A rare case of uterine carcinosarcoma in a 21-year-old woman with hereditary breast and ovarian cancer syndrome: A case report

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Abstract. Uterine carcinosarcoma (UCS) is a rare and aggressive malignancy that typically affects postmenopausal women. Its occurrence in young adults, particularly due to hereditary breast and ovarian cancer syndrome (HBOC), is exceptionally uncommon. The present report describes a case of a 21-year-old woman with persistent uterine bleeding who was found to have a uterine mass with para-aortic and pelvic lymphadenopathy and multiple pulmonary metastases. To control bleeding and obtain a definitive diagnosis, the patient underwent a total hysterectomy with bilateral salpingo-oophorectomy. Histopathological examination revealed International Federation of Gynecology and Obstetrics stage IVB UCS. This manifested as poorly differentiated carcinoma and heterologous sarcomatous components with chondrosarcomatous and rhabdomyosarcomatous differentiation. Comprehensive genomic profiling identified a *BRCA1* frameshift mutation, a *TP53* splice-site mutation, a *PIK3CA* mutation and a positive homologous recombination deficiency (HRD) signature. After genetic counseling, germline genetic testing for *BRCA1* and *TP53* was performed. This confirmed a pathogenic germline *BRCA1* mutation. The patient was treated with four cycles of paclitaxel and carboplatin, followed by an additional four cycles of combination chemotherapy with durvalumab. The patient achieved a complete radiologic response. Maintenance therapy with durvalumab and olaparib was initiated, and the patient has remained progression-free for 10 months. To the best of our knowledge, this represents one of the youngest reported cases of UCS associated with HBOC. The case highlights the value of genomic profiling and germline testing for personalized treatment strategies. The successful use of platinum-based chemotherapy, immune checkpoint blockade and poly (ADP-ribose) polymerase inhibition to treat this HRD-positive, mismatch repair-proficient tumor demonstrates the potential of biomarker-driven therapy for UCS.

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Abbreviations: FIGO, International Federation of Gynecology and Obstetrics; HBOC, hereditary breast and ovarian cancer syndrome; HRD, homologous recombination deficiency; MMR, mismatch repair; PARP, poly (ADP-ribose) polymerase; UCS, uterine carcinosarcoma

Key words: BRCA1, HBOC, UCS

Introduction

Hereditary breast and ovarian cancer syndrome (HBOC) is caused by pathogenic germline variants in *BRCA1* or *BRCA2* and significantly elevates the risk of breast and ovarian cancers. Emerging evidence suggests that there is also a modestly increased risk of uterine cancers, especially uterine papillary serous carcinoma (UPSC) (1-3). A 2021 meta-analysis reported a 2.2-fold greater risk of uterine cancer, particularly UPSC, in *BRCA1/2* mutation carriers [standardized incidence ratio (SIR) 17.97 for UPSC] (4). However, most endometrial carcinomas in carriers are histologically heterogeneous, so the precise relationship between the mutation and uterine carcinosarcoma (UCS) is hard to quantify (3). In addition, a recent genomic study has suggested that UCS shares molecular characteristics with high-grade endometrial carcinomas, highlighting the importance of molecular profiling in understanding tumor biology and guiding therapeutic strategies (5).

UCS is a rare, aggressive neoplasm comprised epithelial and mesenchymal components. It accounts for fewer than 5% of uterine cancers (6). It is typically seen in postmenopausal women, and its incidence peaks in patients over 60. It is exceptionally uncommon in young adults (6). Furthermore, the molecular features and clinical behavior of UCS arising in patients with hereditary cancer syndromes remain poorly characterized.

This report presents a 21-year-old female patient with a *BRCA1* mutation who developed UCS. To the best of our knowledge, this represents one of the youngest reported cases

of uterine carcinosarcoma associated with hereditary breast and ovarian cancer syndrome. This case suggests a potential need to expand the HBOC tumor spectrum to include early UCS, as well as possible reconsideration of surveillance and risk-reduction strategies in young *BRCA* mutation carriers.

Case report

A 21-year-old woman presented with continuous uterine bleeding. When ultrasound revealed a tumor in the uterine cavity, she was referred to our institution for further evaluation. Relevant family history included a paternal grandmother who had suffered breast and peritoneal cancer, and prostate cancer in a maternal grandfather and a maternal uncle. The patient's laboratory results identified severely low hemoglobin levels (5.5 g/dl). Her hormone levels, including estradiol, luteinizing hormone, follicle-stimulating hormone, and prolactin, were within normal limits. Her levels of lactate dehydrogenase (330 U/l) and cancer antigen (CA) 19-9 (81.6 U/ml) were mildly elevated. Other tumor markers, including CA125 and carcinoembryonic antigen, were within normal limits.

Pelvic magnetic resonance imaging revealed an irregular mass with mild contrast enhancement and restricted diffusion. This protruded from the myometrium into the uterine cavity (Fig. 1A-E). As both cervical and endometrial cytology were negative, a hysteroscopic biopsy of the tumor was performed. The tumor was white, irregular, and showed neovascularization (Fig. 2A). Endometrial biopsy revealed densely proliferating epithelioid cells with nuclear atypia and prominent mitoses, consistent with poorly differentiated carcinoma. However, atypical polygonal cells were also present. Immunohistochemistry showed reduced epithelial marker expression and weak positivity for smooth muscle markers, raising the possibility of carcinosarcoma (Fig. 2B-E). Computed tomography (CT) revealed para-aortic and pelvic lymph node metastasis and multiple pulmonary metastases (Fig. 3A-C). The patient underwent total hysterectomy with bilateral salpingo-oophorectomy to control genital bleeding and obtain a definitive diagnosis.

Histopathological examination revealed a mixture of poorly differentiated endometrioid carcinoma and heterologous sarcomatous components with chondrosarcomatous and rhabdomyosarcomatous differentiation (Fig. 4A-F). This led to a diagnosis of UCS at FIGO stage IVB. The patient underwent comprehensive genomic profiling with FoundationOne® CDx (Foundation Medicine, Cambridge, MA, USA), a hybrid-capture next-generation sequencing (NGS) assay that interrogate 324 cancer-related genes, including *POLE*, and selected introns to detect base substitutions, insertions/deletions, copy-number alterations, and gene rearrangements. The report also included tumor mutational burden (TMB), microsatellite (MS) status, and homologous recombination deficiency (HRD) signature. Results showed a *BRCA1* V923fs*76 mutation [variant allele frequency (VAF): 81.5%], a *TP53* splice-site mutation (920-2A>G; VAF: 62.8%), and a *PIK3CA* Q546K mutation (VAF: 10.2%). The tumor was HRD signature-positive, MS-stable, and had a TMB of 2 mutations per megabase (2 Muts/Mb). Germline genetic testing for *BRCA1* and *TP53* was performed after genetic counseling, and a germline mutation was identified in *BRCA1*.

The patient was treated with four cycles of paclitaxel and carboplatin chemotherapy. Subsequently, durvalumab and olaparib were approved for reimbursement under the Japanese national health insurance system for the treatments of endometrial cancer. Therefore, the patient received an additional four cycles of combination chemotherapy with paclitaxel, carboplatin, and durvalumab. Post-chemotherapy CT showed a complete response according to RECIST criteria (Fig. 5A-C). Immunohistochemistry confirmed mismatch repair, and maintenance therapy with durvalumab and olaparib was initiated. The patient has remained progression-free for 10 months.

Discussion

Carcinosarcoma is unusual in young women, with only sporadic cases reported (7-9), and its occurrence within the HBOC spectrum is exceptionally rare and poorly defined. Prior young-onset reports have lacked broad molecular interrogation: Al Dallal *et al* (7) assessed p53 and mismatch-repair proteins by immunohistochemistry without comprehensive genomic testing, whereas Soror *et al* (8) applied a 34-gene NGS panel that included *BRCA1/2* and found no pathogenic *BRCA1/2* variants. Given the very limited number of young-onset cases reported in the literature, it remains difficult to discuss whether or not differences between races and ethnicities may play a role in disease presentation in this age group. In this regard, our 21-year-old patient underwent comprehensive gene testing that identified a germline *BRCA1* frameshift mutation and HRD positivity, establishing HBOC and enabling prediction of sensitivity to platinum chemotherapy and PARP inhibition. This case therefore supports heightened clinical vigilance and a low threshold for early germline evaluation and tumor profiling-particularly in young patients with suggestive personal or family histories-and provides the rationale for the genomically guided treatment strategy described below.

Advances in molecular profiling over the last decade have transformed our understanding of UCS. Integrated genomic analyses, most notably through The Cancer Genome Atlas (TCGA), have characterized UCS as a metaplastic carcinoma arising from a single epithelial clone, with sarcomatous components emerging through epithelial-mesenchymal transition (5). *TP53* mutations have been found in around 90% of UCS cases. As in endometrioid and serous uterine carcinomas, alterations are also often seen in *PTEN*, *PIK3CA*, *PPP2R1A*, *FBXW7*, and *KRAS* (5). TCGA and the Proactive Molecular Risk Classifier for Endometrial Cancer (ProMisE) have identified four novel molecular endometrial cancer subgroups (10,11). These are POLE/ultramutated (POLE), microsatellite-instable/hypermutated (MSI), copy-number-high/TP53-mutant (CNH), and copy-number-low/TP53-wild-type (CNL). A recent meta-analysis of four studies reported the pooled prevalence of the TCGA groups among patients with UCS. These were 5.3% POLE, 7.3% MSI, 73.9% CNH, and 13.5% CNL (12). Travaglino *et al* (13) performed a systematic review and meta-analysis including 263 uterine carcinosarcoma (UCS) classified according TCGA groups. They found that POLE-mutated tumors had an excellent prognosis with no recurrences or deaths, MSI tumors showed intermediate outcomes, while TP53-mutated tumors (CNH group) and no specific molecular profile (NSMP) tumors (CNL group)

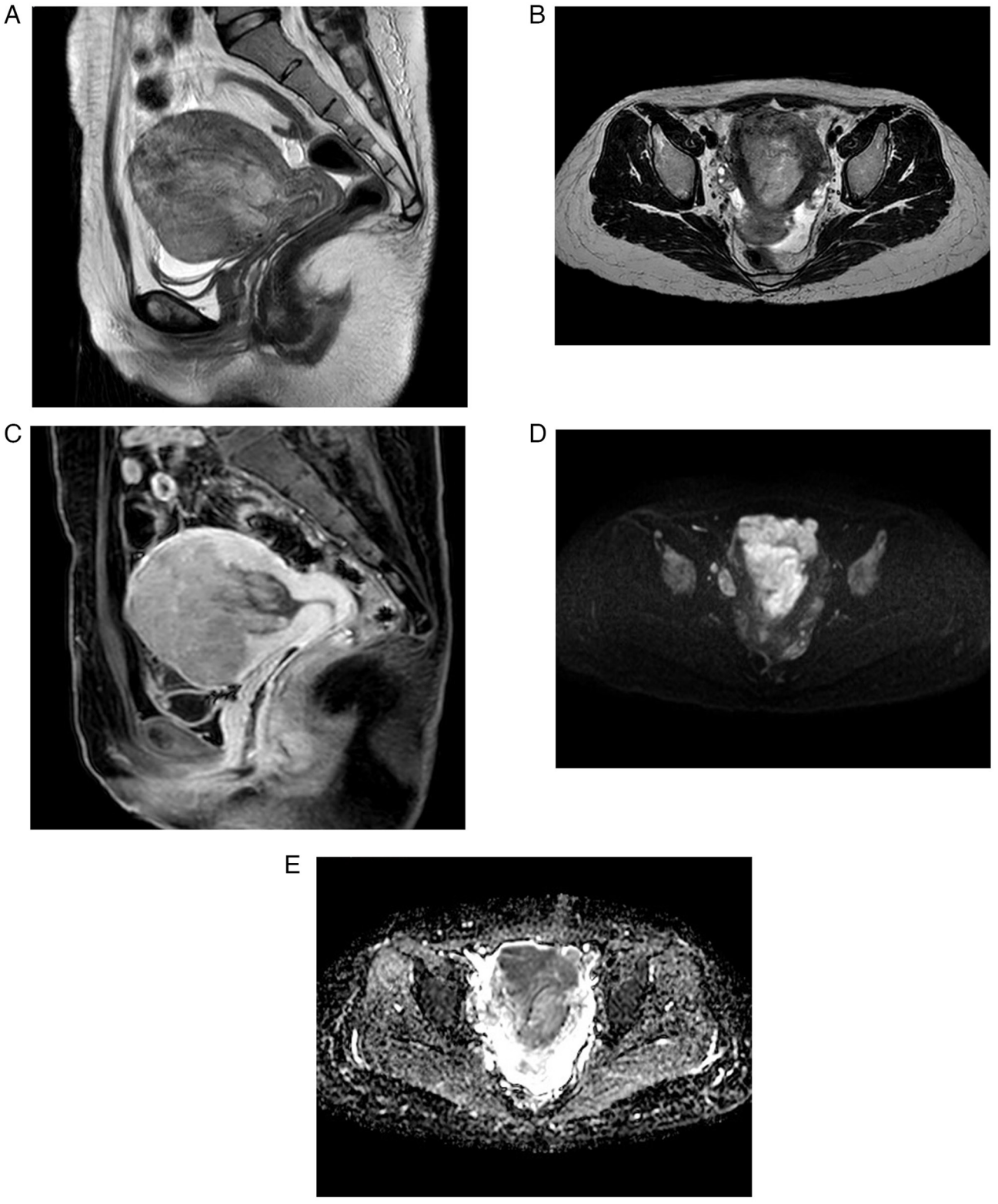

Figure 1. MRI findings. (A) Sagittal T2-weighted MRI showing an irregular uterine mass protruding from the myometrium into the endometrial cavity. (B) Axial T2-weighted MRI demonstrating the same uterine mass with heterogeneous SI. (C) Contrast-enhanced MRI showing mild enhancement of the lesion. (D) Diffusion-weighted imaging demonstrating high SI in the lesion. (E) Apparent diffusion coefficient map showing corresponding low SI, indicating diffusion restriction. SI, signal intensity.

were associated with poor survival, paralleling the prognostic pattern observed in endometrioid and serous endometrial carcinoma (13). Clinically, poorer outcomes in TP53-abnormal endometrial cancer likely reflect enrichment for advanced stage at presentation (14,15) and relative insensitivity to limited adjuvant radiotherapy (15); in addition, p53 overexpression has been linked to radioresistance and inferior survival (15,16). Complementing these observations, a large real-world series (n=2,235) showed tiered survival by molecular class: TP53-mutated UCS had a shorter median overall

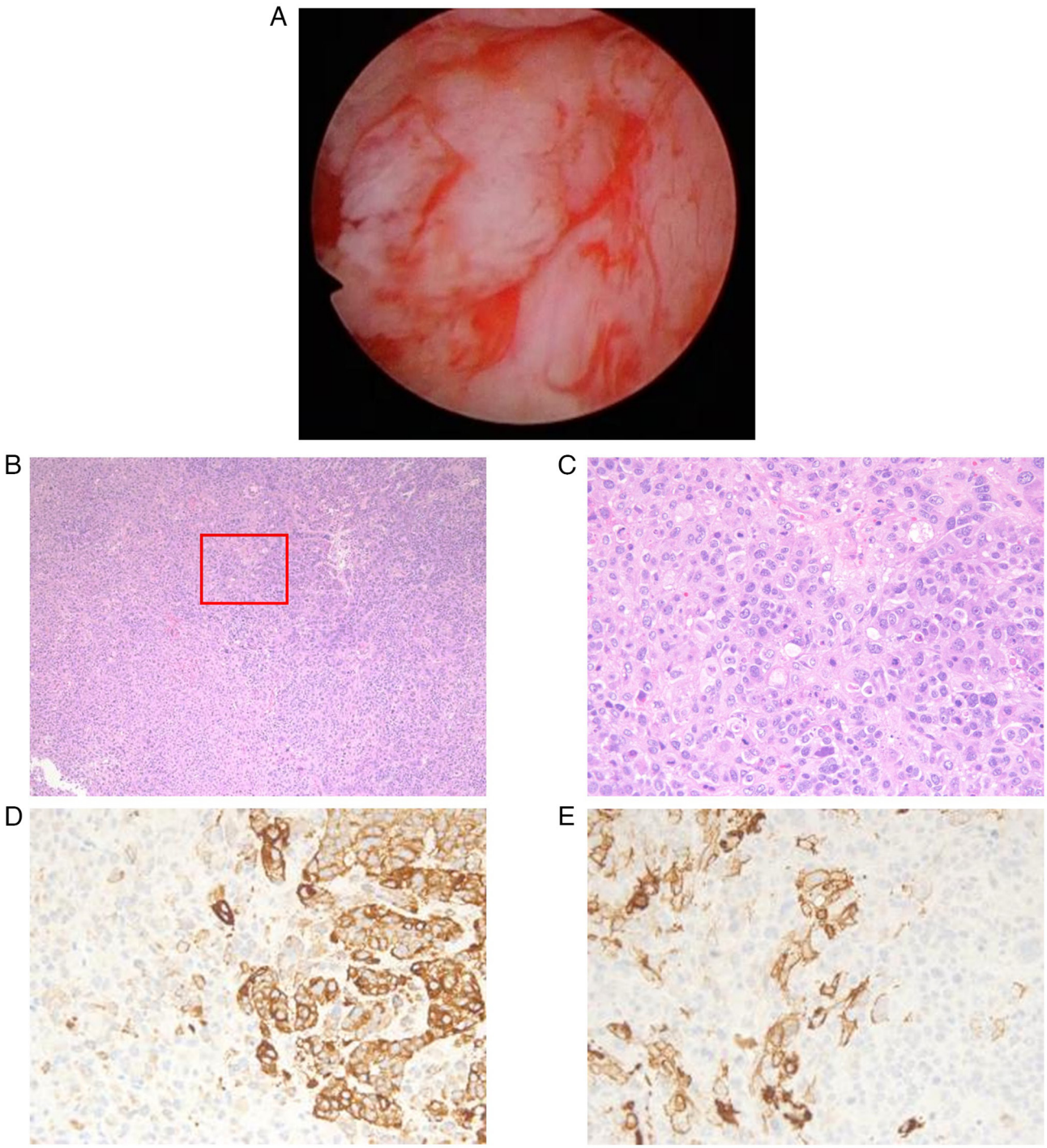

Figure 2. Hysteroscopic and histopathological findings of the uterine tumor. (A) Hysteroscopic examination revealed a white, irregular tumor with abundant neovascularization in the uterine cavity. (B) H&E staining showing dense proliferation of epithelial-like cells with nuclear atypia (magnification, x100). (C) Higher magnification view of the boxed area in (B) showing poorly differentiated epithelial-like tumor cells with numerous mitotic figures, consistent with poorly differentiated carcinoma (magnification, x400). (D) Immunohistochemical staining showing decreased expression of the epithelial marker AE1/3 (magnification, x400). (E) Immunohistochemical staining showing weak positivity for α-smooth muscle actin, suggesting mesenchymal differentiation (magnification, x400).

survival than TP53-mutated endometrioid cancer [27.9 vs. 35.3 months; hazard ratio (HR), 1.3; 95% confidence interval (CI), 1.1-1.5], and even TP53-wild-type UCS underperformed TP53-wild-type endometrioid disease (29.4 vs. 70.7 months; HR, 2.0; 95% CI, 1.5-2.7) (17). Together, these data indicate that while TP53 alterations define the poorest-prognosis subgroup, 'being UCS' adds risk beyond TP53 class alone.

Our patient's tumor harbored a *TP53* splice-site mutation and a *PIK3CA* Q546K mutation, consistent with the molecular hallmarks of UCS that have been identified in large cohorts. To the best of our knowledge, no studies have yet investigated the functional implications of TP53 splice-site mutations and PIK3CA Q546K mutations in UCS. Furthermore, the absence of *POLE* mutations and a microsatellite-stable status led to

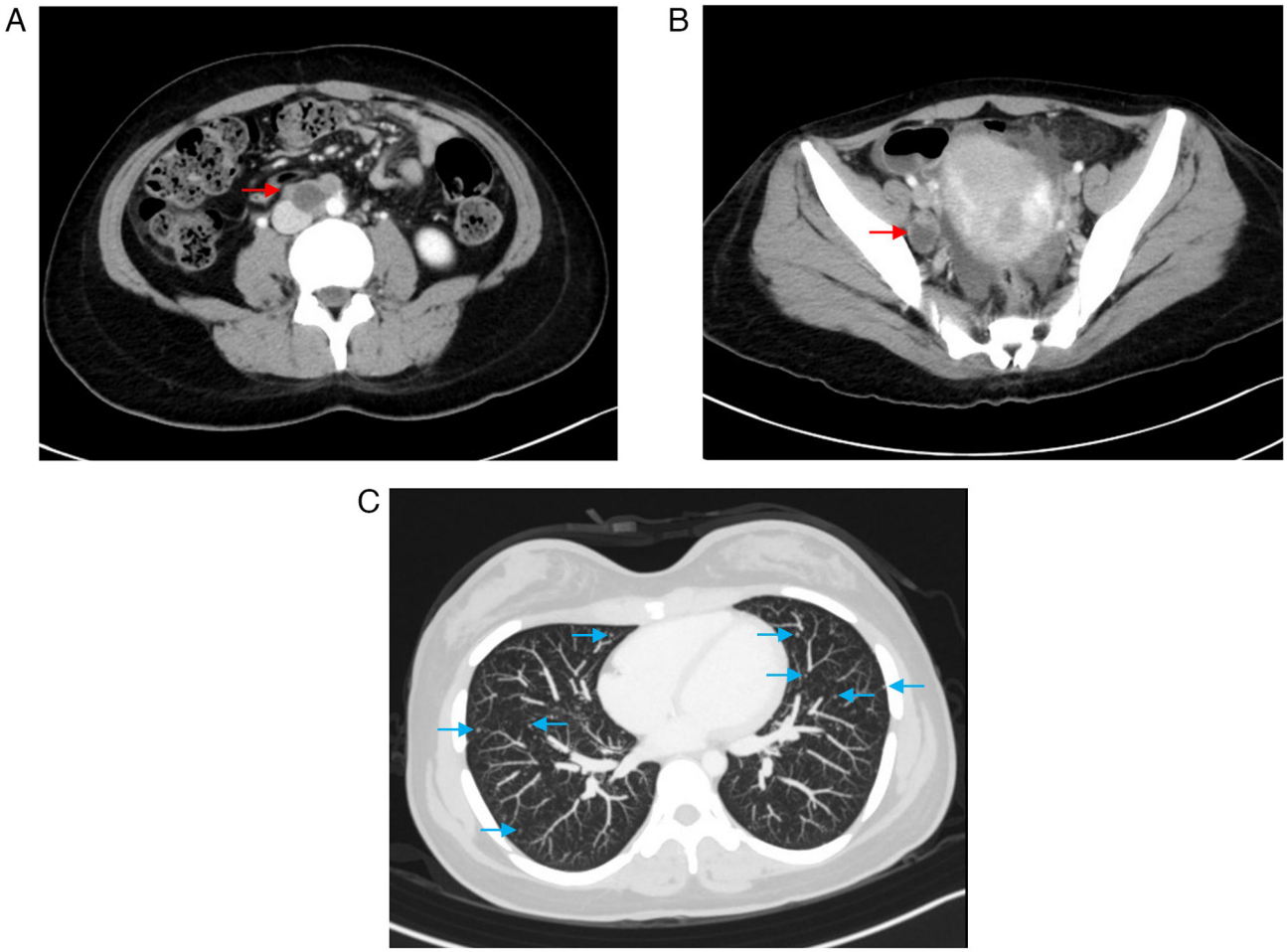

Figure 3. Computed tomography findings. (A) Enlarged para-aortic lymph nodes (red arrow). (B) Enlarged pelvic lymph nodes (red arrow). (C) Multiple pulmonary nodules (blue arrows). These findings were suggestive of metastatic disease.

its classification as the CNH subtype. Importantly, our patient also carried a germline *BRCA1* mutation, and the high variant allele frequency of 81% strongly suggests biallelic inactivation within the tumor, highlighting a potential driver event in tumorigenesis. This finding is particularly notable because, in a cohort of 167 UCS, Sia *et al* (18) reported germline mutations in 19 patients (11%); however, all affected individuals were aged 52-81 years (median, 61), and individual-level data were not available. In contrast, our patient was diagnosed at the exceptionally young age of 21, making this case one of the earliest-onset UCS associated with a germline *BRCA1* mutation reported to date. In the Sia cohort, six out of 167 cases (3.6%) harbored germline *BRCA1/2* mutations, and the corresponding tumors exhibited biallelic inactivation, indicating these alterations are likely oncogenic drivers in UCS (18). Consistently, in a series of endometrial cancers with germline *BRCA* mutations, UCS comprised 28.5% of cases (6/21), further supporting a link between *BRCA*-associated tumorigenesis and this aggressive histologic subtype (19). Nevertheless, a lack of IHC is a limitation of the present study.

Based on the results of the GOG-232B and GOG-261 trials, the recommended first-line treatment for advanced UCS is a carboplatin/ paclitaxel doublet due to its non-inferiority and good toxicity profile (20,21). However, the outcomes previously reported in stage IV cases have been poor, with a median overall survival time of just 8 months (22). Notably, tumors exhibiting HRD, particularly those with *BRCA* mutations, have increased sensitivity to platinum chemotherapy due to their impaired DNA repair capabilities. Thus, our patient's complete radiologic response following four cycles of paclitaxel/carboplatin may be attributable to an underlying HRD-driven susceptibility (23). This suggests the potential utility of HRD and BRCA testing of patients with UCS to predict chemosensitivity.

Recent preclinical studies have elucidated the mechanisms behind the synergistic effects of combined PARP inhibitors (PARPi) and immune checkpoint inhibitors (ICIs), and support the superiority of this approach over PARPi monotherapy. PARP inhibition induces DNA double-strand breaks. The accumulation of these breaks causes the activation of cyclic GMP-AMP synthase (cGAS). This is a stimulator of interferon genes (STING) pathway that enhances type I interferon signaling and dendritic cell recruitment, promoting antitumor immunity (24). PARP inhibition also upregulates PD-L1 expression via the inactivation of GSK3β, contributing to immune evasion and creating a therapeutic window for ICIs (25). These immunomodulatory effects are further amplified in HRD tumor models, where the combination of PARPi and anti-PD-1/PD-L1 antibodies provides superior cytotoxic T-cell infiltration and tumor regression compared to monotherapy (26). Collectively, these findings provide a

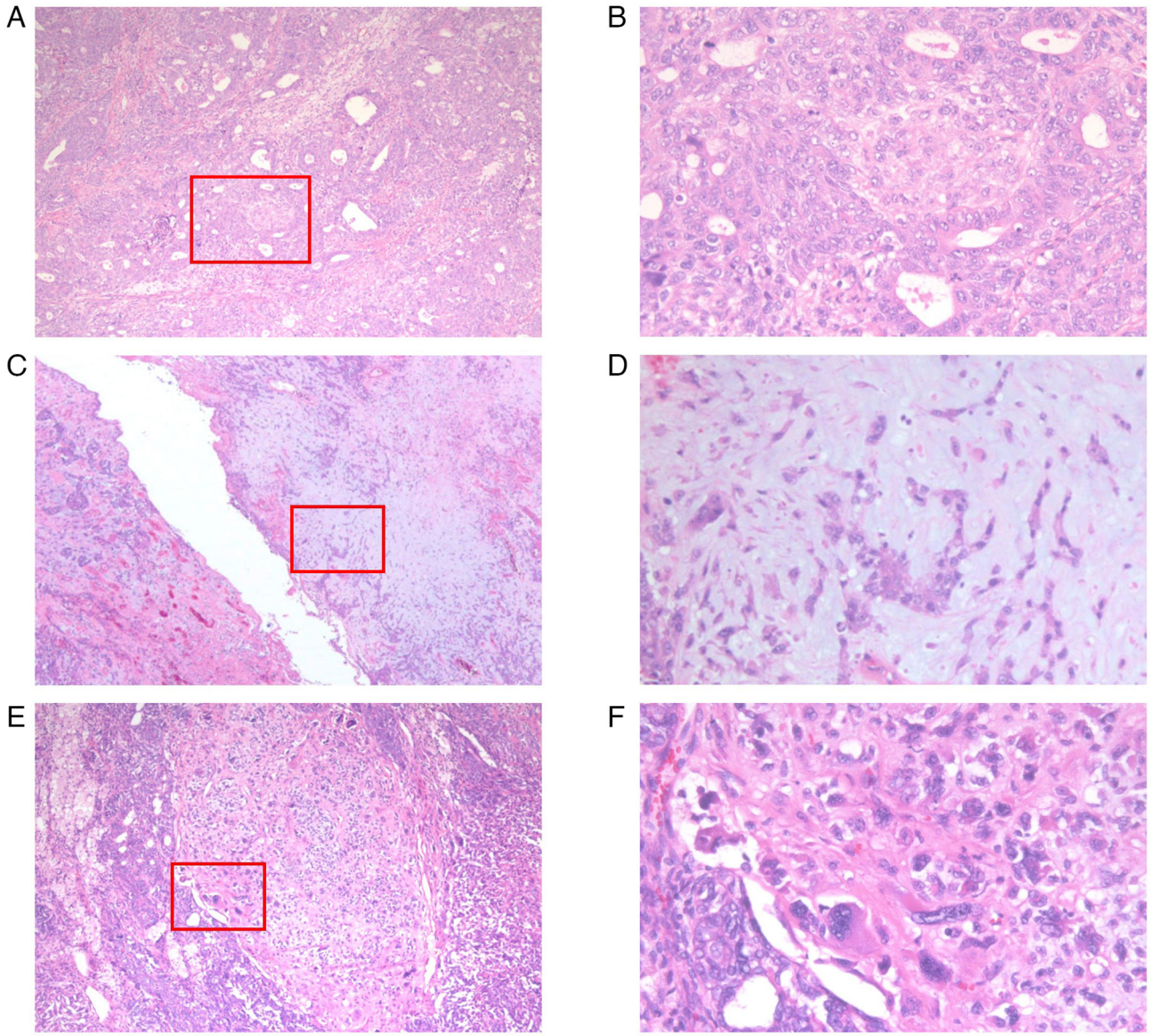

Figure 4. Histopathological features of the uterine tumor. (A) H&E staining showing poorly differentiated epithelial tumor cells (magnification, x100). (B) Higher magnification view of the boxed area in (A) demonstrating numerous mitotic figures, consistent with a carcinomatous component (magnification, x400). (C) H&E staining showing a sarcomatous component embedded in a chondromyxoid stroma (magnification, x100). (D) Higher magnification view of the boxed area in (C) revealing polygonal tumor cells consistent with chondroid differentiation (magnification, x400). (E) H&E staining showing another sarcomatous component (magnification, x100). (F) Higher magnification view of the boxed area in (E) demonstrating bundles of polygonal tumor cells with fibrillar eosinophilic cytoplasm, indicating rhabdomyosarcomatous differentiation (magnification, x400).

strong biological rationale for combined PARPi-ICI therapy in HRD tumors. Combination ICIs and PARPi are garnering increased attention in gynecological oncology. The phase III DUOE trial included patients with advanced or recurrent endometrial cancer, including UCS. The trial found that adding durvalumab to carboplatin and paclitaxel treatment, and following with maintenance durvalumab and olaparib, significantly improved progression-free survival (PFS). Specifically, the PFS hazard ratios (HRs) were 0.71 (durvalumab arm) and 0.55 (durvalumab and olaparib arm) versus chemotherapy alone (27). In a subgroup analysis of tumors harboring homologous recombination repair mutations, the PFS HR was 0.30 in the durvalumab and olaparib arm compared to chemotherapy alone (27). This provides compelling grounds for applying this combination strategy to HRD-positive UCS.

Compared with our case, Wan *et al* (28) reported a 58-year-old postmenopausal UCS with bilateral benign ovarian Brenner tumors showing diffuse mutant-type p53 on IHC; the report focuses on pathology and does not include TCGA/ProMisE molecular stratification, HRD status, germline testing, or systemic-therapy outcomes. By contrast, our case demonstrates the successful application of targeted and immunological therapy guided by precise molecular characterization in a young patient with UCS. Comprehensive genomic profiling (*TP53* mutation, *PIK3CA* alteration, *BRCA1* frameshift, and HRD positivity) informed a personalized treatment plan combining platinum-based chemotherapy, immune checkpoint blockade, and PARP inhibition. This resulted in complete and durable remission. Our findings support the use of

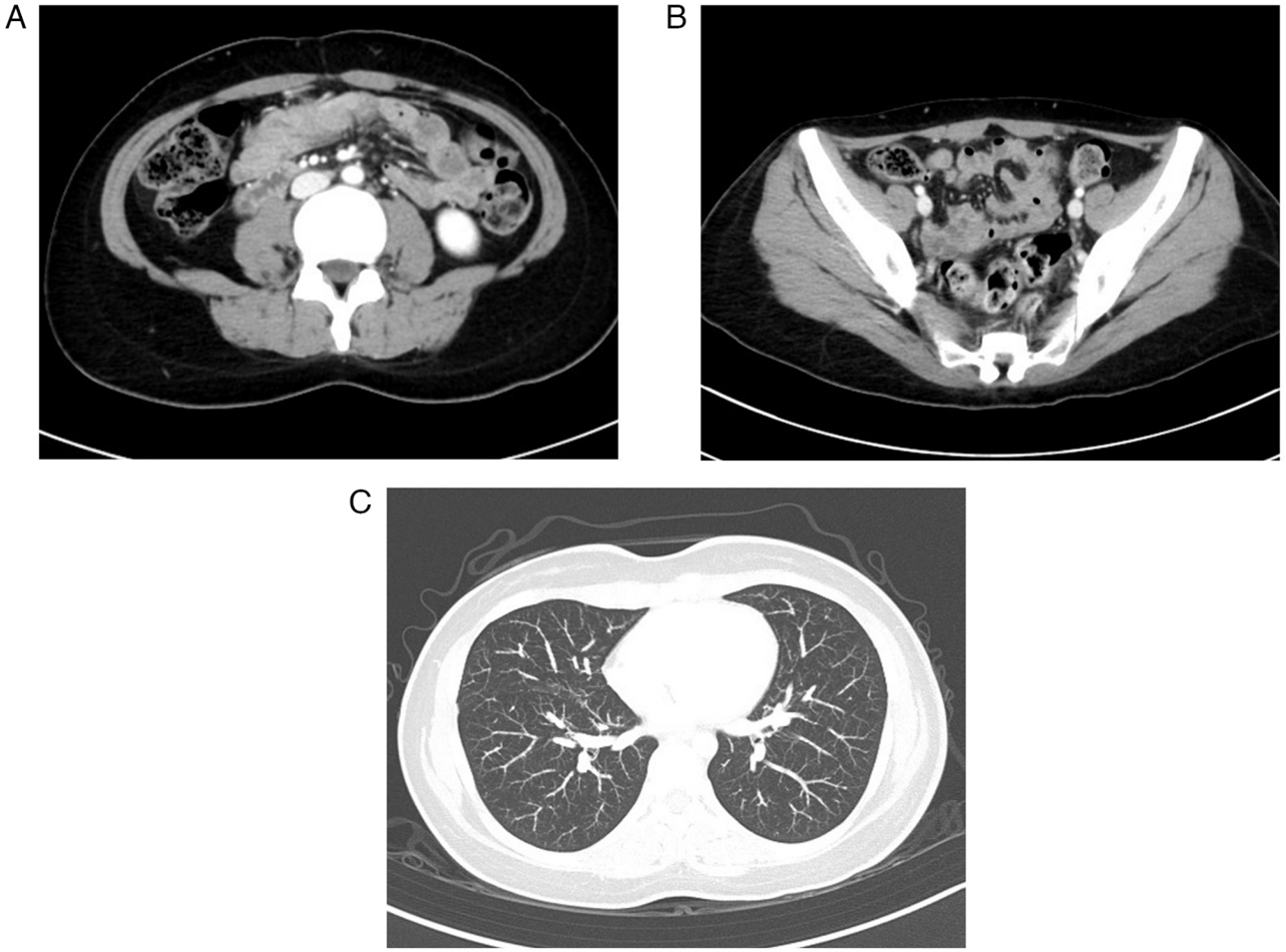

Figure 5. Treatment response on computed tomography. (A) Resolution of the previously enlarged para-aortic lymph nodes. (B) Resolution of the previously enlarged pelvic lymph nodes. (C) Disappearance of multiple pulmonary nodules. These findings were consistent with a complete response.

routine genomic profiling, including HRD testing, in UCS. Clinical trials targeting HRD-positive, MMR-proficient UCS are needed to further investigate treatment strategies such as that described herein. Such strategies may transform the prognosis for this aggressive and historically intractable cancer subtype.

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Availability of data and materials

The raw next-generation sequencing data generated in the present study may be found in the Japanese Genotype-Phenotype Archive, hosted by the DNA Data Bank of Japan, under accession number JGAS000883 (dataset: JGAD001027) or at the following URL: https://ddbj.nig.ac.jp/search/entry/jga-dataset/JGAD001027. All other data generated in the present study may be requested from the corresponding author.

Authors' contributions

YI, AK, YS, TI, JM, MI and ST contributed to study conception and design. YI and AK prepared the materials and collected and analyzed the data. YI drafted the manuscript. All authors reviewed and edited the manuscript. YI and ST confirm the authenticity of all the raw data. All authors have read and approved the final version of the manuscript.

Ethics approval and consent to participate

This case report was conducted in accordance with the tenets of the 1964 Declaration of Helsinki and its later revisions. This case report was approved by the Ethics Committee of Dokkyo Medical University Saitama Medical Center (approval no. 25094; Koshigaya, Japan).

Patient consent for publication

Written informed consent was obtained from the patient for the publication of their images and data.

Competing interests

The authors declare that they have no competing interests.

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