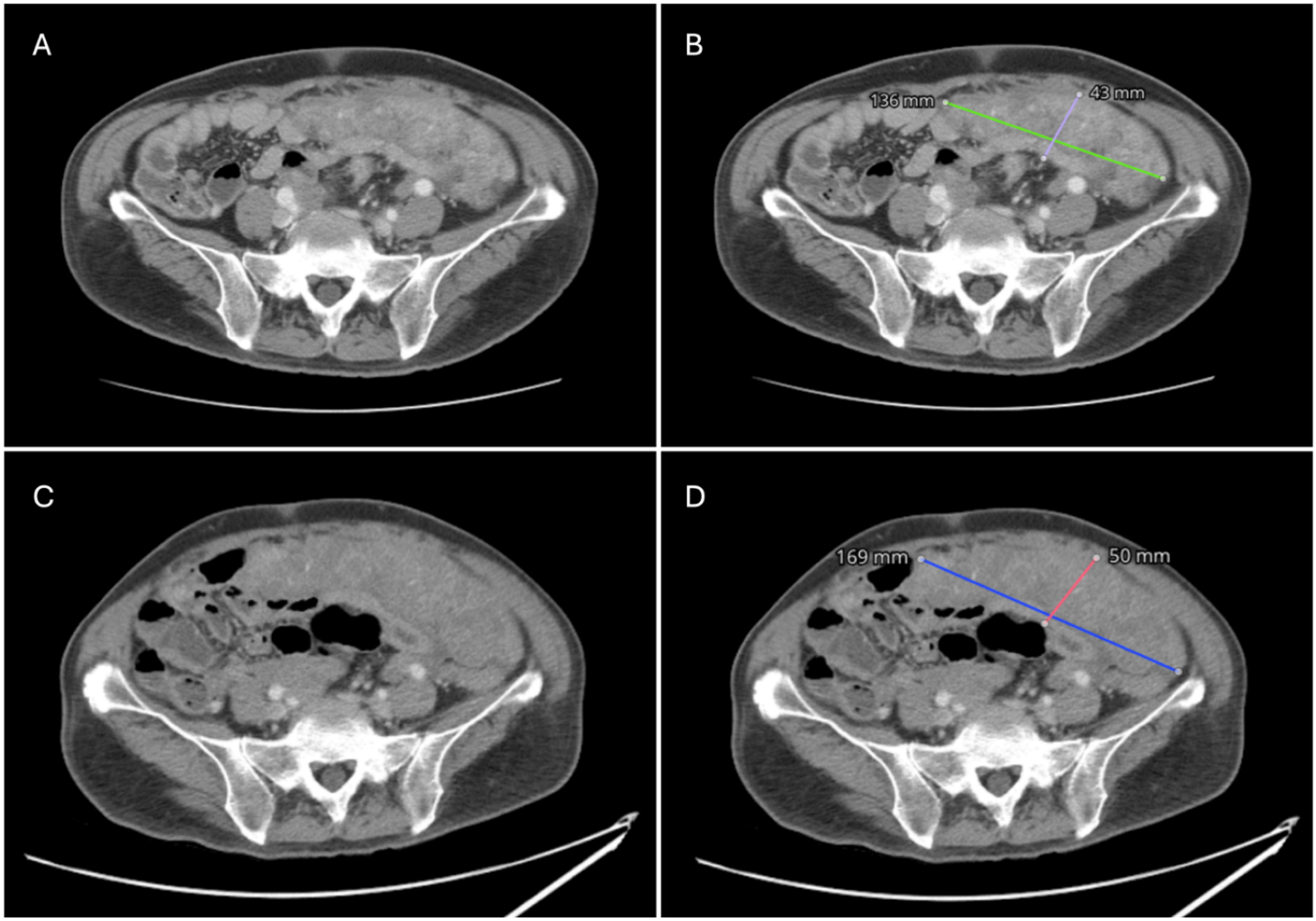


Figure 2

This image shows a comparison of a conglomerate of peritoneal implants in the left anterior abdomen while on an immunotherapy regimen. The top images show an improvement in size of the peritoneal implants as compared to the bottom images. Images A and B are identical, with measurements (136mm x 43mm) superimposed onto image B. This image was taken August 2024 as part of a series of CT images of the chest, abdomen, and pelvis acquired with 150 mL Omnipaque 300 intravenous contrast. Images C and D are identical, with measurements (169mm x 50mm) superimposed onto image D. This image was taken June 2024 as part of a series of CT images of the abdomen and pelvis, acquired with 100 mL Omnipaque 300 intravenous contrast.

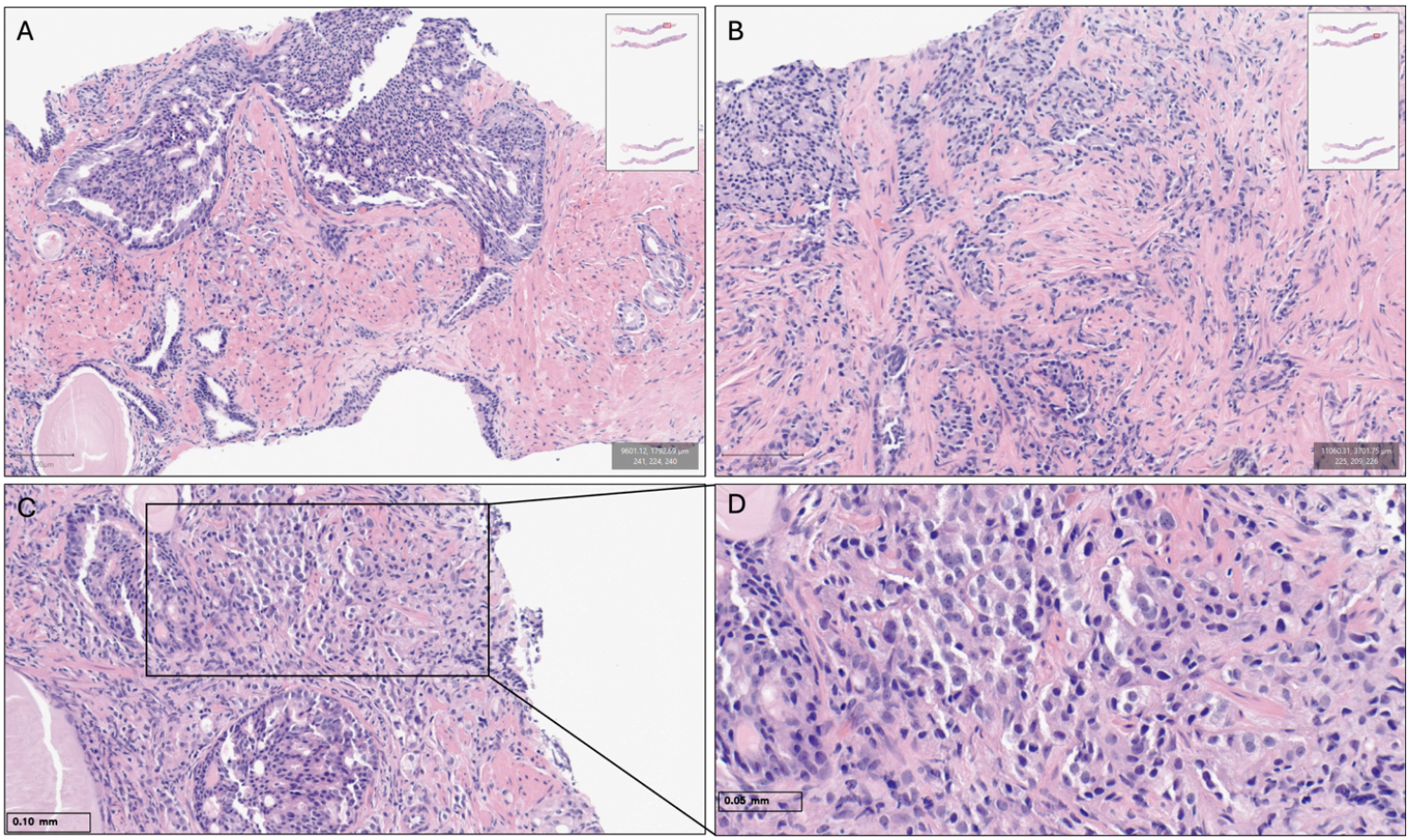


Figure 3

This series of images shows a hematoxylin and eosin-stained prostate biopsy during a prostatectomy. Image A shows the biopsy at 9x magnification. Image B shows the biopsy at 12x magnification. Image C shows the biopsy at 20x magnification. Image D shows the biopsy at 40x magnification.

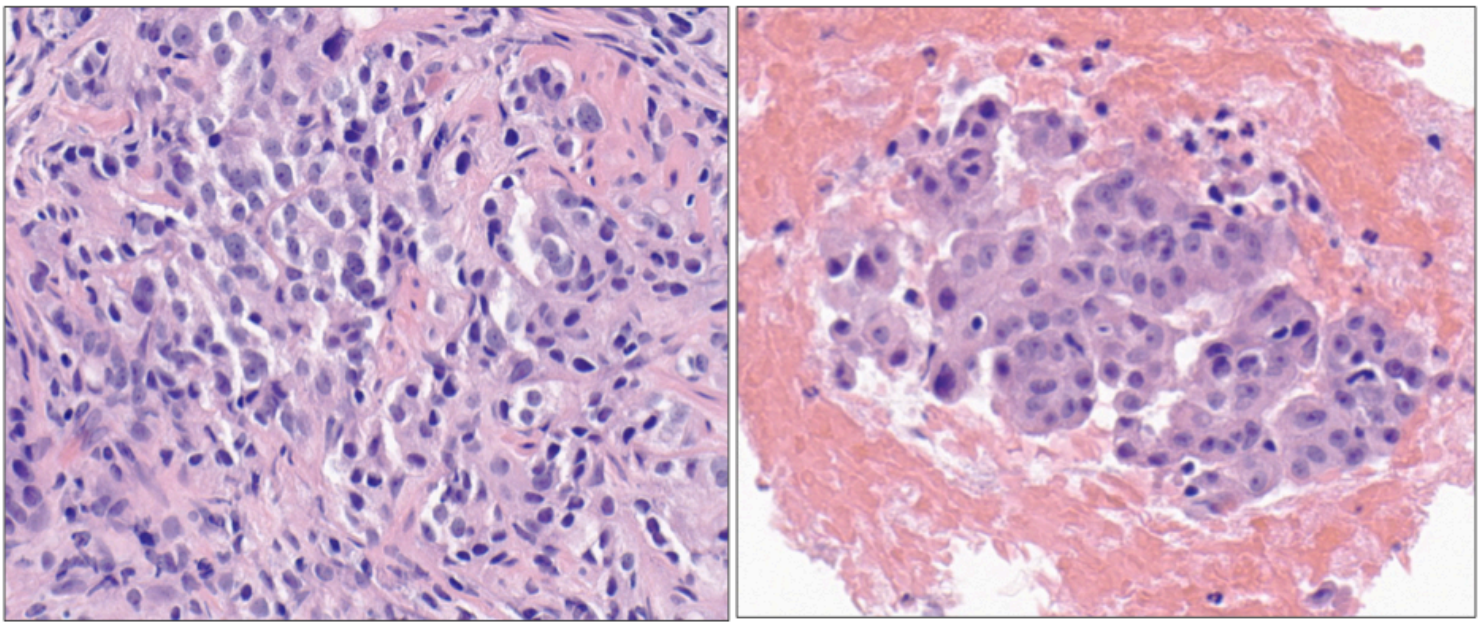


Figure 4

These images show a side-by-side comparison of the hematoxylin and eosin-stained prostate biopsy and omental biopsy. The left image shows the prostate biopsy at 40x magnification. The right image shows the omental biopsy at 40x magnification. The omental biopsy was initially diagnosed as metastatic prostate adenocarcinoma of the peritoneum but later amended to low-grade epithelioid mesothelioma. This shows the potential similarity of prostate adenocarcinoma and low-grade epithelioid mesothelioma on hematoxylin and eosin stain, highlighting the need to differentiate between the two types of malignancies.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- CAREChecklistPeritonealMesothelioma2026.pdf