Furthermore, the standardization of nomenclature allows an easy exchange of surgical information for scientific purposes, that are otherwise difficult to interpret and compare.

414 SURGERY FOR MALIGNANT OVARIAN GERM CELL TUMOURS: A MULTICENTRE RETROSPECTIVE COHORT STUDY

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Introduction/Background* Malignant ovarian germ cell tumours (MOGCTs) are rare with a yearly-adjusted incidence of 3.7 per million [1] and account for 1-2% of all ovarian malignancies in Europe. There is a clinical imperative to clarify the optimal surgical approach and establish surgical radicality since this is a predominantly young population and minimising treatment morbidity and optimising future fertility is of real importance. Here we aim to describe the current surgical management of ovarian germ cell tumours and relate this to clinical outcome. Specifically, we aimed to compare outcomes of open versus laparoscopic surgery, the use of fertility-sparing approaches, surgical staging, and the potential utility of cystectomy alone in the management of patients with stage 1 immature teratoma.

Methodology A retrospective cohort study of all consecutive patients with primary ovarian germ cell tumours treated in four major UK gynaecology oncology centres over 12 years.

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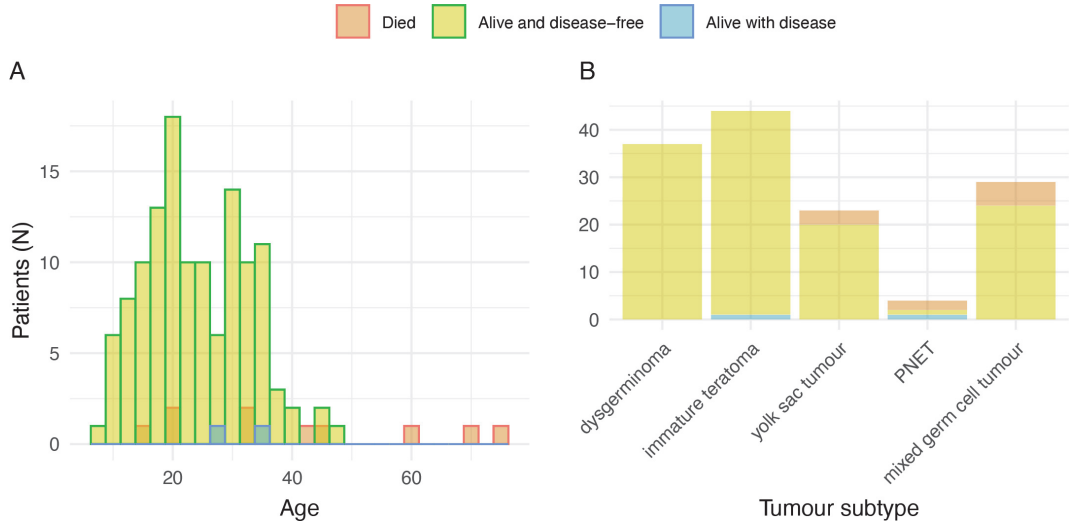
	All pathologies	Dysgerminoma	Yolk sac tumour	Mixed germ cell tumour	Immature teratoma	Primitive neuroectodermal tumour
Total, N (%)	137 (100.0)	37 (27.0)	23 (16.8)	29 (21.1)	44 (32.1)	4 (2.9)
FIGO 2014 stage						
Stage 1	86 (62.3)	24 (64.9)	13 (56.5)	10 (34.5)	39 (88.6)	0 (0.0)
Stage 2	11 (8.0)	2 (5.4)	2 (8.7)	6 (20.7)	0 (0.0)	1 (25.0)
Stage 3	23 (16.7)	6 (16.2)	7 (30.4)	7 (24.1)	1 (2.3)	2 (50.0)
Stage 4	15 (10.9)	4 (10.8)	1 (4.3)	6 (20.7)	3 (6.8)	1 (25.0)
Age						
Median (IQR)	23 (14)	21 (10)	27 (13)	23 (14)	26 (15.5)	23 (11.75)
< 18, N (%)	31 (22.6)	10 (27.0)	3 (13.0)	3 (10.3)	14 (31.8)	1 (25.0)
> 18, N (%)	106 (77.3)	27 (73.0)	20 (87.0)	26 (89.7)	30 (68.2)	3 (75.0)
Surgical route						
Laparotomy	109 (80.0)	23 (62.1)	21 (91.3)	25 (86.2)	36 (81.8)	4 (100.0)
Laparoscopy	22 (16.0)	11 (8.0)	2 (8.7)	2 (6.9)	7 (5.1)	0 (0.0)
Surgery type						
Fertility sparing	120 (87.6)	31 (83.8)	22 (95.7)	24 (82.8)	40 (90.9)	3 (75.0)
Non-fertility sparing	16 (11.7)	6 (16.2)	1 (8.7)	4 (13.8)	4 (9.1)	1 (25.0)
Primary debulking	10	1	1	4	1	1
Interval debulking	5	2	0	0	3	0
Prophylactic surgery	3	3	0	0	0	0
Chemotherapy						
None	61 (44.5)	15 (40.5)	4 (17.4)	9 (31.0)	33 (75.0)	0 (0.0)
Neoadjuvant	16 (11.7)	7 (18.9)	2 (8.7)	2 (6.9)	4 (9.1)	1 (25.0)
Adjuvant	60 (43.8)	15 (40.5)	17 (73.9)	18 (62.1)	7 (15.9)	3 (75.0)
Residual disease						
none	112 (81.8)	35 (94.6)	14 (60.9)	21 (72.4)	39 (88.6)	3 (75.0)
<1cm	4 (2.9)	0 (0.0)	2 (8.7)	1 (3.4)	1 (2.3)	0 (0.0)
>1cm	20 (14.6)	2 (5.4)	7 (30.4)	6 (20.7)	4 (9.1)	1 (25.0)
Completion surgery						
performed	11 (8.0)	1 (2.7)	4 (17.4)	2 (6.9)	4 (9.1)	0 (0.0)
not performed	124 (90.5)	36 (97.3)	19 (82.6)	26 (89.7)	39 (88.6)	4 (100.0)
Recurrence	39 (28.5)	3 (8.1)	8 (34.8)	15 (51.7)	9 (20.5)	4 (100.0)
Time to recurrence (days)						
median (IQR)	211 (249)	363 (1398.5)	153 (296.25)	174 (126.5)	212 (208)	367.5 (368.75)
Censor outcome						
Dead	10 (7.3)	0 (0.0)	3 (13.0)	5 (17.2)	0 (0.0)	2 (50.0)
Alive with disease	3 (2.2)	0 (0.0)	1 (0.0)	0 (0.0)	1 (2.2)	1 (25.0)
Alive and disease free	127 (17.5)	37 (100.0)	20 (87.0)	24 (82.8)	43 (97.7)	1 (25.0)
Time to censor outcome (years)						
median (IQR)	4.6 (4.6)	6.0 (3.2)	4.9 (4.6)	4.9 (5.2)	3.2 (3.4)	1.3 (1.6)



Abstract 414 Figure 1

Result(s)* One hundred and thirty-seven patients were followed-up for a median of 4.6 years with a 5 year survival of 91% (95% CI 86-97%).

Open procedures were likely to be performed for larger (p<0.001) and higher stage tumours (p<0.049), but there was no significant difference in mortality between open (80% n=109) and laparoscopic approaches (16%,n=22).

Assessing the use of surgical staging in our cohort, peritoneal or omental biopsies were infrequently taken (29%, n=40) and were largely negative, returning positive results in 0% of peritoneal and 4% of omental biopsies.

In patients with stage one immature teratoma, outcomes of unilateral cystectomy only (n=9) and unilateral salpingo-oophorectomy (n=29) were compared, with no significant difference in death, recurrence rates or residual disease prevalence between the groups.

The majority (88%, n=120) of patients had fertility-sparing surgery. This was not associated with higher rates of recurrence or death than non fertility-sparing approaches.

Conclusion* Laparoscopic surgery was safe and since routine staging biopsies did not alter outcome, we suggest that their use should be limited. Ovarian cystectomy may be acceptable for early-stage immature teratoma and warrants replication in other cohorts.

415 SINGLE-CELL MAP OF THE DYNAMIC CHANGES UNDERLYING PLATINUM-BASED CHEMOTHERAPY WITH OR WITHOUT BEVACIZUMAB IN HIGH-GRADE SEROUS TUBO-OVARIAN CARCINOMA

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Introduction/Background* The vascular endothelial growth factor (VEGF) plays an import role in emergence and spread of high-grade serous tubo-ovarian carcinoma (HGSTOC). Bevacizumab, a monoclonal antibody targeting VEGFA, has therefore been added to first-line treatment of advanced HGSTOC. We here map the dynamics of different stromal components of the tumour microenvironment under chemotherapy with or without bevacizumab.

Methodology We performed single-cell RNA-sequencing on 62,461 cells sampled from 6 HGSTOC patients before and after neo-adjuvant platin-based chemotherapy with or without bevacizumab. We identified 44 stromal cell subclusters on which we applied Mixed-effects modelling of Associations of Single Cells to identify cell populations associated with bevacizumab exposure and pathological response using the chemotherapeutic response score.

Result(s)* Our study revealed diverse stromal cell subsets associated with bevacizumab exposure. The addition of bevacizumab to frontline chemotherapy increased the odds of endothelial cell (ECs) prevalence by a 3-fold (OR 2.91, 95% CI:2.36-3.58; p<0.001) in comparison to 13-fold when treated with only chemotherapy (OR 13.42, 95%CI:11.29-15.52; p<0.001). Especially for tip cells, essential for vessel sprouting, a negative odds ratio was found for its association with bevacizumab exposure (OR 0.32, 95%CI:0.21-0.50; p<0.001) while chemotherapy only increased the odds of tip cell recruitment (OR 2.67, 95%CI:1.87-3.80; p<0.001). Tip cell receptors KDR, FLT1 and co-receptor NRP1 were significantly downregulated after bevacizumab exposure. In addition, ECs treated with bevacizumab showed lower scores for hypoxia signatures and demonstrated a significant downregulation of hypoxia-induced genes, including HIF1α. Furthermore, bevacizumab was associated with decreased number of regulatory T cells (OR 0.40, 95%CI:0.32-0.51; p<0.001). Interestingly, the addition of bevacizumab was associated with increased influx of tumour-associated macrophages (TAMs). Especially, PDGFC-expressing TAMs were strongly associated with bevacizumab exposure (OR 21.33, 95%CI:9.01-50.46; p<0.001) and poor response (OR 49.06, 95%CI:18.46-130.39 p<0.001). These cells express platelet-derived growth factor-C (PDGFC) and NRP1 providing a possible escape mechanism to activate KDR in absence of VEGFA.

Conclusion* We here provide initial evidence on the mechanisms underlying early response to bevacizumab and frontline chemotherapy in HGSTOC, including tip cell impairment,