Abstracts

Abstract 2 Table 1

Pembrolizumab (n=31)
32 (17–51)
74 (55–88)
NR (1.5+ to 7.9+)
4.4 (4.0-8.5)
6 (24)
4 (21)
29 (94)
21 (68)
1 (3) ^c
uation 4 (13)
le ^d Grade 3
6 (19)
3 (10)
0
0
1 (3)
2 (6)

^aDefined as best overall response of complete or partial response, or stable disease. ^bPercentages are based on the total number of patients in each prior therapy subgroup. ^cOne patient had a serious AE of hypovolemic shock that led to death. ^dAmong treatment-related AEs occurring in ≥10 patients, none were grade 4/5. AE, adverse event; NR, not reached; ORR, objective response rate.

central review per RECIST v1.1) and safety. Secondary endpoints included disease control rate, duration of response, and progression-free survival.

Results 31 patients with ovarian cancer received ≥ 1 dose of lenvatinib plus pembrolizumab in LEAP-005 (median age 62 years [range 40–76]); median study follow-up was 7.8 months (range, 4.6–12.4) as of April 10, 2020. ORR was 32% (95% CI, 17–51); other efficacy endpoints were also favorable (table 1). Treatment-related adverse events occurred in 29 (94%) patients (table 1).

Conclusion Lenvatinib plus pembrolizumab demonstrated encouraging efficacy and manageable safety in patients with heavily pretreated ovarian cancer, including those with prior platinum failure and those with previous bevacizumab exposure.

IGCS20_1268

3

POSTPROGRESSION EFFICACY OUTCOMES FROM THE PHASE 3 ARIEL3 STUDY OF RUCAPARIB IN PATIENTS WITH PLATINUM-SENSITIVE RECURRENT OVARIAN CARCINOMA ASSOCIATED WITH EITHER BRCA1 OR BRCA2 MUTATIONS

¹J Weberpals*, ²A Oza, ³D Lorusso, ³G Scambia, ⁴C Aghajanian, ⁵A Oaknin, ⁶A Dean, ⁷N Colombo, ⁸AR Clamp, ⁹A Leary, ¹⁰RW Holloway, ¹¹M Amenedo Gancedo, ¹²PC Fong, ¹³JC Goh, ¹⁴DM O'Malley, ¹⁵DK Armstrong, ¹⁶S Banerjee, ¹⁷J García-Donas, ¹⁸EM Swisher, ¹⁹T Cameron, ²⁰L Maloney, ²⁰S Goble, ²¹RL Coleman, ²²JA Ledermann. ¹Ottawa Hospital Research Institute. Canada: ²Princess Margaret Cancer Centre. University Health Network. Canada; ³Fondazione Policlinico Universitario A. Gemelli IRCCS, Italy; ⁴Memorial Sloan Kettering Cancer Center, USA; ⁵Vall d'Hebron University Hospital, Vall d'Hebron Institute of Oncology (VHIO), Spain; ⁶St John of God Subiaco Hospital, Australia; ⁷European Institute of Oncology IRCCS and University of Milan-Bicocca, Italy; ⁸The Christie NHS Foundation Trust and University of Manchester, UK; ⁹Gustave Roussy Cancer Center, INSERM U981, and Groupe d'Investigateurs Nationaux pour l'Etude des Cancers Ovariens (GINECO), France; ¹⁰AdventHealth Cancer Institute Orlando, USA; ¹¹Oncology Center of Galicia, Spain; ¹²Auckland City Hospital and University of Auckland, New Zealand; ¹³Royal Brisbane and Women's Hospital, Herston and University of Queensland, Australia; ¹⁴The Ohio State University, James Cancer Center, USA; ¹⁵Johns Hopkins University School of Medicine, USA; ¹⁶The Royal Marsden NHS Foundation Trust and Institute of Cancer Research, UK; ¹⁷HM Hospitales—Centro Integral Oncológico Hospital de Madrid Clara Campal, Spain; ¹⁸University of Washington, USA; ¹⁹Clovis Oncology UK Ltd., UK; ²⁰Clovis Oncology, Inc., USA; ²¹US Oncology Research, USA; ²²UCL Cancer Institute, University College London and UCL Hospitals, UK

10.1136/ijgc-2020-IGCS.3

Abstract 3 Table 1

	BRCA	1	BRCA2			
	Rucaparib (n=80)	Placebo (n=37)	Rucaparib (n=50)	Placebo (n=29)		
TFST						
Median, mo	16.8	8.1	30.4	7.1		
HR (95% CI)	0.41 (0.27-	-0.64)	0.17 (0.09–0.33)			
CFI						
Median, mo	18.4	9.4	36.1	8.7		
HR (95% CI)	0.40 (0.26-	-0.62)	0.16 (0.08–0.32)			
PFS2	•					
Median, mo	25.1	21.8	34.1	18.4		
HR (95% CI)	0.84 (0.53-	-1.32)	0.51 (0.29–0.91)			
TSST			M			
Median, mo	25.9	18.5	34.2	19.4		
HR (95% CI)	0.65 (0.41-	-1.04)	0.55 (0.31–0.96)			

Visit cutoff date: 31 Dec 2019

CFI, chemotherapy-free interval; CI, confidence interval; HR, hazard ratio; PFS2, time to disease progression on subsequent therapy or death; TFST, time to first subsequent therapy; TSST, time to second subsequent therapy.

Introduction In ARIEL3 (NCT01968213), rucaparib maintenance for recurrent ovarian cancer (rOC) significantly improved investigator-assessed PFS and postprogression efficacy outcomes versus placebo regardless of biomarker status. PFS was also improved in patients with rOC associated with either BRCA1 or BRCA2 mutations (HR, 0.32 [95% CI, 0.19–0.53] and 0.12 [0.06–0.26], respectively). This exploratory analysis further examined the subgroup of patients with rOC associated with BRCA1 or BRCA2 mutations to assess the durability of the clinical benefit of rucaparib maintenance following disease progression.

Methods Patients were randomised 2:1 to oral rucaparib (600 mg twice daily) or placebo. Postprogression efficacy endpoints were assessed in patients with germline or somatic BRCA1 or BRCA2 mutations.

Results Investigator-assessed postprogression efficacy endpoints for patients with either BRCA1 or BRCA2 mutations are presented in the table 1.

There was a trend for better outcomes across all endpoints in patients with BRCA1 and BRCA2 mutations, with larger differences between the median values among patients with a BRCA2 mutation. The treatment-by-mutation group interaction test reached statistical significance for TFST and CFI.

Among rucaparib-treated patients, the most common treatment-emergent adverse events (any grade) in the BRCA1 and BRCA2 subgroups were nausea (81.0% and 78.0%) and asthenia/fatigue (74.7% and 80.0%).

Conclusions/Implications All postprogression efficacy endpoints were longer with rucaparib maintenance than with placebo in both BRCA-mutant subgroups. Safety data for the two subgroups were similar and were consistent with the overall safety population.

Plenary II

IGCS20_1447

4 REFINING PATHOLOGIC INTERPRETATION OF ENDOMETRIAL CARCINOMAS: LESSONS LEARNED FROM A NATIONWIDE STUDY IN A NEW ERA OF MOLECULAR CLASSIFICATION

¹E Thompson*, ¹J Huvila, ²S Leung, ³J Irving, ³N van der Westhuizen, ⁴M Kinloch, ⁵A Lytwyn, ⁵M Sur, ⁶C Parra-Herran, ⁷A Yasmeen, ⁸F Gougeon, ⁹C Morin, ⁹K Grondin, ¹⁰S Offman, ¹¹T Salisbury, ¹²E He, ¹²J Lawson, ¹³J Vanden Broek, ¹⁴C Bell, ⁹K Ennour-Idrissi, ¹⁵C Wohlmuth, ¹⁶D Vicus, ¹⁶D Vicus, ¹⁷W Gotlieb, ¹⁸L Helpman, ¹A Lum, ¹J Senz, ¹D Huntsman, ¹¹B Gilks, ¹⁹JN McAlpine. ¹Molecular Oncology, University of British Columbia, Canada; ²Genetic Pathology Evaluation Centre, University of British Columbia, Canada; ³Pathology and Laboratory Medicine, Royal Jubilee Hospital and the University of British Columbia, Canada; ⁴Pathology and Laboratory Medicine, University of Saskatchewan, Canada: ⁵Pathology and Molecular Medicine, McMaster University, Canada: ⁶Laboratory Medicine and Pathobiology, University of Toronto and Sunnybrook Health Sciences Centre, Canada; ⁷Gynecologic Oncology, Segal Cancer Center, Jewish General Hospital, McGill University, Canada; ⁸Department of Pathology, University of Montreal, Canada; ⁹Pathology Department, Centre Hospitalier Universitaire de Québec, L'Hôtel-Dieu de Québec, Laval University, Canada; ¹⁰Anatomical Pathology, Queen Elizabeth II Health Sciences Centre and Dalhousie University, Canada; ¹¹Pathology and Laboratory Medicine, Vancouver General Hospital and University of British Columbia, Canada; ¹²MD Undergraduate Program, University of British Columbia, Canada; ¹³Faculty of Science, University of British Columbia, Canada; ¹⁴College of Medicine, University of Saskatchewan, Canada; ¹⁵Gynecologic Oncology, Department of Surgical Oncology, University Health Network, Canada; ¹⁶Gynecologic Oncology, Centre Hospitalier Universitaire de Québec, L'Hôtel-Dieu de Québec, Canada; ¹⁷Gynecologic Oncology, Jewish General Hospital, McGill University, Canada; ¹⁸Gynecologic Oncology, Juravinski Cancer Center and McMaster University, Canada; ¹⁹Gynecologic Oncology, Vancouver General Hospital and the University of British Columbia, Canada

10.1136/ijgc-2020-IGCS.4

Abstract 4 Table 1	Univariable association	of clinicopathologic	characteristics by	/ proactive	molecular	risk	classifier	for	endometrial	cancer
(ProMisE) subtype										

Variable	Total		POLE		I	MMR		NSMP/p53wt		53abn	p value
Total		862	55	(6.4%)	247	(28.7%)	387	(44.9%)	173	(20.1%)	
Age at dx											<0.001
≤60	321	(37.2%)	35	(63.6%)	78	(31.6%)	181	(46.8%)	27	(15.6%)	
>60	541	(62.8%)	20	(36.4%)	169	(68.4%)	206	(53.2%)	146	(84.4%)	
BMI ≥30	396	(45.9%)	20	(36.4%)	117	(47.4%)	180	(46.5%)	79	(45.7%)	0.004
Histotype											<0.001
Endometrioid	681	(79.0%)	49	(89.1%)	231	(93.5%)	371	(95.9%)	30	(17.3%)	
LG	584	(67.7%)	38	(69.1%)	186	(75.3%)	349	(90.2%)	11	(6.4%)	
HG	97	(11.3%)	11	(20.0%)	45	(18.2%)	22	(5.7%)	19	(11.0%)	
Non endometrioid	183	(21.2%)	6	(10.9%)	16	(6.5%)	16	(4.1%)	143	(82.7%)	
FIGO stage											<0.001
1	635	73.7%)	44	(80.0%)	188	(76.1%)	315	(81.4%)	88	(50.9%)	
II-IV	198	(23.0%)	11	(20.0%)	52	(21.1%)	56	(14.5%)	79	(45.7%)	
LVI											<0.001
positive	274	(31.8%)	16	(29.1%)	101	(40.9%)	79	(20.4%)	78	(45.1%)	
negative	550	(63.8%)	38	(69.1%)	135	(54.7%)	288	(74.4%)	89	(51.4%)	
LN sampling											
performed	E10	(60.2%)	26	(CE E9/)	155	(62.89/)	170	(46.20/)	140	(96 19/)	
res (any)	213	(60.2%)	30	(05.5%)	155	(02.8%)	1/9	(40.5%)	149	(00.1%)	<0.001
	02	(10.9%)	E	(0.1%)	22	(9.0%)	22	(5 7%)		(25.4%)	VU.UUI
yes Dect surgical	93	(10.8%)	3	(9.1%)	22	(0.9%)	22	(5.7%)	44	(25.4%)	-0.001
Treatment											<0.001
yes	374	(43.4%)	19	(34.5%)	122	(49.4%)	110	(28.4%)	123	(71.1%)	