nature cancer

Article

https://doi.org/10.1038/s43018-025-01085-z

Suvemcitug plus chemotherapy in women with platinum-resistant recurrent ovarian cancer: the SCORES randomized, double-blinded, phase 3 trial

Received: 9 April 2025

Accepted: 30 October 2025

Published online: 9 January 2026

Check for updates

A list of authors and their affiliations appears at the end of the paper

In the SCORES study (NCT04908787), women with ovarian cancer that progressed within 6 months after completing platinum-based therapy were randomized (2:1) to receive suvemcitug (1.5 mg kg^{-1}), an antibody to vascular endothelial growth factor or placebo every 2 weeks, with chemotherapy (paclitaxel, topotecan or PEGylated liposomal doxorubicin). The primary endpoint was progression-free survival (PFS). The key secondary endpoint was overall survival (OS). Other secondary endpoints included objective response rate, disease control rate, duration of response, quality of life, safety, pharmacokinetics and antidrug antibodies. Between June 5, 2021 and October 11, 2024, 421 participants were randomized (49.4% and 49.4% previously exposed to antiangiogenic agents and poly(ADP-ribose) polymerase inhibitors, respectively). Median PFS was 5.5 and 2.7 months in the suvemcitug and placebo arms, respectively (hazard ratio: 0.46, 95% confidence interval (CI): 0.35–0.60, $P < 0.001$), meeting the primary endpoint. Median OS was 15.3 versus 14.0 months, respectively (hazard ratio: 0.77, 95% CI: 0.60–0.99, $P = 0.03$). Decreased neutrophil count and decreased white blood cell count were the most common grade ≥3 treatment-emergent adverse events (TEAEs) in the suvemcitug arm. No suvemcitug-related grade 5 TEAE occurred. In conclusion, the addition of suvemcitug to chemotherapy significantly improved PFS and OS, with tolerable toxicities.

Ovarian cancer (OC) is the most lethal gynecological malignancy, with 324,938 new cases and 206,834 deaths in 2022 globally[1]. Platinum-based chemotherapy plus paclitaxel with or without bevacizumab, recently with maintenance poly(ADP-ribose) polymerase (PARP) inhibitors and/or bevacizumab, is the primary treatment option for advanced OC[2–6]. Despite a 75–80% response rate with first-line therapy, relapse occurs within 18 months in the majority of persons[7,8]. Standard non-platinum chemotherapy for platinum-resistant OC has limited efficacy, with ≤15% of persons showing an objective response and a median progression-free survival (PFS) between 3 and 4 months[7,9,10].

Bevacizumab, a monoclonal antibody to vascular endothelial growth factor (VEGF), has demonstrated efficacy for both platinum-sensitive and resistant OC[11,12]. In the AURELIA trial, bevacizumab, when added to chemotherapy, extended the median PFS by 3.3 months in participants with platinum-resistant OC[13]. On the basis of these findings, bevacizumab is recommended for the treatment of persons with platinum-resistant OC who have received ≤2 prior lines of cytotoxic therapy[14]. The efficacy of bevacizumab, however, needs to be reexamined as persons who received PARP inhibitors were not included. Furthermore, the AURELIA trial only included participants who received ≤2 prior lines of cytotoxic therapy and only 7.2% of the participants received prior antiangiogenic therapy.

Antiangiogenic agents other than bevacizumab, including ofranergene obadenovec, failed to improve objective response and survival

e-mail: 13854158117@163.com; wulingying@csco.org.cn

https://doi.org/10.1038/s43018-025-01085-z

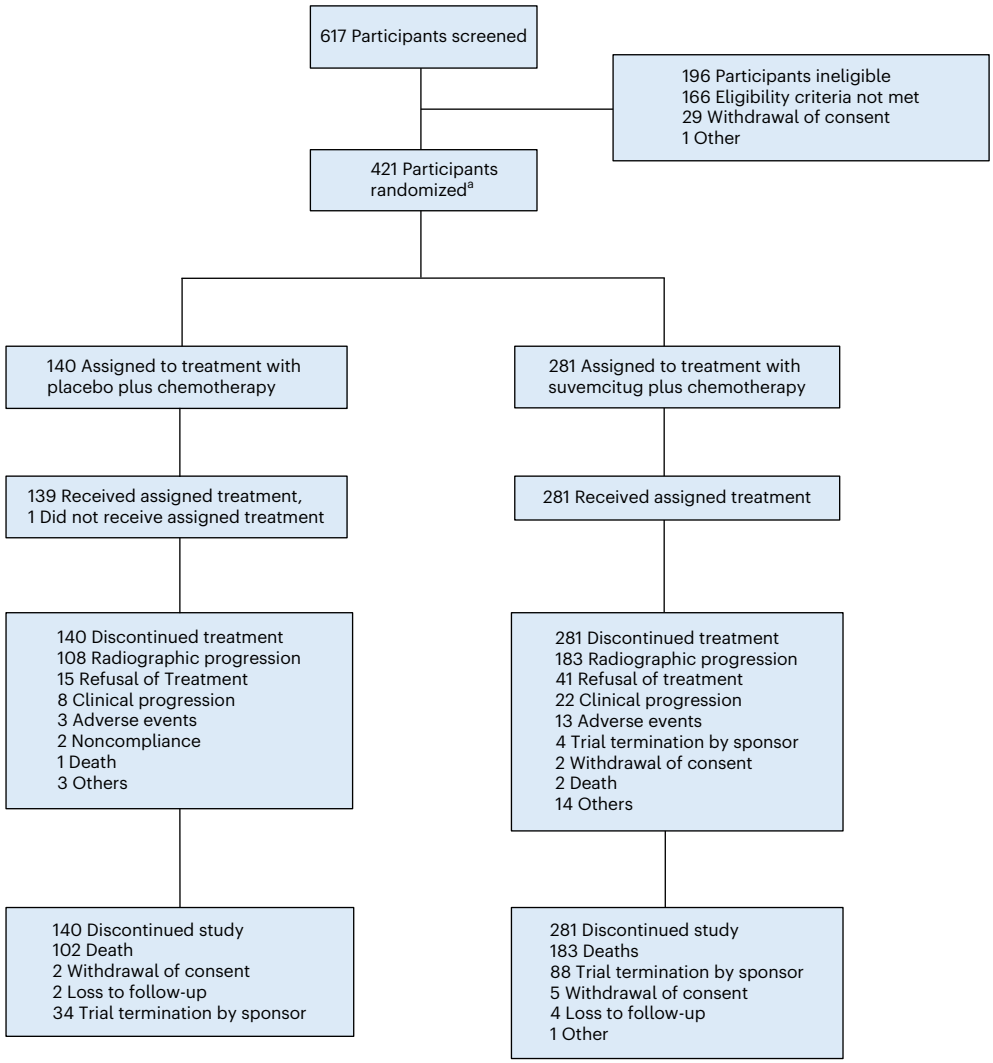


Fig. 1 | Participant flow in the SCORES trial. [a]Participants were randomized in a 2:1 ratio and stratified according to platinum-refractory status (yes versus no), number of prior systemic therapies (one versus two), chemotherapeutic agent (paclitaxel versus PEGylated liposomal doxorubicin versus topotecan) and prior antiangiogenic therapy (yes versus no). More information is provided in the Table 1 footnotes. For the safety analysis, one participant who was randomized but did not receive planned treatment was excluded.

in persons with platinum-resistant OC when added to chemotherapy[8,15]. Novel safe and effective antiangiogenic drugs are urgently needed for persons with platinum-resistant OC.

Suvemcitug (BD0801), a humanized rabbit monoclonal IgG1 (κ) anti-VEGF antibody, selectively binds to and prevents VEGF-A from binding to VEGF receptors 1 and 2 (VEGFR1 and VEGFR2)[16,17]. VEGF-A is secreted in multiple forms by alternative splicing[18]; these include $VEGF_{121}$, $VEGF_{165}$ and $VEGF_{189}$. Suvemcitug and bevacizumab have comparable binding affinity for $VEGF_{121}$ and $VEGF_{189}$ (ref. 19). Suvemcitug and bevacizumab bind to different epitopes of human $VEGF_{165}$ (ref. 17) but have comparable affinity for $VEGF_{165}$ (K_d: 1.2×10^{-11} M versus 1.0×10^{-11} M; half-maximal effective concentration: 7.0 ng ml^{-1} versus 5.8 ng ml^{-1}). Suvemcitug also binds to $VEGF_{164}$ with an affinity similar to $VEGF_{165}$, whereas bevacizumab does not bind to $VEGF_{164}$. In comparison to bevacizumab, suvemcitug has a lower half-maximal inhibitory concentration for inhibition of VEGF binding to VEGFR1 (21.0 ng ml^{-1} versus 6760 ng ml^{-1}) and VEGFR2 (275.4 ng ml^{-1} versus 1451 ng ml^{-1})[19]. Early-stage trials of suvemcitug have shown promising antitumor activities when used in combination with chemotherapy for previously treated advanced solid tumors[19]. A phase 1b trial of suvemcitug plus paclitaxel or topotecan reported objective response in nine of 29 participants (31%) with platinum-resistant OC and a median PFS of 5.4 months[20]. In these trials, the safety profile of suvemcitug was manageable without unexpected toxicities.

We conducted a phase 3 trial (SCORES) to examine the efficacy and safety of suvemcitug plus chemotherapy in persons with platinum-refractory or resistant OC.

Results

Participants

This randomized, double-blind, placebo-controlled, phase 3 trial (SCORES) was conducted at 55 tertiary-care centers in China between June 5, 2021 and October 11, 2024. Randomization was stratified according to platinum-refractory status (yes versus no), number of prior systemic therapies (one versus two), chemotherapeutic agent (paclitaxel versus PEGylated liposomal doxorubicin versus topotecan) and prior antiangiogenic therapy (yes versus no). A total of 617 women (aged ≥18 years) with histologically confirmed epithelial ovarian, fallopian tube or primary peritoneal cancer were screened for eligibility. Participants were required to have platinum-refractory or resistant disease (disease progression within 6 months of platinum therapy), at least one measurable lesion per the response evaluation criteria in solid tumors (RECIST; v.1.1), an Eastern Cooperative Oncology Group (ECOG) performance status of 0–1 and adequate hematologic and organ function. In total,

https://doi.org/10.1038/s43018-025-01085-z

Table 1 | Demographic and baseline characteristics of the participants in the full analysis set

	Suvemcitug plus chemotherapy (*n*=281)	Placebo plus chemotherapy (*n*=140)	*P* value[#]
Age, years			0.9999
Median age (range)	56.0 (36–76)	55.0 (34–79)	
>65	38 (13.5)	20 (14.3)	
Ethnic groups			0.873
Han Chinese	266 (94.7)	132 (94.3)	
Other	15 (5.3)	8 (5.7)	
Origin of cancer			0.014
Epithelial OC	263 (93.6)	120 (85.7)	
Fallopian tube cancer	15 (5.3)	19 (13.6)	
Primary peritoneal cancer	3 (1.1)	1 (0.7)	
ECOG performance status score[†]			0.063
0	106 (37.7)	40 (28.6)	
1	175 (62.3)	100 (71.4)	
Histologic diagnosis			0.176
High-grade serous adenocarcinoma	278 (98.9)	136 (97.1)	
Endometrioid carcinoma	3 (1.1)	4 (2.9)	
Sum of the target lesion diameter, median (range), mm	41.20 (10.0–322.8)	43.05 (10.7–229.1)	0.495
FIGO stage			0.901
I	7 (2.5)	4 (2.9)	
II	13 (4.6)	9 (6.4)	
III	195 (69.4)	95 (67.9)	
IV	62 (22.1)	31 (22.1)	
Unknown	4 (1.4)	1 (0.7)	
Confirmed distant metastasis			
Peritoneum	160 (56.9)	79 (56.4)	0.923
Lymph node	181 (64.4)	78 (55.7)	0.084
Pelvic cavity	98 (34.9)	53 (37.9)	0.548
Liver	86 (30.6)	39 (27.9)	0.561
Lungs	22 (7.8)	16 (11.4)	0.225
Spleen	26 (9.3)	13 (9.3)	0.991
Bone	9 (3.2)	4 (2.9)	0.847
Kidney	0	2 (1.4)	0.045
Other	151 (53.7)	81 (57.9)	0.423
Previous lines of systemic therapy			0.812
1	87 (31.0)	40 (28.6)	
2	106 (37.7)	52 (37.1)	
3	59 (21.0)	35 (25.0)	
≥4	29 (10.3)	13 (9.3)	
Platinum-free interval, months			0.369
<1	29 (10.3)	21 (15.0)	
1–3	81 (28.8)	37 (26.4)	
≥3	171 (60.9)	82 (58.6)	
Previous chemotherapy			
Platinum-based drugs	281 (100)	140 (100)	-
Taxanes	279 (99.3)	138 (98.6)	0.475
Anthracyclines	71 (25.3)	23 (16.4)	0.040
Topoisomerase 1 inhibitors	4 (1.4)	2 (1.4)	0.997
Other	49 (17.4)	24 (17.1)	0.940
Chemotherapeutic agents			0.998
Paclitaxel	124 (44.1)	62 (44.3)	
PEGylated liposomal doxorubicin	88 (31.3)	44 (31.4)	
Topotecan	69 (24.6)	34 (24.3)	
Previous antiangiogenic therapy			0.972
Yes	139 (49.5)	69 (49.3)	
No	142 (50.5)	71 (50.7)	
Previous bevacizumab therapy	123 (43.8)	61 (43.6)	0.969
Previous PARP inhibitor therapy	138 (49.1)	70 (50.0)	0.863
Ascites			0.981
Yes	92 (32.7)	46 (32.9)	
No	189 (67.3)	94 (67.1)	
Pleural effusion			0.634
Yes	26 (9.3)	11 (7.9)	
No	255 (90.7)	129 (92.1)	
CA-125			0.597
≤2×ULN	36 (12.8)	14 (10.0)	
2×ULN–1,000	178 (63.3)	95 (67.9)	
>1,000	67 (23.8)	31 (22.1)	
Platinum-refractory			0.713
No	256 (91.1)	126 (90.0)	
Number of prior systemic therapies[‡]			0.807
1	190 (67.6)	93 (66.4)	
2	91 (32.4)	47 (33.6)	

Data are numbers (%) unless otherwise specified. Percentages may not total 100 because of rounding. [#]Two-sided chi-square test; no adjustment for multiple comparisons. [†]ECOG performance status scores are on a scale of 0–5, with higher scores indicating greater disability. [‡]A value of 1 represents no systemic therapy after platinum resistance; a value of 2 indicates systemic therapy after platinum resistance.

421 eligible participants were randomized (2:1) to receive suvemcitug (1.5 mg kg^{-1} infused on days 1 and 15 of each 4-week cycle) plus chemotherapy (suvemcitug arm; $n = 281$) or placebo plus chemotherapy (placebo arm; $n = 140$) (Fig. 1). Most participants (414, 98.3%) had high-grade serous adenocarcinoma and 383 (91.0%) had International Federation of Gynecology and Obstetrics (FIGO) stage III or IV disease. The majority of the participants (294, 69.8%) received ≥2 prior lines of systemic therapy and 208 (49.4%) had previous exposure to an antiangiogenic agent (bevacizumab: 184, 43.7%) and a PARP inhibitor. Demographic and baseline characteristics of the participants are shown in Table 1.

https://doi.org/10.1038/s43018-025-01085-z

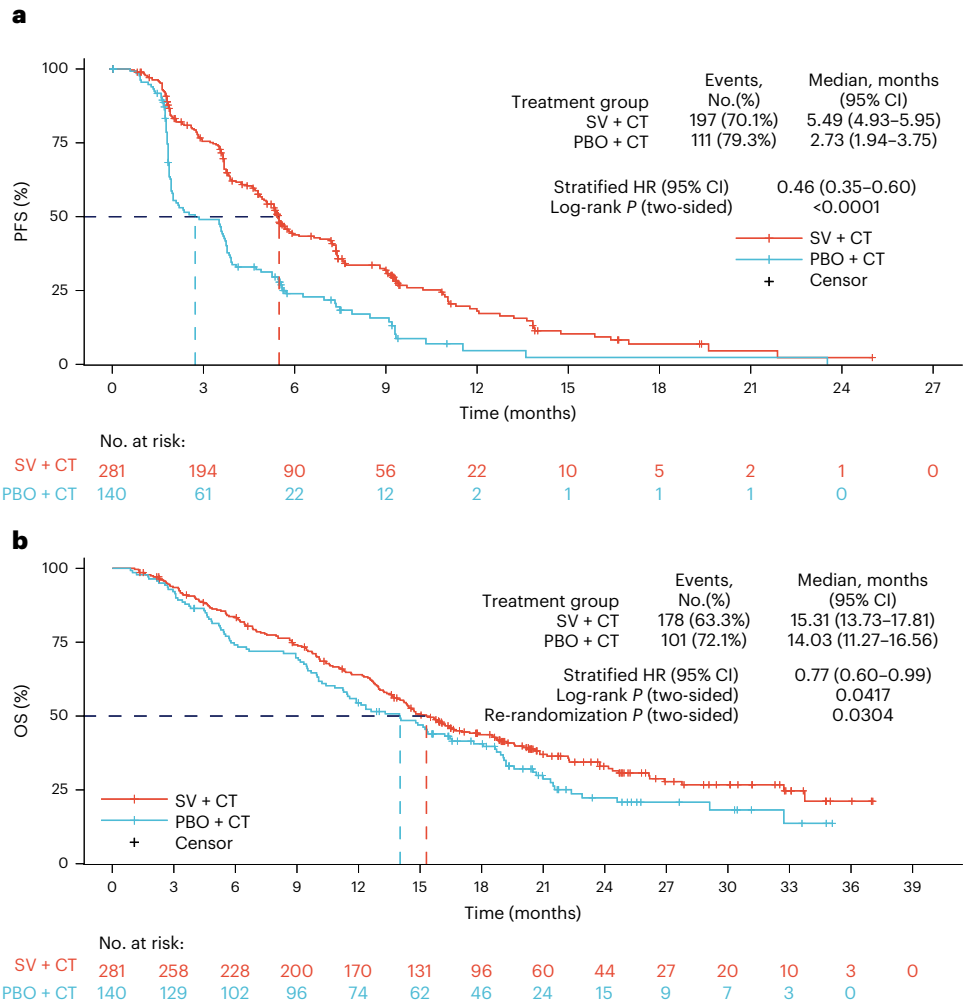


Fig. 2 | Survival outcomes. a, Kaplan–Meier curve of PFS at the first efficacy analysis in the full analysis set assessed by BIRC per RECIST (v.1.1). Number of participants: 281 and 140 in the suvemcitug and placebo arms, respectively. $P < 0.0001$. **b**, Kaplan–Meier curve of OS in the full analysis set at the final analysis. Number of participants: 281 and 140 in the suvemcitug and placebo arms, respectively. $P = 0.03$. PBO + CT, placebo plus chemotherapy; SV + CT, suvemcitug plus chemotherapy.

At the final analysis (October 11, 2024), all participants discontinued the treatment, mostly for disease progression (73.0% and 82.9% in the suvemcitug and placebo arms, respectively) (Fig. 1).

Efficacy

The primary endpoint was PFS, as assessed by a blinded independent review committee (BIRC) per RECIST (v.1.1) when 308 events had occurred. At the data cutoff (December 8, 2023), the median follow-up duration was 14.4 and 14.3 months in the suvemcitug and placebo arms, respectively. The median PFS was 5.5 months in the suvemcitug arm (95% confidence interval (CI): 4.9–6.0) versus 2.7 months in the placebo arm (95% CI: 1.9–3.8; stratified hazard ratio (HR): 0.46, 95% CI: 0.35–0.60, $P < 0.001$) (Fig. 2a and Supplementary Table 1).

At the final analysis (October 11, 2024), the median follow-up duration was 23.7 and 23.4 months in the suvemcitug and placebo arms, respectively. A total of 178 and 101 overall survival (OS) events occurred in the suvemcitug and placebo arms, respectively. The median OS was 15.3 months (95% CI: 13.7–17.8) in the suvemcitug arm versus 14.0 months (95% CI: 11.3–16.6) in the placebo arm (stratified HR: 0.77, 95% CI: 0.60–0.99, $P = 0.03$) (Fig. 2b).

The results of subgroup analyses of BIRC-assessed PFS consistently favored the suvemcitug arm across all prespecified and unplanned post hoc analysis for previous PARP inhibitor exposure (Fig. 3). In the paclitaxel cohort, suvemcitug led to a 2.2-month extension in the median PFS (6.0 months versus 3.7 months in the placebo arm; HR: 0.45, 95% CI: 0.31–0.65). In the topotecan cohort, the median PFS was 3.9 months in the suvemcitug arm versus 2.0 months in the placebo arm (HR: 0.37, 95% CI: 0.22–0.62). In the doxorubicin cohort, the median PFS was 5.3 months in the suvemcitug arm versus 3.7 months in the placebo arm (HR: 0.69, 95% CI: 0.45–1.05). Notably, suvemcitug increased the median PFS regardless of previous exposure to PARP inhibitors (no, HR: 0.55, 95% CI: 0.40–0.77; yes, HR: 0.49, 95% CI: 0.35–0.69) (Fig. 3 and Extended Data Fig. 1a,b) and regardless of previous exposure to antiangiogenic agents (no, HR: 0.59, 95% CI: 0.42–0.83; yes, HR: 0.45, 95% CI: 0.33–0.63) (Fig. 3).

Supplementary analysis that was undertaken to account for subsequent antitumor therapy as intercurrent events showed that suvemcitug led to a 10.4-month extension of median OS compared to placebo (22.3 months, 95% CI: 13.2–NE (not evaluable) versus 11.9 months, 95% CI: 9.2–22.9; stratified HR: 0.59, 95% CI: 0.39–0.90, $P = 0.01$) (Extended Data Fig. 2a).

Subgroup analyses of OS showed significant reduction in the risk of death across in the suvemcitug arm across almost all prespecified and unplanned post hoc analysis for previous PARP inhibitor exposure (Extended Data Fig. 2b). In participants who were previously treated with anti-VEGF agents, suvemcitug led to a 27% reduction in the risk of death compared to placebo (HR: 0.73, 95% CI: 0.53–1.01). Similar

https://doi.org/10.1038/s43018-025-01085-z

Subgroup	Events/N SV + CT	Events/N PBO + CT	Hazard ratio 95% CI	Median, months SV + CT	Median, months PBO + CT	Hazard ratio 95% CI
Overall	197/281	111/140		5.49	2.73	0.53 (0.42,0.67)
Age						
≤65	169/243	95/120		5.39	2.53	0.51 (0.39,0.66)
>65	28/38	16/20		5.65	3.78	0.62 (0.33,1.16)
Baseline ECOG						
0	72/106	33/40		5.42	2.73	0.45 (0.29,0.69)
1	125/175	78/100		5.49	2.53	0.57 (0.42,0.76)
Ascites						
Y	68/91	34/45		4.93	2.00	0.54 (0.36,0.83)
N	129/190	77/95		5.59	3.61	0.51 (0.38,0.68)
Sum of diameters for all target lesions						
<41.70	93/143	51/67		5.68	3.52	0.45 (0.32,0.65)
≥41.70	104/138	60/73		5.42	2.53	0.60 (0.43,0.82)
Number of lines of previous antitumor regimens						
1	58/88	32/40		5.91	3.84	0.56 (0.36,0.87)
2	77/105	41/52		5.39	3.52	0.42 (0.28,0.63)
≥3	62/88	38/48		4.80	2.00	0.58 (0.39,0.88)
Platinum-free interval (month)						
<3	85/109	48/59		4.63	2.00	0.61 (0.42,0.87)
≥3	112/172	63/81		6.83	3.65	0.47 (0.34,0.64)
Number of previous systemic therapies						
1	126/190	75/94		5.78	3.55	0.50 (0.38,0.67)
2	71/91	36/46		3.88	2.33	0.57 (0.38,0.86)
Platinum-refractory ovarian cancer patients						
Y	21/27	11/15		4.01	3.52	0.70 (0.33,1.48)
N	176/254	100/125		5.59	2.53	0.51 (0.40,0.66)
Chemotherapy						
Paclitaxel	87/124	51/62		5.95	3.71	0.45 (0.31,0.65)
Topotecan	53/69	25/34		3.88	2.00	0.37 (0.22,0.62)
Doxorubicin	57/88	35/44		5.32	3.68	0.69 (0.45,1.05)
Received antiangiogenic therapy						
Y	98/138	59/70		4.93	2.00	0.45 (0.33,0.63)
N	99/143	52/70		5.95	3.75	0.59 (0.42,0.83)
Received PARP inhibitors						
Y	94/136	56/70		5.32	2.00	0.49 (0.35,0.69)
N	103/145	55/70		5.78	3.58	0.55 (0.40,0.77)

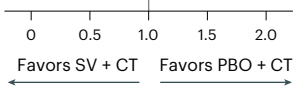


Fig. 3 | Forest plots for PFS per BIRC. Shown are the results of prespecified and unplanned post hoc analysis for previous received PARP (yes, no) subgroup analyses in the full analysis set. The HR for progression or death was based on Cox proportional-hazards regression analysis for all randomized participants. No stratification was used in the forest plots.

findings were observed in participants previously exposed to PARP inhibitors (HR: 0.82, 95% CI: 0.58–1.16).

Objective response at the first analysis was confirmed in 73 of 281 participants (26.0%; 95% CI: 21.0–31.5%) by BIRC in the suvemcitug arm versus 17 of 140 participants (12.1%) in the placebo arm (95% CI: 7.2–18.7%, $P = 0.001$) (Fig. 4). The median duration of response (DOR) was 8.8 months (95% CI: 6.1–10.9) versus 6.1 months (95% CI 4.2–NE). Disease control per BIRC was attained in 215 of 281 participants (76.5%; 95% CI: 71.1–81.3%) in the suvemcitug arm versus 69 of 140 participants (49.3%; 95% CI: 40.7–57.9%) in the placebo arm ($P < 0.001$) (Supplementary Table 1).

Safety

At the final analysis, the median duration of treatment was 18.9 weeks and 9.1 weeks for suvemcitug and placebo, respectively. One participant in the placebo arm was not treated and excluded from safety analysis. The mean relative dose intensities of paclitaxel, PEGylated liposomal doxorubicin or topotecan were slightly lower in the suvemcitug arm than the placebo arm (Supplementary Table 2).

Treatment-emergent adverse effects (TEAEs) of any grade occurred in 281 participants (100%; grade ≥3: 234 participants, 83.3%) in the suvemcitug arm and 137 participants (98.6%; grade ≥3: 92, 66.2%) in the placebo arm (Table 2). The most frequently reported grade ≥3 TEAEs (occurring in ≥15% of the participants in either arm) included neutrophil count decreased (suvemcitug: 49.8%, 140/281 versus placebo: 41.0%, 57/139), white blood cell count decreased (suvemcitug: 35.9%, 101/281 versus placebo: 27.3%, 38/139), hypertension (suvemcitug: 18.9%, 53/281 versus placebo: 0.7%, 1/139) and anemia (suvemcitug: 16.7%, 47/281 versus placebo: 17.3%, 24/139) (Table 2). One participant (0.4%) in the suvemcitug arm had gastrointestinal perforation versus none in the placebo arm. TEAEs led to suvemcitug dose reduction in 26 participants (9.3%) and placebo dose reduction in none of the participants in the placebo arm. TEAEs led to suvemcitug treatment interruption in 232 participants (82.6%) and placebo treatment interruption in 86 participants (61.9%). Suvemcitug treatment was discontinued in 19 participants (6.8%) and placebo treatment was discontinued in three (2.2%) participants (Table 2).

AEs of any grade related to suvemcitug or placebo occurred in 267 (95.0%; grade ≥3: 199 participants, 70.8%) participants in the suvemcitug arm and 124 (89.2%; grade ≥3: 69, 49.6%) participants in the placebo arm (Supplementary Table 3). Serious AEs related to suvemcitug or placebo occurred in 63 participants (22.4%) in the suvemcitug arm and 22 participants (15.8%) in the placebo arm (Supplementary Table 4). No suvemcitug-related grade 5 TEAE occurred.

Participant-reported outcomes

The European Organization for Research and Treatment of Cancer (EORTC) questionnaires QLQ-OV28 and QLQ-C30 did not differ between the two arms (Extended Data Figs. 3 and 4).

Exploratory analyses

In total, 11 of 280 participants (3.9%) in the suvemcitug arm with samples at screening were positive for antidrug antibody (ADA) before treatment

https://doi.org/10.1038/s43018-025-01085-z

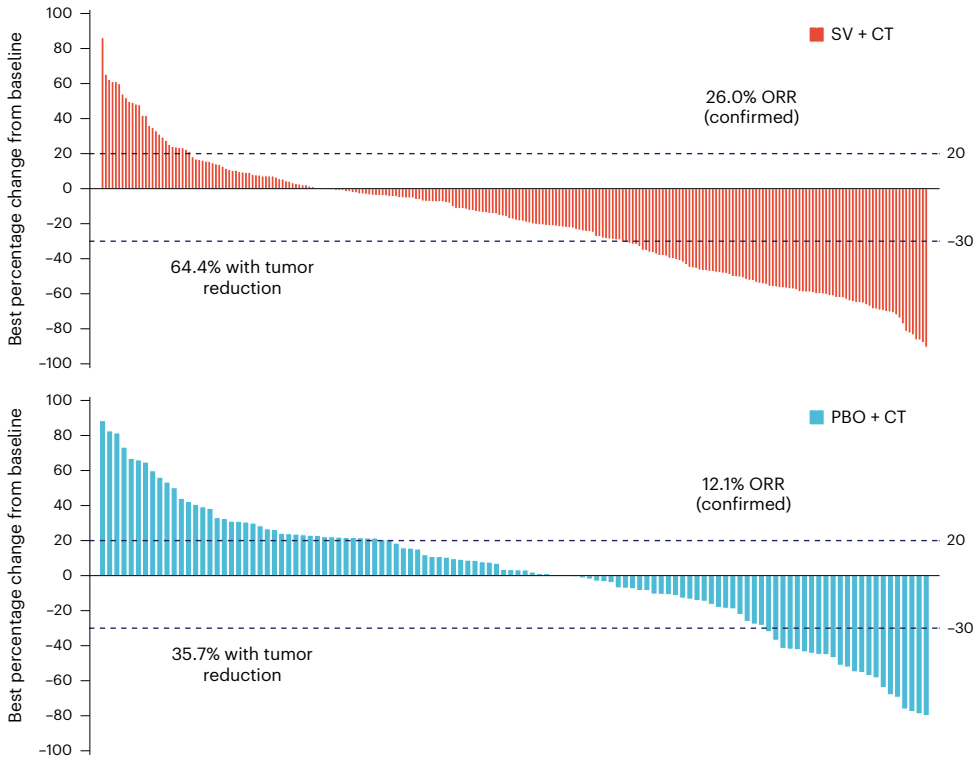


Fig. 4 | Treatment responses. Waterfall plots of the best percentage changes for the sum of target lesion diameters are shown for individual participants with platinum-refractory or resistant recurrent OC assessed by BIRC per RECIST (v.1.1). Number of participants: 281 and 140 in the suvemcitug and placebo arms, respectively. The lower dashed line indicates a 30% reduction and the upper dashed line represents a 20% increase in the target lesion size. The ORR was defined as the proportion of participants in the full analysis set with a complete response or a partial response.

initiation but none were positive for neutralizing ADA. In total, 38 of 277 participants (13.7%) were positive for treatment-emergent ADA and four (1.4%) were positive for neutralizing ADA. ADAs persisted for ≥16 weeks in 6.1% (17/277) participants.

Discussion

The SCORES trial met its primary endpoint, demonstrating that suvemcitug, when added to chemotherapy, increased the median PFS from 2.7 to 5.5 months, with a corresponding 54% reduction in the risk of progression or death. At data maturity, a significant OS benefit with suvemcitug was observed, with a 23% reduction in the risk of death.

This trial enrolled a broader population than the AURELIA trial, including participants who previously received bevacizumab and/or a PARP inhibitor[13,21]. In the AURELIA trial[13,21], 7.2% of the participants received prior antiangiogenic therapy compared to 49.4% in this trial (bevacizumab: 43.7%). Approximately one third (32.3%) of participants in this trial received ≥3 prior lines of systemic therapies, whereas the AURELIA trial excluded persons who received >2 prior lines of systemic therapies. These discrepancies may explain the shorter median PFS (2.7 months) in the placebo arm in this trial. The improvement in PFS by suvemcitug was also supported by the higher response rate and the longer DOR (8.8 months versus 6.1 months in the placebo arm).

Suvemcitug conferred broad PFS benefit across all subgroups in this trial, including participants with previous exposure to PARP inhibitors and/or antiangiogenic therapy. PARP inhibitors have become the standard of care for women with advanced OC, particularly for newly diagnosed persons with a *BRCA* mutation or with homologous recombination deficiency (HRD)-positive tumors[5,22,23]. The efficacy of antiangiogenic therapy for persons with platinum-resistant, recurrent OC who have been previously treated with a PARP inhibitor remains unclear. In this trial, approximately half of the participants (49.4%, 208/421) received prior PARP inhibitor therapy and the proportions of participants who previously received PARP inhibitors were well balanced in the two arms. In subgroup analyses, suvemcitug conferred a significant PFS benefit regardless of previous exposure to PARP inhibitors. Most notably, suvemcitug led to a 51% reduction in the risk of progression or death compared to placebo in participants with previous exposure to PARP inhibitor (HR: 0.49, 95% CI: 0.35–0.69) and a statistically nonsignificant trend of lower risk of death (HR: 0.82, 95% CI: 0.58–1.16), suggesting that suvemcitug could be offered as an effective treatment option in persons with platinum-resistant, recurrent OC who were previously exposed to PARP inhibitors. These findings are consistent with the association between longer PFS and bevacizumab plus chemotherapy (8.9 months versus 3.1 months alone, $P = 0.022$) in a retrospective study in persons with OC who received prior PARP inhibitor[24] and support the incorporation of suvemcitug into the therapeutic regimens for platinum-resistant recurrent OC, including those were previously treated with a PARP inhibitor.

The efficacy of antiangiogenic therapy in persons with platinum-resistant OC was established in the AURELIA trial of bevacizumab and in subsequent studies, including the TRIAS trial of sorafenib[13,25], the APPROVE trial of apatinib[26], a phase 3 trial of pazopanib[27] and a phase 2 study of anlotinib[28]. The phase 2 APPROVE trial demonstrated a significant improvement in PFS with apatinib combined with liposomal doxorubicin when compared to liposomal doxorubicin only[26]. In the TRIAS study[25], sorafenib showed a statistically significant and clinically meaningful improvement in PFS in persons with platinum-resistant OC when given orally in combination with topotecan and continued as maintenance therapy. With the increasing use of bevacizumab, however, there is a rising population of persons who have failed prior bevacizumab or anti-VEGF tyrosine kinase inhibitors. There is also a subset of persons who could not tolerate bevacizumab toxicities. In this trial, 43.7% and 49.4% of the participants received prior bevacizumab and antiangiogenic therapy, respectively.

https://doi.org/10.1038/s43018-025-01085-z

Table 2 | Incidence of TEAEs occurring in at least 10% of the participants in either arm of the safety population at the final analysis

	Suvemcitug plus chemotherapy (*n*=281)		Placebo plus chemotherapy (*n*=139)		*P* value*	
Preferred term	All grades	Grade ≥3	All grades	Grade ≥3	All grades	Grade ≥3
Any	281 (100)	234 (83.3)	137 (98.6)	92 (66.2)	0.044	<0.001
Neutrophil count decreased	238 (84.7)	140 (49.8)	94 (67.6)	57 (41.0)	<0.001	0.089
White blood cell count decreased	237 (84.3)	101 (35.9)	102 (73.4)	38 (27.3)	0.007	0.078
Anemia	201 (71.5)	47 (16.7)	111 (79.9)	24 (17.3)	0.066	0.890
Platelet count decreased	143 (50.9)	34 (12.1)	43 (30.9)	10 (7.2)	0.001	0.122
Proteinuria	112 (39.9)	11 (3.9)	19 (13.7)	0	<0.001	0.018
Asthenia	97 (34.5)	4 (1.4)	34 (24.5)	4 (2.9)	0.036	0.305
Alanine aminotransferase increased	83 (29.5)	1 (0.4)	24 (17.3)	0	0.007	0.481
Aspartate aminotransferase increased	81 (28.8)	0	26 (18.7)	1 (0.7)	0.025	0.155
Hypertension	79 (28.1)	53 (18.9)	4 (2.9)	1 (0.7)	<0.001	<0.001
Weight decreased	78 (27.8)	7 (2.5)	17 (12.2)	0	<0.001	0.061
Alopecia	72 (25.6)	0	30 (21.6)	0	0.364	-
Nausea	70 (24.9)	1 (0.4)	30 (21.6)	1 (0.7)	0.451	0.611
Vomiting	65 (23.1)	2 (0.7)	25 (18.0)	1 (0.7)	0.227	0.993
Decreased appetite	64 (22.8)	1 (0.4)	24 (17.3)	1 (0.7)	0.192	0.611
Constipation	62 (22.1)	0	25 (18.0)	1 (0.7)	0.332	-
Urinary tract infection	62 (22.1)	5 (1.8)	18 (12.9)	0	0.025	0.114
Hypertriglyceridemia	58 (20.6)	21 (7.5)	12 (8.6)	1 (0.7)	0.002	0.004
Diarrhea	53 (18.9)	3 (1.1)	18 (12.9)	3 (2.2)	0.128	0.375
COVID-19	51 (18.1)	0	14 (10.1)	1 (0.7)	0.031	0.319
Hypoalbuminemia	50 (17.8)	0	18 (12.9)	0	0.205	-
Hypercholesterolemia	49 (17.4)	3 (1.1)	11 (7.9)	0	0.009	0.222
Pyrexia	44 (15.7)	0	15 (10.8)	0	0.177	-
Abdominal pain	43 (15.3)	2 (0.7)	15(10.8)	0	0.207	0.319
Lymphocyte count decreased	42 (14.9)	15 (5.3)	26 (18.7)	7 (5.0)	0.325	0.896
Epistaxis	41 (14.6)	0	0	0	<0.001	-
Hypoesthesia	41 (14.6)	3 (1.1)	21 (15.1)	1 (0.7)	0.888	0.730
Blood creatinine increased	38 (13.5)	0	7 (5.0)	0	0.008	-
Hyperuricemia	37 (13.2)	0	4 (2.9)	0	<0.00	-
Hyponatremia	37 (13.2)	1 (0.4)	13 (9.4)	0	0.256	0.481
Cough	32 (11.4)	1 (0.4)	11 (7.9)	0	0.269	0.481
Stomatitis	30 (10.7)	9 (3.2)	5 (3.6)	2 (1.4)	0.098	0.287
Hyperglycemia	28 (10.0)	0	5 (3.6)	0	0.023	-
Abdominal distension	20 (7.1)	2 (0.7)	14 (10.1)	0	0.296	0.319
Leading to suvemcitug or placebo dose interruption	232 (82.6)		86 (61.9)		-	-
Leading to suvemcitug or placebo dose reduction	26 (9.3)		0		-	-
Leading to discontinuation of suvemcitug or placebo	19 (6.8)		3 (2.2)		-	-

Data are numbers (%) and shown for adverse events that occurred in at least 10% of the participants in either arm during the study intervention or up to 28 days after discontinuation of the intervention. The adverse events were graded according to the NCI CTCAE (v.5.0). *Two-sided chi-square test; no adjustment for multiple comparisons.

In subgroup analysis, suvemcitug demonstrated a clear PFS benefit in participants who were previously treated with an antiangiogenic agent, suggesting that suvemcitug could offer an effective treatment option in persons who have failed antiangiogenic therapy.

Optimal chemotherapy regimen for platinum-resistant, recurrent OC remains an area of uncertainty. In this trial, participants received investigator's choice of chemotherapy (paclitaxel versus PEGylated liposomal doxorubicin versus topotecan) and randomization was stratified on the basis of chemotherapeutic agent. In all three chemotherapy subgroups, adding suvemcitug to chemotherapy significantly prolonged PFS. In the paclitaxel subgroup, suvemcitug led to a 55% reduction in the risk of progression or death (HR: 0.45, 95% CI: 0.31–0.65). In the PEGylated

liposomal doxorubicin subgroup, there was a statistically nonsignificant trend for reduced risk of progression or death (HR: 0.69, 95% CI: 0.45–1.05). Participants receiving paclitaxel appeared to stay on treatment longer than those receiving liposomal doxorubicin (median exposure duration: 22.2 weeks versus 17.3 weeks; Supplementary Table 2). Similarly, in the AURELIA trial, PFS benefit with bevacizumab over placebo was greater in the paclitaxel cohort (HR: 0.46, 95% CI: 0.30–0.71) than the liposomal doxorubicin cohort (5.4 versus 3.5 months; HR: 0.57, 95% CI: 0.39–0.83)[29]. The findings from the AURELIA trial and the current trial, as well as real-world evidence, indicate that the combination of antiangiogenic therapy with paclitaxel is optimal[30]. Experiences with OC and breast cancer indicate that the pairing of bevacizumab and weekly paclitaxel may enhance antiangiogenic activities, resulting in a more pronounced antitumor effect than other chemotherapies[29,31,32]. Given the differential toxicity profile and cost of paclitaxel, topotecan and liposomal doxorubicin, choice of chemotherapy is worthy of further scrutiny. The small number of participants receiving topotecan in this trial makes it difficult to draw firm conclusions.

PFS benefit with suvemcitug was observed in the subgroup of participants with ascites at baseline. VEGF is involved in ascites formation in persons with OC and VEGF inhibition with bevacizumab resulted in improvement in PFS in the AURELIA trial[13,33,34]. A PFS benefit with suvemcitug was also seen in the subgroups of participants with at least three lines of prior antitumor therapies and with <3-month platinum-free interval in this trial. Considering the fact that these subgroups of participants have very limited therapeutic options, these findings are particularly encouraging.

A key issue in drug development for platinum-resistant OC has been the extent to which PFS benefit translates to an OS benefit[35,36]. Response patterns of targeted therapy including immune therapy and antiangiogenic therapy can differ greatly from traditional anticancer drugs such as chemotherapeutic drugs, with distinct kinetics of survival curves[37,38]. The OS benefit with suvemcitug in this trial was statistically significant albeit modest. The Kaplan–Meier OS curves of the two arms were relatively close during the first half of the trial period but the difference became more apparent as the follow-up time extended, especially after 18 months, supporting long-term survival benefits. Subsequent antitumor therapy may have attenuated the observed advantage in OS, which may have accounted for the modest prolongation of OS over the control arm. A preplanned supplementary analysis of OS in this trial that addressed subsequent antitumor therapy demonstrated that suvemcitug led to a 10.4-month extension of median OS, with a 41% reduction in the risk of death. The findings suggest that suvemcitug, when added to chemotherapy, conferred substantial benefits in terms of both PFS and OS. The robustness of these findings is supported by sensitivity and supplementary analyses, with the use of unstratified and stratified Cox proportional-hazards models.

The safety profile of suvemcitug in this trial is consistent with that reported by early-stage clinical trials[19,20], with no new safety concerns. The suvemcitug arm had higher rates of any grade TEAEs including neutropenia and thrombocytopenia but grade ≥3 TEAEs did not differ between the two arms with the exception of proteinuria and hypertension, two AEs consistently reported for antiangiogenic agents. The higher rates of TEAEs could be partially attributed to the myelosuppressive effects of longer chemotherapy exposure in the suvemcitug arm than the control arm in our view.

Suvemcitug is a humanized rabbit monoclonal IgG1 (κ) antibody and, except for the complementarity-determining region, has a similar sequence to bevacizumab. During the trial, ADA against suvemcitug was measured during treatment and up to 28 days after the last dose. ADA was identified in 13.7% of the participants in the suvemcitug arm but the rate of neutralizing ADA was low (1.4%).

This trial differs from the AURELIA trial in two key aspects. First, the AURELIA trial had an open-label design and the primary endpoint of PFS was assessed by investigators; in contrast, the current trial was double-blinded, with the primary endpoint of PFS evaluated by the BIRC. Second, participants in the AURELIA trial had no more than two prior lines of systemic treatment, none received prior PARP inhibitor therapy and only 7.2% of the participants were previously treated with bevacizumab; in contrast, participants in the current trial had up to six prior lines of systemic treatment and nearly half of the participants were previously treated with a PARP inhibitor and an antiangiogenic agent.

This trial had several limitations. Firstly, the exclusive recruitment of participants within China may limit the generalizability of findings to broader global populations. Secondly, this trial excluded persons who received ≥2 lines of systemic therapy for OC after platinum resistance, as well as persons with primary platinum-refractory OC who progressed during the first platinum-based chemotherapy, limiting applicability to the most heavily pretreated or more aggressive disease states. Thirdly, the trial did not did not examine germline and somatic *BRCA* mutations (or other HRD-related factors). Lastly, the COVID-19 pandemic caused notable disruptions in study treatment and assessment.

In conclusion, the addition of suvemcitug to chemotherapy led to a significant improvement in both PFS and OS in persons with platinum-resistant OC, with a manageable safety profile and no unexpected toxicities. The findings suggest that suvemcitug should be incorporated as a part of standard treatment in persons with platinum-resistant OC, including those who have received bevacizumab/PARP inhibitor.

Methods

The trial protocol and amendments were approved by the ethics committees of all participating centers (master protocol approved by the Ethics Committee of Cancer Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College). A full list of participating centers are available at ClinicalTrials.gov (NCT04908787). All participants provided written informed consent before any trial-related activities. The trial was conducted according to the Declaration of Helsinki and the Good Clinical Practice guidelines.

Study design and participants

This randomized, double-blind, placebo-controlled, phase 3 trial (SCORES) was conducted at 55 tertiary-care centers in China. Recruitment was conducted by screening persons seeking medical attention during daily practice. The inclusion criteria were as follows:

1. Age ≥ 18 years;
2. Histologically confirmed epithelial OC, fallopian tube cancer or primary peritoneal cancer; pathological types were high-grade serous adenocarcinoma, endometrioid carcinoma (G2 or G3), mixed epithelial carcinoma (high-grade serous adenocarcinoma and G2/G3 endometrioid carcinoma components had to account for more than 50%), malignant Brunner's tumor, undifferentiated carcinoma, dedifferentiated carcinoma and other rare types such as mesonephric duct-like carcinoma, gastric adenocarcinoma;
3. Persons with platinum-resistant recurrent OC who received a platinum-containing regimen and progressed on a platinum-containing regimen (platinum-refractory) or had a time to relapse of <6 months (184 calendar days) from the end of platinum-containing therapy (at least four cycles) until 28 days after the last dose.
   Definition of relapse or progression (any of the following):
   a) Documented radiographic progression;
   b) Persistent elevation of cancer antigen 125 (CA-125 ≥ 2 times upper limit of normal (ULN) and confirmed 1 week later) with clinical symptoms or physical examination suggestive of disease progression;
4. Progression during or after the most recent line of systemic therapy or intolerable therapy and at least one measurable lesion (assessed by investigator according to RECIST v.1.1) within 4 weeks before randomization;

5. ECOG performance score of 0–1 within 7 days before the first dose;
6. Previous chemotherapy ended ≥3 weeks from the first dose of this study, monoclonal antibody antitumor therapy ended ≥4 weeks from the first dose of this study, and small-molecule targeted therapy ended ≥2 weeks from the first dose of this study;
7. Treatment-related AEs recovered to the National Cancer Institute (NCI) Common Terminology Criteria for Adverse Events (CTCAE) grade ≤1 (except grade 2 alopecia);
8. Adequate organ function and meeting all of the following laboratory test results before enrollment:
   a) Bone marrow (no blood transfusion or blood products, granulocyte colony-stimulating factor or other hematopoietic stimulating factors were not used for correction within 14 days before blood routine examination during the screening period): neutrophils ≥ 1.5 × 109 L−1, hemoglobin ≥ 90 g L−1, platelets ≥ 100 × 109 L−1;
   b) Liver function: total bilirubin ≤ 1.5 × ULN, aspartate aminotransferase ≤ 3 × ULN, alanine aminotransferase ≤ 3 × ULN, alkaline phosphatase ≤ 3 × ULN; if liver metastasis, aspartate aminotransferase ≤ 5 × ULN, alanine aminotransferase ≤ 5 × ULN;
   c) Renal function: serum creatinine ≤ 1.5 ULN or creatinine clearance ≥ 60 ml min^{-1} calculated according to the Cockroft–Gault formula;
   d) Coagulation: international normalized ratio (INR) ≤ 1.5 (INR range should be 2–3 if individual is on a stable dose of warfarin for venous thrombosis management), activated partial thromboplastin time ≤ 1.5 ULN;
9. Estimated survival time ≥ 12 weeks;
10. Women of childbearing age agreed to remain abstinent or used contraception with an annual failure rate of <1% during treatment and for at least 6 months following the last dose of suvemcitug, placebo, paclitaxel, liposomal doxorubicin or topotecan, whichever occurred later.

The exclusion criteria were as follows:

1. Received >1 lines of systemic therapy for OC after platinum resistance and/or >1 lines of nonplatinum systemic therapy before platinum resistance;
2. Progression during the first platinum-based chemotherapy (from first dose to within 28 days after last dose);
3. Ovarian epithelial tumors with low malignant potential, such as low-grade serous adenocarcinoma, borderline tumors;
4. Ovarian mucinous carcinoma or clear cell carcinoma;
5. Nonepithelial tumors, such as sex cord and stromal tumors, germ cell tumors, carcinosarcoma;
6. Persons with other active malignant tumors within 5 years or at the same time (cured localized tumors, such as cutaneous basal cell carcinoma, cutaneous squamous cell carcinoma or cervical carcinoma in situ, can be enrolled);
7. Any pelvic or abdominal radiotherapy;
8. Recent major surgery or anticipated surgical intervention:
   A) Major surgery or notable trauma within 28 days before enrollment;
   B) Major surgical procedures anticipated during the course of the study, including but not limited to abdominal surgery (laparotomy or laparoscopy) before disease progression;
   C) Open biopsy performed within 7 days before enrollment;
9. Known hereditary or acquired bleeding and thrombophilia (for example, hemophilia, coagulopathy, thrombocytopenia or hypersplenism); clinically notable bleeding events, arterial or deep venous thromboembolic events or superficial venous thrombosis and myenteric venous thrombosis requiring intervention within 6 months before enrollment;
10. Taking aspirin (>325 mg per day) currently or recently (within 10 days before first dose);
11. Persons with a history of intestinal obstruction (including incomplete intestinal obstruction) within 3 months before enrollment; persons with a history of abdominal fistula, gastrointestinal perforation or abdominal abscess; persons with intestinal invasion found by imaging examination (computed tomography, magnetic resonance imaging) or pelvic examination during the screening period;
12. Severe infection requiring systemic antibiotic infusion or hospitalization during the screening period;
13. Persons with clinically manifested central nervous system disease, brain metastasis, stroke or transient ischemic attack within 6 months before enrollment;
14. Clinically notable cardiovascular disease:
   a) Uncontrolled hypertension (defined as systolic blood pressure ≥ 150 mmHg and/or diastolic blood pressure ≥ 100 mmHg after drug treatment);
   b) History of myocardial infarction or unstable angina within 6 months before enrollment;
   c) New York Heart Association class II and above heart failure;
   d) Severe arrhythmia requiring medication, excluding asymptomatic atrial fibrillation with controlled ventricular rate;
15. Left-ventricular ejection fraction < 50%;
16. Presence of neuropathy grade ≥2 (CTCAE 5.0) at screening;
17. Presence of severe nonhealing wound, ulcer or fracture; serous effusion (including pleural effusion and pericardial effusion) with clinical symptoms and requiring surgical treatment; difficult-to-control ascites;
18. Known serious hypersensitivity to the therapeutic agents or excipients used in the trial;
19. Pregnant or lactating women;
20. Persons with proteinuria (urine protein > 1 found in screening examination or urine protein > 1 that failed to return to normal within 24 h after retest);
21. Currently participating in another clinical study or planning to start treatment in this study less than 30 days before the end of treatment in the previous clinical study;
22. Other conditions that the investigator considered inappropriate for participation in this study.
23. Persons who have previously used BD0801. Participants were required to have platinum-refractory or resistant disease. Other key inclusion criteria included ≥1 measurable lesions according to the investigators per RECIST (v.1.1), an ECOG performance status of 0–1 and adequate hematologic and organ function. Persons who had primary platinum-refractory disease or who received ≥2 lines of systemic therapy for OC after platinum resistance were excluded. The full eligibility criteria are available in the trial protocol. Trial reporting followed the CONSORT 2010 statement[39].

Randomization and masking

Participants were randomized (2:1) to receive the investigator's choice of chemotherapy plus suvemcitug or placebo using a minimization technique. Randomization was conducted using an interactive web response system and stratified according to platinum-refractory status (yes versus no), number of prior systemic therapies (one versus two), chemotherapeutic agent (paclitaxel versus PEGylated liposomal doxorubicin versus topotecan) and prior antiangiogenic therapy (yes versus no). Participants were enrolled and assigned to interventions by site investigators. Investigators, participants and the sponsor were blinded to allocation assignment.

Procedures

Suvemcitug (1.5 mg kg^{-1}) or placebo was infused on days 1 and 15 of each 4-week cycle. Paclitaxel (80 mg m^{-2}; days 1, 8, 15 and 22), topotecan (4 mg m^{-2}; days 1, 8 and 15) or PEGylated liposomal doxorubicin (40 mg m^{-2}; day 1) was given intravenously every 4 weeks. Treatments were continued until disease progression, unacceptable toxicities, withdrawal of consent or death. Participants who ended treatment were followed up every 3 months for data on subsequent antitumor treatment and survival. Dose modifications of suvemcitug and chemotherapeutic drugs were allowed at the discretion of investigators. Two levels of dose modifications were permitted for suvemcitug (1.5 mg kg^{-1} to 1.0 mg kg^{-1} and 1.0 mg kg^{-1} to 0.5 mg kg^{-1}). Other protocol-mandated treatment changes are available in the trial protocol.

Tumor response was assessed radiologically by BIRC and investigators per RECIST (v.1.1) at baseline and every 8 weeks for the first 48 weeks and every 12 weeks thereafter until disease progression by BIRC, start of new antitumor therapy, death or withdrawal from the study, whichever occurred first.

Safety was assessed throughout the study using the NCI CTCAE (v.5.0). The occurrences, frequencies and severities of AEs were tabulated and all AEs were described in MedDRA (v.27.1) preferred terms and CTCAE grade.

Quality of life was assessed with the use of the EORTC QLQ-C30 and QLQ-OV28 questionnaires.

Outcomes

The primary endpoint was BIRC-assessed PFS, defined as the time from randomization to the first radiologically documented tumor progression or death, whichever occurred first, per RECIST (v.1.1). The key secondary endpoint was OS, defined as the interval from randomization to death of any cause. Other secondary endpoints included objective response rate (ORR), disease control rate (DCR), DOR, defined as the time from the first confirmed complete response or partial response to the first documented progressive disease or death of any cause, and quality of life. ORR, DCR and DOR were assessed by investigators and the BIRC. Safety endpoints included the incidence of AEs and serious AEs. Other endpoints including pharmacokinetics of suvemcitug and anti-suvemcitug antibodies will be reported elsewhere.

Statistics and reproducibility

The planned sample size was 411. The statistical power was based on the total number events of PFS per RECIST (v.1.1) by BIRC and OS. Assuming a treatment effect HR of 0.69, corresponding to an improvement in median PFS from 4.4 months in the placebo arm to 6.4 months in the suvemcitug arm, 304 PFS events would provide 87% power to detect the PFS treatment effect at one-sided significance level of 0.025. For the key secondary endpoint of OS, 278 events would provide 80% power to detect an HR of 0.70, corresponding to an improvement in median OS from 13.3 months in the placebo arm to 19.0 months in the suvemcitug arm. The familywise type I error was controlled in a fixed sequential testing manner, that is, the OS was tested only if the treatment effect of PFS was statistically significant at a one-sided α level of 0.025. An administrative one-sided α level of 0.0001 would be spent on the OS analysis along with PFS primary analysis.

Efficacy was assessed in the full analysis set, which included all randomized participants, when 304 PFS events had occurred in all randomized participants. The second analysis was performed when 278 OS events occurred. PFS and other time-to-event endpoints were analyzed using the Kaplan–Meier method and the corresponding 95% CIs for median time were calculated using the Brookmeyer–Crowley method. The primary hypothesis for BIRC-assessed PFS was evaluated using a stratified log-rank test. HRs were estimated using a stratified Cox proportional-hazards model with Efron's method for tie handling. Unstratified HRs were calculated as well. ORR and DCR were estimated for each arm, along with their two-sided 95% CIs, using the Clopper–Pearson method. The rate differences between arms were calculated using the Miettinen–Nurminen methods[40]. Prespecified subgroup analyses of PFS by BIRC and OS were conducted using similar methods to those for the primary endpoint. Sensitivity and supplementary analyses for PFS were performed as specified in the statistical analysis plan. At the final analysis, the actual values of corrected stratification factors were used for stratified analyses. Given the use of dynamic randomization method without increasing type I error, log-rank test *P* values were calculated using the rerandomization method[41] and original log-rank test *P* values served as nominal *P* values. Supplementary analyses for OS were carried out using a hypothetical strategy by censoring subsequent antitumor therapy.

The Cox regression is based on the proportional-hazards model assumption. Before analysis, the proportional-hazards assumption was verified for the following endpoints in this study: BIRC-assessed PFS and investigator-assessed PFS and OS.

The safety set included participants who received at least one dose of the study medications. An independent data monitoring committee monitored the ongoing safety data until the first analysis.

All analyses and data processing were completed using SAS (v.9.4).

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

The trial protocol and statistical analysis plan are available in the Supplementary Information. All other data supporting the findings of this study (detailed AEs and Kaplan–Meier curves in the subgroup analyses) are available from the corresponding authors on reasonable request. Source data are provided with this paper.

Code availability

There was no custom code or mathematical algorithm central to this study's conclusion.

References

1. Bray, F. et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J. Clin.* **74**, 229–263 (2024).
2. Colombo, N. et al. ESMO–ESGO consensus conference recommendations on ovarian cancer: pathology and molecular biology, early and advanced stages, borderline tumours and recurrent disease. *Ann. Oncol.* **30**, 672–705 (2019).
3. Kuroki, L. & Guntupalli, S. R. Treatment of epithelial ovarian cancer. *Brit. Med. J.* **371**, m3773 (2020).
4. González-Martín, A. et al. The systemic treatment of recurrent ovarian cancer revisited. *Ann. Oncol.* **32**, 710–725 (2021).
5. Tattersall, A., Ryan, N., Wiggans, A. J., Rogozińska, E. & Morrison, J. Poly(ADP-ribose) polymerase (PARP) inhibitors for the treatment of ovarian cancer. *Cochrane Database Syst. Rev.* **2**, CD007929 (2022).
6. Dewani, D., Jaiswal, A. & Karwade, P. Poly(adenosine diphosphate ribose) polymerase (PARP) inhibitors in the treatment of advanced ovarian cancer: a narrative review. *Cureus* **16**, e68463 (2024).
7. Ledermann, J. A. et al. Newly diagnosed and relapsed epithelial ovarian carcinoma: ESMO clinical practice guidelines for diagnosis, treatment and follow-up. *Ann. Oncol.* **24**, vi24–vi32 (2013).
8. Richardson, D. L., Eskander, R. N. & O'Malley, D. M. Advances in ovarian cancer care and unmet treatment needs for patients with platinum resistance: a narrative review. *JAMA Oncol.* **9**, 851–859 (2023).

9. Lortholary, A. et al. Weekly paclitaxel as a single agent or in combination with carboplatin or weekly topotecan in patients with resistant ovarian cancer: the CARTAXHY randomized phase II trial from Groupe d'Investigateurs Nationaux pour l'Etude des Cancers Ovariens (GINECO). *Ann. Oncol.* **23**, 346–352 (2012).
10. Berek, J. S., Renz, M., Kehoe, S., Kumar, L. & Friedlander, M. Cancer of the ovary, fallopian tube, and peritoneum: 2021 update. *Int. J. Gynaecol. Obstet.* **155**, 61–85 (2021).
11. Jin, C., Yuan, M., Bu, H. & Jin, C. Antiangiogenic strategies in epithelial ovarian cancer: mechanism, resistance, and combination therapy. *J. Oncol.* **2022**, 4880355 (2022).
12. Dhani, N. C. & Oza, A. M. Targeting angiogenesis: taming the Medusa of ovarian cancer. *Hematol. Oncol. Clin. North Am.* **32**, 1041–1055 (2018).
13. Pujade-Lauraine, E. et al. Bevacizumab combined with chemotherapy for platinum-resistant recurrent ovarian cancer: the AURELIA open-label randomized phase III trial. *J. Clin. Oncol.* **32**, 1302–1308 (2014).
14. Tew, W. P., Lacchetti, C. & Kohn, E. C., PARP Inhibitors in the Management of Ovarian Cancer Guideline Expert Panel. Poly(ADP-ribose) polymerase inhibitors in the management of ovarian cancer: ASCO Guideline rapid recommendation update. *J. Clin. Oncol.* **40**, 3878–3881 (2022).
15. Arend, R. C. et al. Ofranergene obadenovec (Ofra-Vec, VB-111) with weekly paclitaxel for platinum-resistant ovarian cancer: randomized controlled phase III trial (OVAL Study/GOG 3018). *J. Clin. Oncol.* **42**, 170–179 (2024).
16. Xue, L. et al. Antiangiogenic antibody BD0801 combined with immune checkpoint inhibitors achieves synergistic antitumor activity and affects the tumor microenvironment. *BMC Cancer* **21**, 1134 (2021).
17. Liu, L. et al. Molecular targeting of VEGF/VEGFR signaling by the anti-VEGF monoclonal antibody BD0801 inhibits the growth and induces apoptosis of human hepatocellular carcinoma cells in vitro and in vivo. *Cancer Biol. Ther.* **18**, 166–176 (2017).
18. Holmes, D. I. & Zachary, I. The vascular endothelial growth factor (VEGF) family: angiogenic factors in health and disease. *Genome Biol.* **6**, 209 (2005).
19. Mao, C. et al. Suvemcitug as second-line treatment of advanced or metastatic solid tumors and with FOLFIRI for pretreated metastatic colorectal cancer: phase Ia/Ib open label, dose-escalation trials. *ESMO Open* **8**, 101540 (2023).
20. Yuan, G. et al. Suvemcitug plus chemotherapy for platinum-resistant epithelial ovarian, fallopian tube and primary peritoneal cancer: a phase 1b dose-escalation trial. *Gynecol. Oncol.* **187**, 212–220 (2024).
21. Bamias, A. et al. Bevacizumab with or after chemotherapy for platinum-resistant recurrent ovarian cancer: exploratory analyses of the AURELIA trial. *Ann. Oncol.* **28**, 1842–1848 (2017).
22. Moore, K. N. & Pothuri, B. Poly(ADP-ribose) polymerase inhibitor inhibition in ovarian cancer: a comprehensive review. *Cancer J.* **27**, 432–440 (2021).
23. Moore, K. et al. Maintenance olaparib in patients with newly diagnosed advanced ovarian cancer. *N. Engl. J. Med.* **379**, 2495–2505 (2018).
24. Kokabu, T. et al. Effects of PARP inhibitors on subsequent platinum-based chemotherapy in patients with recurrent ovarian cancer. *Cancers (Basel)* **16**, 2651 (2024).
25. Chekerov, R. et al. Sorafenib plus topotecan versus placebo plus topotecan for platinum-resistant ovarian cancer (TRIAS): a multicentre, randomised, double-blind, placebo-controlled, phase 2 trial. *Lancet Oncol.* **19**, 1247–1258 (2018).
26. Wang, T. et al. Effect of apatinib plus pegylated liposomal doxorubicin vs pegylated liposomal doxorubicin alone on platinum-resistant recurrent ovarian cancer: the APPROVE randomized clinical trial. *JAMA Oncol.* **8**, 1169–1176 (2022).
27. du Bois, A. et al. Incorporation of pazopanib in maintenance therapy of ovarian cancer. *J. Clin. Oncol.* **32**, 3374–3382 (2014).
28. Shen, W. et al. Anlotinib in patients with recurrent platinum resistant/refractory ovarian cancer: a prospective, single arm, phase II study. *Int. J. Gynecol. Cancer* **33**, 1764–1770 (2023).
29. Poveda, A. M. et al. Bevacizumab combined with weekly paclitaxel, pegylated liposomal doxorubicin, or topotecan in platinum-resistant recurrent ovarian cancer: analysis by chemotherapy cohort of the randomized phase III AURELIA trial. *J. Clin. Oncol.* **33**, 3836–3838 (2015).
30. Moffat, G. T. et al. Real-world outcomes associated with bevacizumab combined with chemotherapy in platinum-resistant ovarian cancer. *Gynecol. Oncol.* **184**, 51–56 (2024).
31. Miller, K. et al. Paclitaxel plus bevacizumab versus paclitaxel alone for metastatic breast cancer. *N. Engl. J. Med.* **357**, 2666–2676 (2007).
32. Ai, B., Bie, Z., Zhang, S. & Li, A. Paclitaxel targets VEGF-mediated angiogenesis in ovarian cancer treatment. *Am. J. Cancer Res.* **6**, 1624–1635 (2016).
33. Liang, B., Guo, Z., Li, Y. & Liu, C. Elevated VEGF concentrations in ascites and serum predict adverse prognosis in ovarian cancer. *Scand. J. Clin. Lab. Invest.* **73**, 309–314 (2013).
34. Herr, D. et al. VEGF induces ascites in ovarian cancer patients via increasing peritoneal permeability by downregulation of claudin 5. *Gynecol. Oncol.* **127**, 210–216 (2012).
35. Eskander, R. N. et al. Overcoming the challenges of drug development in platinum-resistant ovarian cancer. *Front. Oncol.* **13**, 1258228 (2023).
36. Bachmann, C. New achievements from molecular biology and treatment options for refractory/relapsed ovarian cancer—a systematic review. *Cancers (Basel)* **15**, 5356 (2023).
37. Thorén, F. B., Anderson, H. & Strannegård, Ö. Late divergence of survival curves in cancer immunotherapy trials: interpretation and implications. *Cancer Immunol. Immunother.* **62**, 1547–1551 (2013).
38. Johnson, M. L. Delayed separation of Kaplan–Meier curves is commonly observed in studies of advanced/metastatic solid tumors treated with anti-PD-(L)1 therapy: systematic review and meta-analysis. *Target. Oncol.* **20**, 45–56 (2025).
39. Schulz, K. F., Altman, D. G. & Moher, D., CONSORT Group. CONSORT 2010 statement: updated guidelines for reporting parallel group randomized trials. *Brit. Med. J.* **23**, 340 (2010).
40. Miettinen, O. & Nurminen, M. Comparative analysis of two rates. *Stat. Med.* **4**, 213–226 (1985).
41. US Food and Drug Administration. *Adaptive Designs for Clinical Trials of Drugs and Biologics Guidance for Industry*. Available at https://www.fda.gov/regulatory-information/search-fda-guidance-documents/adaptive-design-clinical-trials-drugs-and-biologics-guidance-industry (accessed November 29, 2019).

Acknowledgements

This trial was sponsored by Simcere Zaiming Pharmaceuticals Co., Ltd. The sponsor participated in data analysis, methodology, project administration and software but not in trial design and data collection. We thank all participants; writing support was provided by B. Cui and K. Zhang of Ivy Medical Editing.

Author contributions

Conceptualization, L.W., Q.L. and G.Y. Data curation, G.Y., G. Lou and J.L. Formal analysis, G.Y., G. Lou, J.L., M.X., X.L., D.W., K.Z., T.Z., X.L., Y.H., W.D., K.W., Q.Z., G. Li, C.Y., J.Z., H.S. and R.T. Investigation, G.Y.,

G. Lou, J.L., M.X., X.L., D.W., K.Z., T.Z., X.L., Y.H., W.D., K.W., Q.Z., G. Li, Q.L. and L.W. Methodology, C.Y., J.Z., H.S. and R.T. Project administration, G.Y., G. Lou, J.L., M.X., X.L., D.W., K.Z., T.Z., X.L., Y.H., W.D., K.W., Q.Z., G. Li, C.Y., J.Z., H.S. and R.T. Software, H.S. Writing—original draft, G.Y., G. Lou, J.L., M.X., X.L., D.W., K.Z., T.Z., X.L., Y.H., W.D., K.W., Q.Z., G. Li, C.Y., J.Z., H.S. and R.T. Writing—review and editing, all authors.

Competing interests

Simcere Zaiming Pharmaceuticals Co., Ltd., the sponsor of this trial, covered all trial costs, including investigational drugs, laboratory tests and associated operational cost. C.Y., J.Z., H.S. and R.T. are full-time employees of Simcere Zaiming Pharmaceuticals Co., Ltd. The other authors declare no competing interests.

Additional information

Extended data is available for this paper at https://doi.org/10.1038/s43018-025-01085-z.

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s43018-025-01085-z.

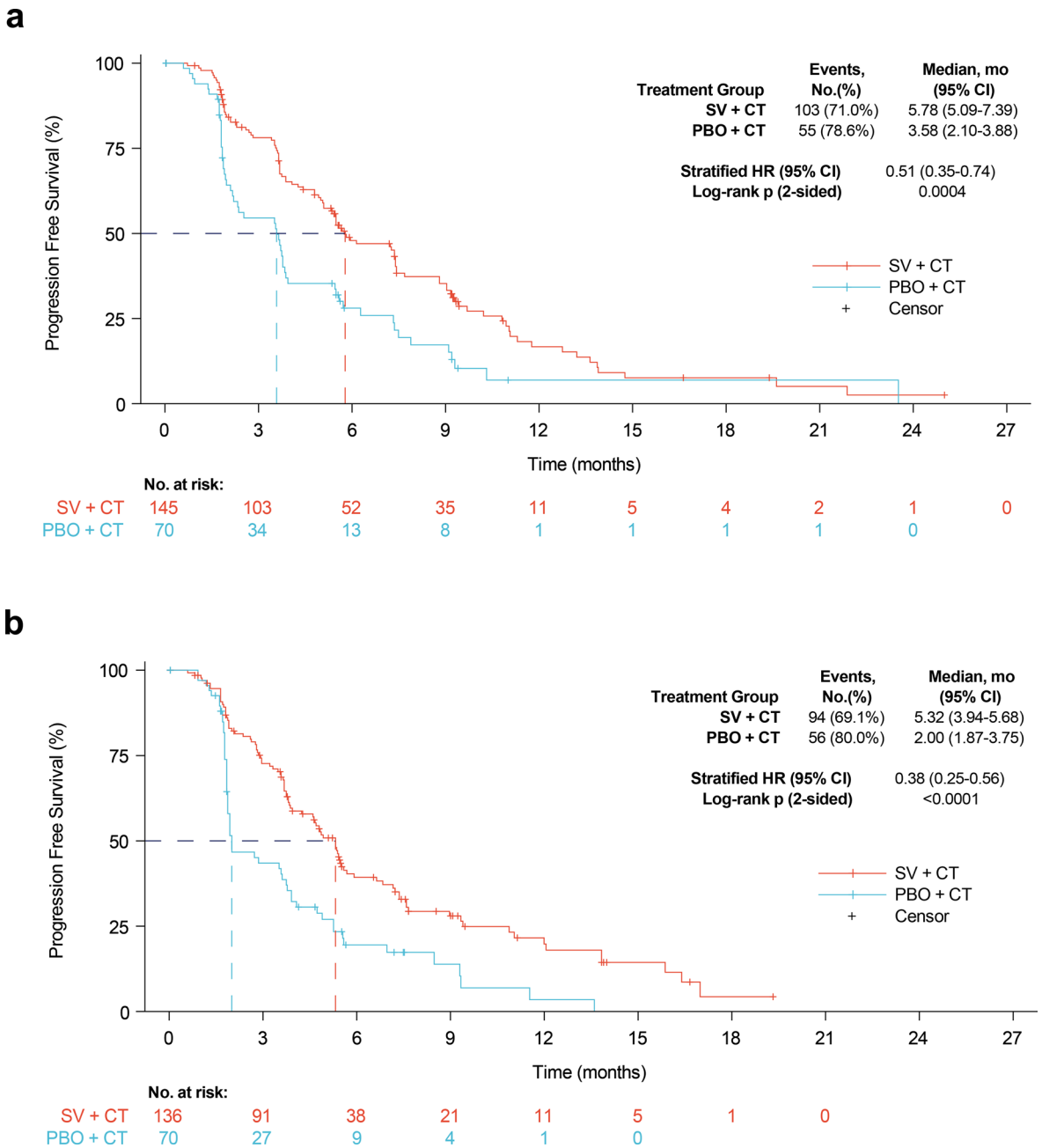
Correspondence and requests for materials should be addressed to Qingshui Li or Lingying Wu.

Peer review information *Nature Cancer* thanks the anonymous reviewer(s) for their contribution to the peer review of this work.

Guangwen Yuan[1,21], Ge Lou[2,21], Jundong Li[3,21], Mei Xu[4], Xiaowei Liu[5], Danbo Wang[6], Keqiang Zhang[7], Tao Zhu[8], Xiumin Li[9], Yi Huang[10], Wei Duan[11], Ke Wang[12], Qi Zhou[13], Guiling Li[14], Chen Yang[15,16], Jiajing Zhang[15,16], Haolin Sun[16,17], Renhong Tang[16,18], Qingshui Li[19] ✉ & Lingying Wu[1,20] ✉

[1]Department of Gynecology Oncology, National Cancer Center/National Clinical Research Center for Cancer/Cancer Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China. [2]Department of Gynecology, Harbin Medical University Cancer Hospital, Harbin, China. [3]Department of Gynecology, Sun Yat-Sen University Cancer Center, Guangzhou, China. [4]Department of Obstetrics and Gynecology, Xuzhou Central Hospital, Xuzhou, China. [5]Department of Oncology, Affiliated Hospital of Jining Medical University, Jining, China. [6]Department of Gynecology, Liaoning Cancer Hospital and Institute, Shenyang, China. [7]Department of Gynecologic Oncology, Hunan Cancer Hospital, Changsha, China. [8]Department of Gynecologic Oncology, Zhejiang Cancer Hospital, Hangzhou, China. [9]Department of Gynecologic Oncology, Linyi Cancer Hospital, Linyi, China. [10]Department of Gynecologic Oncology, Hubei Cancer Hospital, Wuhan, China. [11]Department of Gynecologic Oncology, Beijing Obstetrics and Gynecology Hospital, Capital Medical University, Beijing, China. [12]Department of Gynecologic Oncology, Tianjin Medical University Cancer Institute & Hospital, Tianjin, China. [13]Department of Gynecologic Oncology, Chongqing Cancer Hospital, Chongqing, China. [14]Department of Gynecologic Oncology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China. [15]Clinical Sciences, Simcere Zaiming Pharmaceutical Co., Ltd., Shanghai, China. [16]State Key Laboratory of Neurology and Oncology Drug Development, Nanjing, China. [17]Clinical Statistics, Simcere Zaiming Pharmaceutical Co., Ltd., Shanghai, China. [18]Simcere Zaiming Pharmaceutical Co., Ltd., Shanghai, China. [19]Department of Gynecologic Oncology, Shandong Cancer Hospital and Institute, Shandong First Medical University and Shandong Academy of Medical Sciences, Jinan, China. [20]National Cancer Center/National Clinical Research Center for Cancer/Cancer Hospital & Liaoning Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Shenyang, China. [21]These authors contributed equally: Guangwen Yuan, Ge Lou, Jundong Li. ✉e-mail: 13854158117@163.com; wulingying@csco.org.cn

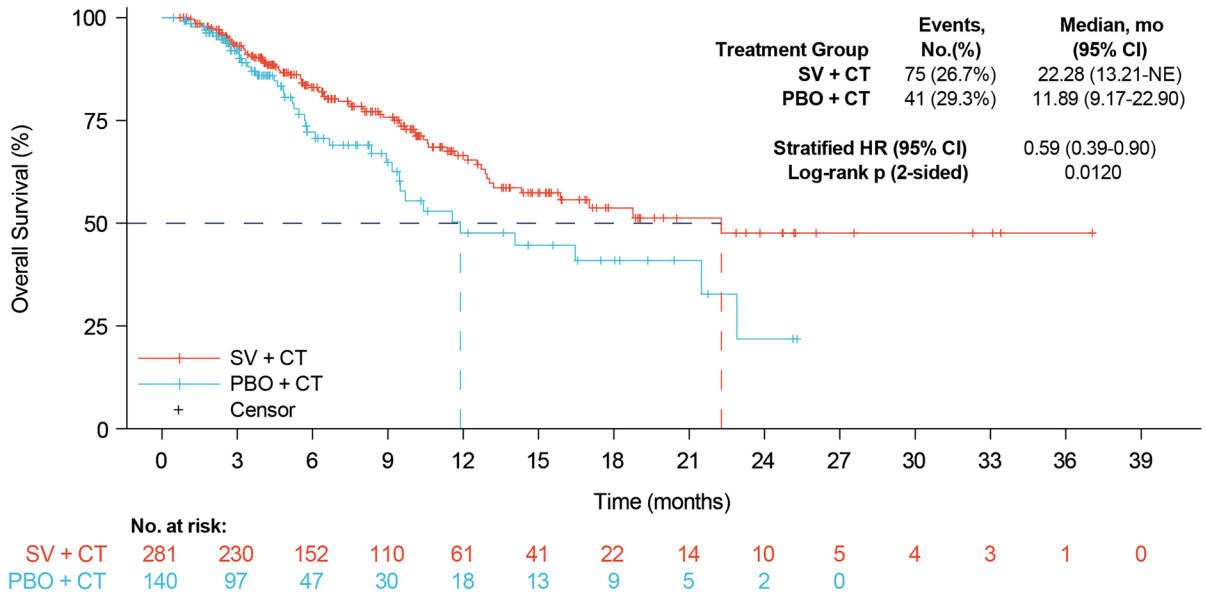
https://doi.org/10.1038/s43018-025-01085-z



Extended Data Fig. 1 | Subgroup analyses of progression-free survival based on prior exposure to PARP inhibitors. a, patients who were PARP inhibitor naïve. Number of patients: 145 and 70 in the suvemcitug and placebo groups, respectively. *P = 0.0004*. **b**, patients who had received prior PARP inhibitor therapy. Number of patients: 136 and 70 in the suvemcitug and placebo groups, respectively. *P < 0.0001*.

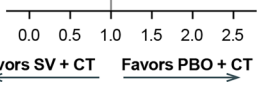
https://doi.org/10.1038/s43018-025-01085-z

a



b

Subgroup	Events/N SV + CT	Events/N PBO + CT	Hazard Ratio 95% CI	Median, mo SV + CT	Median, mo PBO + CT	Hazard Ratio 95% CI
Overall	178/281	101/140		15.31	14.03	0.80(0.63,1.02)
Age						
<=65	151/243	87/120		14.78	14.03	0.78(0.60,1.02)
>65	27/38	14/20		16.23	15.10	0.94(0.50,1.80)
Baseline ECOG						
0	63/106	24/40		16.43	19.19	1.01(0.63,1.61)
1	115/175	77/100		14.72	12.12	0.74(0.56,0.99)
Ascites						
Y	71/92	33/46		9.53	11.27	1.15(0.76,1.74)
N	107/189	68/94		18.92	15.24	0.65(0.48,0.89)
Sum of diameters for all target lesions						
<42.00	79/142	53/68		16.62	11.52	0.56(0.39,0.80)
>=42.00	99/139	48/72		14.39	16.56	1.13(0.80,1.59)
Number of lines of previous anti-tumor regimens						
1	50/87	29/40		17.81	14.88	0.70(0.44,1.10)
2	73/106	41/52		14.19	13.31	0.77(0.53,1.14)
>=3	55/88	31/48		15.47	12.62	0.91(0.59,1.42)
Platinum-free interval (month)						
<3	73/110	44/58		13.90	11.66	0.79(0.54,1.14)
>=3	105/171	57/82		16.66	16.46	0.80(0.58,1.11)
Number of previous systemic therapy						
1	116/190	67/93		16.07	14.78	0.77(0.57,1.05)
2	62/91	34/47		14.46	11.89	0.87(0.57,1.32)
Platinum-refractory ovarian cancer patients						
Y	17/25	9/14		14.46	9.86	0.89(0.40,2.02)
N	161/256	92/126		15.74	14.78	0.79(0.61,1.02)
Chemotherapy						
Paclitaxel	71/124	40/62		17.81	16.46	0.82(0.56,1.21)
Topotecan	49/69	26/34		13.37	11.89	0.81(0.50,1.30)
Doxorubicin	58/88	35/44		13.90	12.48	0.78(0.51,1.19)
Received anti-angiogenic therapy						
Y	98/139	57/69		13.24	10.05	0.73(0.53,1.01)
N	80/142	44/71		18.76	19.09	0.86(0.60,1.25)
Received PARP inhibitors						
Y	86/138	50/70		14.78	15.38	0.82(0.58,1.16)
N	92/143	51/70		15.93	11.56	0.78(0.55,1.10)

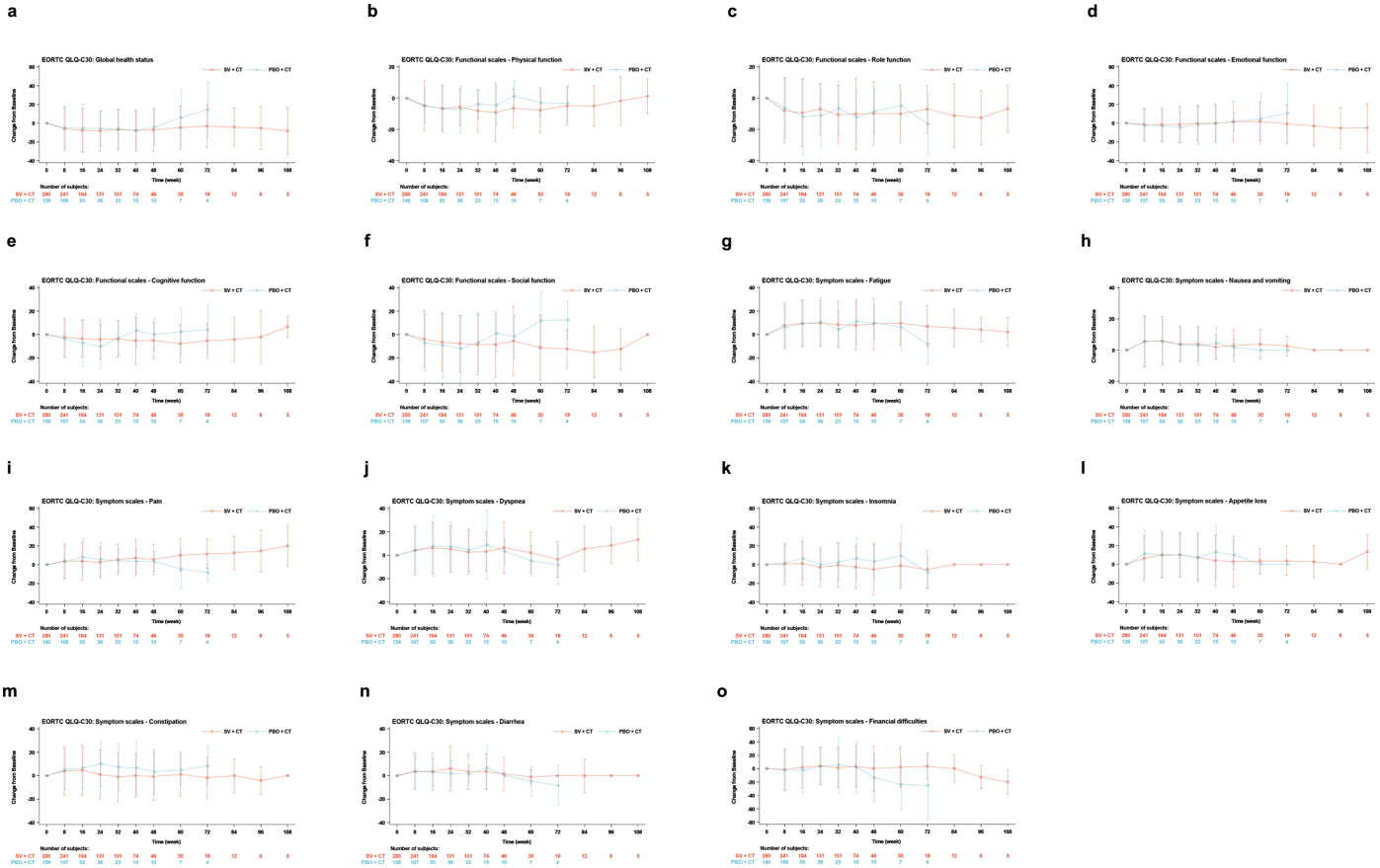


Extended Data Fig. 2 | See next page for caption.

https://doi.org/10.1038/s43018-025-01085-z

Extended Data Fig. 2 | Supplementary and subgroup analyses of overall survival. a, Kaplan-Meier curve of overall survival of patients without subsequent antitumor therapy. Number of patients: 281 and 140 in the suvemcitug and placebo groups, respectively. *P = 0.01*. Hypothetical estimands strategy was used for the supplemental analysis. 95% CI, 95% confidence interval; HR, hazard ratio; PBO + CT, placebo plus chemotherapy; SV + CT, suvemcitug plus chemotherapy. **b**, Forest plots for overall survival. Shown are the results of prespecified subgroup analyses of overall survival at the final analysis in the full analysis set. The hazard ratio (HR) for progression or death that is reported for all the randomized patients was based on a Cox proportional-hazards model.

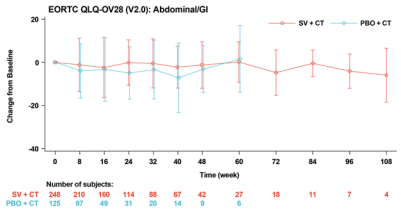
https://doi.org/10.1038/s43018-025-01085-z



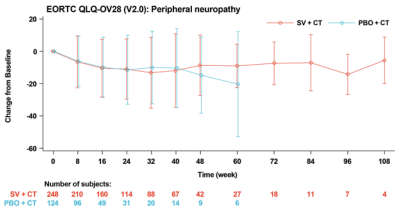
Extended Data Fig. 3 | EORTC QLQ-C30 change from baseline over time. **a**, global health scale, functional scales. **b**, physical function. **c**, role function. **d**, emotional function. **e**, cognitive function. **f**, social function and symptom scales. **g**, fatigue. **h**. nausea and vomiting. **i**, pain. **j**, dyspnea. **k**, insomnia. **l**, appetite loss. **m**, constipation. **n**, diarrhea. **o**, financial difficulties. Scores range from 0 to 100; a higher score represents higher ('better') global health status/overall quality of life and function or symptoms. EORTC, European Organization for Research and Treatment of Cancer; GHS, global health scale; QLQ-C30, Quality of Life Core 30 questionnaire; QoL, quality of life; SE, standard error; PBO + CT, placebo plus chemotherapy; SV + CT, suvemcitug plus chemotherapy.

https://doi.org/10.1038/s43018-025-01085-z

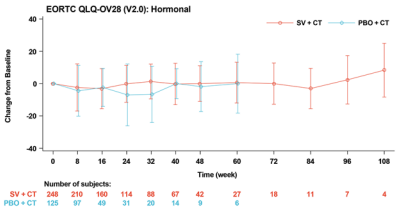
a



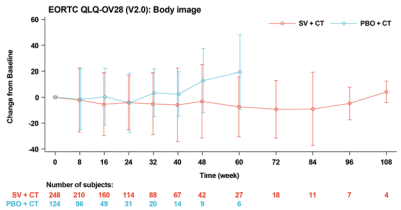
b



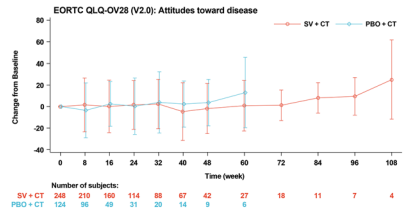
c



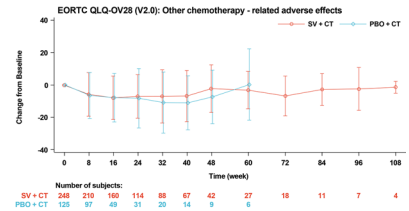
d



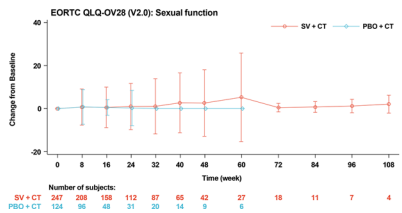
e



f



g



Extended Data Fig. 4 | Mean changes in EORTC QLQ Ovarian Cancer Module (EORTC QLQ-OV28) QoL parameters. a, QLQ-OV28 abdominal/gastrointestinal symptoms. **b**, peripheral neuropathy. **c**, hormonal. **d**, body image. **e**, attitudes toward disease. **f**, other chemotherapy-related adverse effects. **g**, sexual function. Scores range from 0 to 100; a higher score represents better function or symptoms. EORTC, European Organization for Research and Treatment of Cancer; QoL, quality of life; SE, standard error; PBO + CT, placebo plus chemotherapy; SV + CT, suvemcitug plus chemotherapy.

nature portfolio

Corresponding author(s): Lingying Wu

Last updated by author(s): Sep 2, 2025

Reporting Summary

Nature Portfolio wishes to improve the reproducibility of the work that we publish. This form provides structure for consistency and transparency in reporting. For further information on Nature Portfolio policies, see our Editorial Policies and the Editorial Policy Checklist.

Statistics

For all statistical analyses, confirm that the following items are present in the figure legend, table legend, main text, or Methods section.

n/a	Confirmed	
☐	☒	The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement
☒	☐	A statement on whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
☐	☒	The statistical test(s) used AND whether they are one- or two-sided *Only common tests should be described solely by name; describe more complex techniques in the Methods section.*
☐	☒	A description of all covariates tested
☐	☒	A description of any assumptions or corrections, such as tests of normality and adjustment for multiple comparisons
☐	☒	A full description of the statistical parameters including central tendency (e.g. means) or other basic estimates (e.g. regression coefficient) AND variation (e.g. standard deviation) or associated estimates of uncertainty (e.g. confidence intervals)
☐	☒	For null hypothesis testing, the test statistic (e.g. *F*, *t*, *r*) with confidence intervals, effect sizes, degrees of freedom and *P* value noted *Give P values as exact values whenever suitable.*
☒	☐	For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings
☒	☐	For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes
☒	☐	Estimates of effect sizes (e.g. Cohen's *d*, Pearson's *r*), indicating how they were calculated

Our web collection on statistics for biologists contains articles on many of the points above.

Software and code

Policy information about availability of computer code

Data collection	The EDC system of cubeCDMS® v1.1 from Shanghai CRScube Co., Ltd.; The IRT system of IRTON® v5.0 from Shanghai Shanhu Health Technology Co., Ltd.
Data analysis	SAS® 9.4 from SAS Institute.

For manuscripts utilizing custom algorithms or software that are central to the research but not yet described in published literature, software must be made available to editors and reviewers. We strongly encourage code deposition in a community repository (e.g. GitHub). See the Nature Portfolio guidelines for submitting code & software for further information.

Data

Policy information about availability of data

All manuscripts must include a data availability statement. This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A description of any restrictions on data availability
- For clinical datasets or third party data, please ensure that the statement adheres to our policy

The trial protocol and statistical analysis plan are available in Supplementary Information. The deidentified individual data are available in Source Data files. All other data supporting the findings of this study (detailed AEs and Kaplan-Meier curves in the subgroup analyses) are available from the corresponding author on reasonable request.

Research involving human participants, their data, or biological material

Policy information about studies with human participants or human data. See also policy information about sex, gender (identity/presentation), and sexual orientation and race, ethnicity and racism.

Reporting on sex and gender	Adult women
Reporting on race, ethnicity, or other socially relevant groupings	Chinese
Population characteristics	Women with histologically-confirmed ovarian cancer that had progressed during or within 6 months after completing platinum-based therapy
Recruitment	At both outpatient and inpatient departments
Ethics oversight	Institutional review board at all participating centres

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Field-specific reporting

Please select the one below that is the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

☒ Life sciences ☐ Behavioural & social sciences ☐ Ecological, evolutionary & environmental sciences

For a reference copy of the document with all sections, see nature.com/documents/nr-reporting-summary-flat.pdf

Life sciences study design

All studies must disclose on these points even when the disclosure is negative.

Sample size	The planned sample size was 411. The statistical power was based on the total number events of PFS per RECIST, version 1.1 by BIRC and OS. Assuming a treatment effect (hazard ratio [HR]) of 0.69, corresponding to an improvement in median PFS from 4.4 months in the placebo plus chemotherapy arm, hereafter referred to as the placebo arm, to 6.4 months in the suvemcitug plus chemotherapy arm, hereafter referred to as the suvemcitug arm, 304 PFS events would provide about 87% power to detect the PFS treatment effect at one-sided significant level of 0.025.
Data exclusions	No data were excluded from analysis.
Replication	Not applicable.
Randomization	Eligible patients were randomized at a 2:1 ratio.
Blinding	Both the patients and outcome assessors were blinded.

Reporting for specific materials, systems and methods

We require information from authors about some types of materials, experimental systems and methods used in many studies. Here, indicate whether each material, system or method listed is relevant to your study. If you are not sure if a list item applies to your research, read the appropriate section before selecting a response.

Materials & experimental systems

n/a	Involved in the study
☒	☐ Antibodies
☒	☐ Eukaryotic cell lines
☒	☐ Palaeontology and archaeology
☒	☐ Animals and other organisms
☐	☒ Clinical data
☒	☐ Dual use research of concern
☒	☐ Plants

Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging

Clinical data

Policy information about clinical studies

All manuscripts should comply with the ICMJE guidelines for publication of clinical research and a completed CONSORT checklist must be included with all submissions.

Clinical trial registration	ClinicalTrials.gov Identifier: NCT04908787
Study protocol	Uploaded as supplementary document
Data collection	June 5, 2021 to Oct 11, 2024
Outcomes	The primary endpoint was progression-free survival as assessed by an independent review committee. The key secondary endpoint was overall survival. Other secondary endpoints included objective response rate, disease control rate, duration of response, quality of life (QoL), safety, pharmacokinetics of suvemcitug, and anti-suvemcitug antibodies.

Plants

Seed stocks	Not applicable
Novel plant genotypes	Not applicable
Authentication	Not applicable