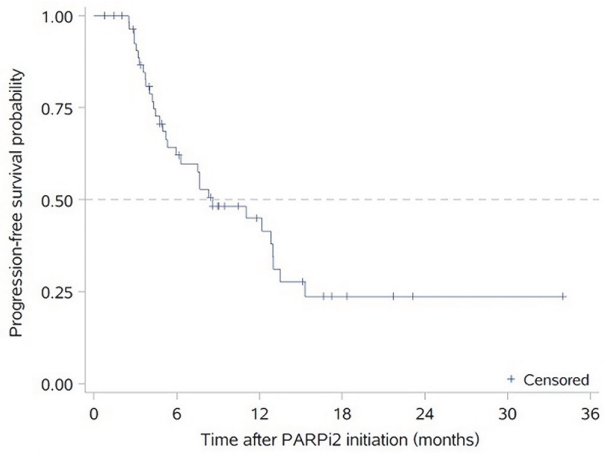
Abstract PR064/#206 Table 1

Patients' Characteristic	Total (N = 70) N (%)
Age, years	
Median (Q1-Q3)	58 (53-63.75)
***BRCA* mutation status, *n* (%)**	
Wild-type or unknown	43 (61.4)
BRCA1/2 mutated	27 (38.6)
Histology, *n* (%)	
High-grade serous carcinoma	68 (97.1)
Endometrioid	1 (1.4)
Others	1 (1.4)
Name of PARPi1	
Olaparib	50 (71.4)
Niraparib	20 (28.6)
Treatment phase of PARPi1	
Neoadjuvant	1 (1.4)
Maintenance therapy	67 (95.7)
Salvage treatment	2 (2.9)
PARPi1 treatment outcomes	
Disease progression	61 (87.2)
Adverse events	5 (7.1)
Others	4 (5.7)
Switch to other PARPi or not after PARPi1	
Switch	48 (68.6)
No switch	22 (31.4)
Treatment phase of PARPi2	
Maintenance therapy	57 (81.4)
Salvage treatment	13 (18.6)
Lines of PARPi2	
1	1 (1.4)
2	18 (25.7)
3	30 (42.9)
≥4	21 (30.0)
Treatment pattern of PARPi rechallenge	
Maintenance after maintenance	56 (80.0)
Treatment after maintenance	11 (15.7)
Treatment after treatment	2 (2.9)
Maintenance after neoadjuvant	1 (1.4)
Name of PARPi2	
Olaparib	23 (32.8)
Niraparib	42 (60.0)
Fuzoloparib	3 (4.3)
Pamiparib	2 (2.9)
PARPi2 treatment outcomes	
Still on treatment	24 (34.3)
Disease progression	43 (61.4)
Adverse events	3 (4.3)



Abstract PR064/#206 Figure 1 Kaplan-Meier plot of progression-free survival of patients receiving PARPi2 as maintenance therapy after PARPi1 maintenance therapy (N=56)

Results Seventy patients were included, and the median follow-up time was 13.0 months. Fifty-six (80%, 56/70) patients received PARPi as maintenance after maintenance therapy (table 1). The median PFS (mPFS) was 10.6 months (95% confidence interval [CI], 7.1–12.0) with first PARPi (PARPi1) and 8.6 months (95% CI, 5.3–13.0) with PARPi retreatment (PARPi2) (figure 1). 32.1%(18/56) patients were BRCA1/2 mutated, the PFS were not significantly different from BRCA wild-type or unknown patients (BRCAm vs. BRCAwt or unknown, HR=0.997 [95%CI: 0.480–2.072], P=0.9935). 87.5% (39/56) of patients switched to other PARPi when rechallenging. Patients switched to other PARPi rechallenging had numerically longer mPFS compared with those didn't switch (mPFS: 8.6 vs. 7.7 months; HR=0.820 [95%CI: 0.394–1.707], P=0.5958). Overall, 4.3% (3/70) discontinued PARPi2 due to adverse events, most commonly due to hematologic adverse events.

Conclusion/Implications Our study is the first multicenter real-world study to evaluate the rechallenge of PARPi in ovarian cancer patients in China. There is a pressing need to identify the biomarkers except BRCA to select appropriate patients for PARPi rechallenge.

PR065/#611 METASTATIC PATTERN OF OVARIAN CANCER DELINEATED BY TRACING THE EVOLUTION OF MITOCHONDRIAL DNA MUTATIONS

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Introduction Ovarian cancer (OC) is the most lethal gynecologic tumor and is characterized by a high rate of metastasis. Challenges in accurately delineating the metastatic pattern have greatly restricted the improvement of treatment in OC patients.

Methods We applied multiregional sampling and high-depth mitochondrial DNA (mtDNA) sequencing to determine the metastatic patterns in advanced-stage OC patients. Somatic mtDNA mutations were profiled from a total of 195 primary and 200 metastatic tumor tissue samples from 35 OC patients.

Results Our results revealed remarkable sample-level and patient-level heterogeneity. In addition, distinct mtDNA mutational patterns were observed between primary and metastatic OC tissues. Further analysis identified the different mutational spectra between shared and private mutations among primary and metastatic OC tissues. Analysis of the clonality index calculated based on mtDNA mutations supported a monoclonal tumor origin in 14 of 16 patients with bilateral ovarian cancers. Notably, mtDNA-based spatial phylogenetic analysis revealed distinct patterns of OC metastasis, in which a linear metastatic pattern exhibited a low degree of mtDNA mutation heterogeneity and a short evolutionary distance, whereas a parallel metastatic pattern showed the opposite trend. Moreover, a mtDNA-based tumor evolutionary score (MTEs) related to different metastatic patterns was defined. Our data showed that patients with different MTESs responded differently to combined debulking surgery and chemotherapy. Finally, we observed that tumor-derived mtDNA mutations were more likely to be detected in ascitic fluid than in plasma samples.

Conclusion/Implications Our study presents an explicit view of the OC metastatic pattern.