American society of clinical oncology 2011 annual meeting update: Summary of selected gynecologic cancer abstracts

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Meeting Report

American Society of Clinical Oncology 2011 Annual Meeting Update: Summary of Selected Gynecologic Cancer Abstracts

Introduction

The 2011 Annual Meeting of the American Society of Clinical Oncology (ASCO) was held in Chicago, June 3–7, 2011 and focused on “Patients, Pathways, Progress”. 40,000 cancer specialists from around the world gathered to discuss the latest innovations in research, quality, practice, and technology. Over 100 studies in gynecologic cancer were presented, including novel therapeutic approaches in not only ovarian cancer, but also in endometrial and cervical cancers. This report will highlight phase III randomized trials in ovarian cancer and other selected studies.

Phase II trials in locally advanced and metastatic/recurrent cervical cancer

Several studies examined the efficacy and tolerability of combining anti-EGF-based therapy to chemoradiation and gene therapy to reconstitute wild type p53 function for locally advanced cervical carcinoma (Table 1). Erlotinib, a tyrosine kinase EGFR inhibitor, yielded no objective responses when studied previously by the GOG in women with recurrent disease. However, Rodrigues et al. combined erlotinib 150 mg/d with chemoradiation plus brachytherapy and reported a 94.4% CR, with 3-yr OS 80%, and g3 skin rash in 13%. Two additional phase II trials in locally advanced tumors studied weekly intra-tumoral recombinant adenoviral human p53 (rAd-p53) gene therapy in conjunction with pelvic radiation. In a randomized phase II trial, the overall response rate (ORR) of pelvic RT with and without gene therapy (1–4 × 10^12 rAD-p53 viral particles × 6 weeks) was 100% vs 72.2%, respectively (p = 0.0149). Anticipated side effects reported in both studies included transient fever. For metastatic disease, the second generation platinum doublet nedaplatin plus paclitaxel was associated with a 42.2% ORR and median OS of 8 mos.

Phase II trials of mammalian target of rapamycin inhibitors (mTORi) in endometrial cancer

Loss of phosphatase and tensin homolog (PTEN) protein function occurs in 26–83% of endometrial carcinomas leading to deregulation of the PI3K/AKT/mTOR signaling. Four phase II studies evaluated the response, survival and toxicity in advanced and recurrent disease (Table 2). Two randomized phase II trials studied mTORi(s) versus hormonal therapy. In one study, patients with unresectable disease were randomized to oral ridaforolimus 40 mg for 5 days/week versus medroxyprogesterone 200 mg/d or megestrol 60 mg/d. Interim analysis of the first 114 patients treated demonstrated a median PFS of 36 mos for ridaforolimus and 1.9 mos for progestin therapy (HR 0.53, p = 0.008) with grade 3/4 AEs of hyperglycemia (19%) and anemia (9%). The second study (intravenous tensirolimus vs tensirolimus plus megestrol acetate alternating with tamoxifen) was closed due to an unacceptable rate of venous thromboses in the combined regimen. A non-randomized phase II study of daily everolimus plus letrozole was associated with an objective RR of 21%.

Phase II trials of anti-angiogenesis agents in ovarian cancer

Agents that target the angiogenic pathway continue to generate interest in ovarian cancer (Table 3). Simultaneous targeting of the MET and VEGF signaling pathways with cabozantinib was reported for recurrent disease. Randomization was halted and patients were unblinded based on an observed high rate of clinical activity, including an ORR 24%. In two non-randomized phase II trials, bevacizumab was studied with pegylated liposomal doxorubicin (PLD) plus carboplatin (PLD 30 mg/m2 and carboplatin (AUC5) day 1 plus bevacizumab 10 mg/kg on days 1 and 15 every 28 days) in platinum sensitive disease and with the VEGFR2/Raf kinase inhibitor, sorafenib, in bevacizumab-naïve patients with recurrence (sorafenib 200 mg twice daily with bevacizumab 5 mg/kg every 2 weeks every 28 days). In the former trial the ORR was 72.2% with a median PFS of 14 mos. Thirty-nine patients (72.2%) discontinued therapy due to an adverse event. In the latter study, 24% PR lasting a median 15.5 mos was noted with hypertension (47%) and thrombosis (13%) among the AEs. The combination of docetaxel plus afibertcept for recurrence resulted in an ORR of 54% (77% PltS, 45% PltR; 10 Crs; PFS 6.2 mos, OS 24.3 mos). Neutropenia (72%), fatigue (50%) and dyspnea (22%) represented grade 3/4 toxicities.

Phase II trials of poly(ADP-ribose) polymerase inhibitors (PARPi) in ovarian cancer

Olaparib is an oral PARPi that is active in high grade serous ovarian cancer with and without BRCA1 and BRCA2 mutations (Table 3). Maintenance olaparib (400 mg twice daily) was studied in a randomized placebo-controlled phase II trial in platinum sensitive...
patients in sustained partial or complete response. When the pre-determined 153 progression events (58%) had occurred, PFS was significantly longer in the olaparib than placebo group (HR 0.35).

Despite promising phase II data indicating efficacity and tolerability of the intravenous PARPi, iniparib, in triple-negative breast cancer (TNBC), the Phase III registration trial ground to a halt when it was reported at this year’s meeting that the combination of iniparib plus carboplatin and gemcitabine failed to meet its co-primary endpoints of PFS and OS in TNBC. In two non-randomized phase II studies of the iniparib–carboplatin–gemcitabine triplet in platinum sensitive and platinum resistant ovarian carcinoma, the ORRs were 70.6% and 31.6%, respectively (Table 3). In the platinum resistant population, the median PFS was 5.9 mos.

**Phase II trials of other novel agents in ovarian cancer**

The PRECEDENT trial was an international, open-label, randomized phase II study comparing pegylated liposomal doxorubicin (PLD) plus the folic acid/desacetylvinblastine hydrazide conjugate, EC145, to PLD alone in women with platinum resistant disease (Table 3). Patients were randomized 2:1 to PLD (50 mg/m² IV q 28 days) with and without EC145 (2.5 mg IV weeks 1 and 3). Folic receptor (FR) status was determined prior to randomization using technetium-labeled EC20, an FR targeted imaging agent. In the intent-to-treat population of patients with measureable disease, EC145 plus PLD was found to be the first combination to show a statistically significant impact on PFS over standard therapy in women with platinum resistant disease (HR 0.626). For patients with 100% EC20 positive tumors, the PFS was 24.0 wks (investiga-
tional arm) and 6.6 wks (control) (HR 0.381). A phase III randomized trial is pending activation.

The hypomethylating agent, decitabine, was used with carboplatin to reverse acquired platinum resistance (Table 3). The ORR was 35% and included 1 CR and 5 PRs (median PFS 309 days). Grade 3/4 hematologic AEs in all principal cell lineages occurred in 11 patients in sustained partial or complete response. When the pre-determined 153 progression events (58%) had occurred, PFS was significantly longer in the olaparib than placebo group (HR 0.35).

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The hypomethylating agent, decitabine, was used with carboplatin to reverse acquired platinum resistance (Table 3). The ORR was 35% and included 1 CR and 5 PRs (median PFS 309 days). Grade 3/4 hematologic AEs in all principal cell lineages occurred in 11–23%. Demethylation of ovarian cancer-associated genes, MLH1, RASSF1a, HOXA10, and HOXA11 in tumors from day 1 to day 8 positively correlated with PFS (p<0.05). This trial constitutes the first-in-class proof of concept that epigenetic intervention can restore platinum sensitivity in ovarian cancer.

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**Table 1**

Novel drugs and treatment strategies for locally advanced and recurrent cervical carcinoma.

<table>
<thead>
<tr>
<th>Type of trial</th>
<th>Abs #</th>
<th>Agents/dose</th>
<th>Mechanism</th>
<th>Type of patients</th>
<th>Results</th>
<th>P Value</th>
<th>Major toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Randomized phase II</td>
<td>5097</td>
<td>Weekly intra-tumoral rAd-p53 plus pelvic RT + HDR BT</td>
<td>Gene therapy</td>
<td>Locally advanced cervix (IIB–IVA)</td>
<td>ORR 100 v 72.2%*</td>
<td>*=0.01</td>
<td>Fever</td>
</tr>
<tr>
<td>Phase II combinations</td>
<td>5033</td>
<td>Erlotinib plus CDDP and pelvic RT + BT</td>
<td>Oral TKI (EGFR); cytotoxic</td>
<td>Locally advanced SCCA cervix (IIB–IIIB)</td>
<td>94.4% CR</td>
<td>5.6% PR</td>
<td>Skin rash</td>
</tr>
<tr>
<td>5038</td>
<td>IFX-Mesna plus carboplatin and pelvic RT followed by RHBPLND vs BT (non-randomized)</td>
<td>Cytotoxic, “down-staging” to assess operability</td>
<td>Locally advanced cervix (IIB, IIIB)</td>
<td>ORR 89.7%</td>
<td>CR 35.9%</td>
<td>Leukopenia PLTS An</td>
<td></td>
</tr>
<tr>
<td>5102</td>
<td>Paclitaxel plus nedaplatin</td>
<td>Second generation platinum doublet</td>
<td>Metastatic/recurrent cervix</td>
<td>ORR 42.2%</td>
<td>CR 22%</td>
<td>ANC, An, elevated serum creatinine, alopecia</td>
<td></td>
</tr>
<tr>
<td>Phase II single agents</td>
<td>5096</td>
<td>Weekly intra-tumoral rAd-p53 plus pelvic RT + BT</td>
<td>Gene therapy</td>
<td>Locally advanced cervix (IIB, IIIB)</td>
<td>100% CR</td>
<td>5YS 85.7%</td>
<td>Transient fever</td>
</tr>
</tbody>
</table>

rAd-p53 = recombinant adenooviral human p53 gene, CDDP = cisplatin, RT = radiotherapy, HDR = high dose rate, BT = brachytherapy, TKI = tyrosine kinase inhibitor, EGFR = epidermal growth factor receptor, IFX = ifosfamide, RHBPLND = radical hysterectomy with bilateral pelvic lymph node dissection, ANC = neutropenia, An = anemia, PLTS = thromboctopenia.

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**Table 2**

Phase II trials of mammalian target of rapamycin inhibitors (mTORi) in endometrial carcinoma.

<table>
<thead>
<tr>
<th>Type of trial</th>
<th>Abs #</th>
<th>Agents/dose</th>
<th>Mechanism</th>
<th>Type of patients</th>
<th>Results</th>
<th>HR</th>
<th>P Value</th>
<th>Major toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Randomized phase II</td>
<td>5009</td>
<td>Daily ridaforolimus vs progestin or chemorX</td>
<td>mTORi</td>
<td>Advanced/metastatic endometrium</td>
<td>PFS: 3.6 vs 1.9 mos</td>
<td>HR = 0.53</td>
<td>P = 0.008</td>
<td>An, h, d, s, back pain, anemia, Combined regimen closed due to unacceptable rate of venous thrombosis</td>
</tr>
<tr>
<td>5014</td>
<td>Tensirolimus vs tensirolimus plus megestrol acetate alternating with tamoxifen</td>
<td>mTORi, Progestin–and estrogen-based therapy</td>
<td>Advanced/recurrent endometrium</td>
<td>At least 4 responses (single agent tensirolimus, 2nd stage accrual opened)</td>
<td>–</td>
<td>–</td>
<td>F, n, s, h, hyperglycemia, Objective RR 21%</td>
<td></td>
</tr>
<tr>
<td>Phase II combinations</td>
<td>5012</td>
<td>Daily everolimus plus letrozole</td>
<td>mTORi, aromatase inhibition</td>
<td>Recurrent endometrium</td>
<td>CBR 42%</td>
<td>Objective RR 21%</td>
<td>PR 7%</td>
<td></td>
</tr>
<tr>
<td>Phase II single agents</td>
<td>5013</td>
<td>Daily ridaforolimus</td>
<td>mTORi</td>
<td>Recurrent endometrium</td>
<td>–</td>
<td>–</td>
<td>F, n, s, h, hyperglycemia, Mucositis, f, n, d, anorexia, taste alteration, rash</td>
<td></td>
</tr>
</tbody>
</table>

mTORi = mammalian target of rapamycin inhibitor, n = nausea, f = fatigue, An = anemia, d = diarrhea, h = hyperglycemia, s = stomatitis.
**Eribulin mesylate is a tubulin inhibitor distinct from taxanes. It suppresses microtubule growth without affecting depolymerization, resulting in sequestration of tubulin into non-functional aggregates. A platinum sensitive population was treated with eribulin 1.4 mg/m² intravenously on days 1 and 8 every 21 days.**

**Phase III randomized trials in ovarian cancer**

This year’s ASCO meeting was disappointing as only one “positive” randomized phase III trial was reported. However, four important updates of previously reported phase III studies were presented in poster form and a large trial of maintenance immunotherapy was negative.

The OCEANS trial investigated the ability of bevacizumab to prolong PFS when added to approved doses and schedules of gemcitabine and carboplatin in treating second-line platinum sensitive recurrent ovarian cancer (Abstract LBA 5007). Study subjects that received bevacizumab during and after chemotherapy were 52% less likely to have progression of their disease than were patients given placebo (median PFS 12.4 mos with bevacizumab vs 8.4 mos placebo). Results also showed that patients in the bevacizumab group had a comparatively higher ORR (78.5% vs. 57.4%, \(p<0.0001\)) and longer duration of response (10.4 vs. 7.4 months, \(P<0.0001\)). No new safety concerns were identified. Importantly, the survival data from this trial are not yet mature and quality of life was not measured.

Updates of two front-line bevacizumab studies were also presented. The results of an independent radiologic review of GOG218 confirmed the results presented at last year’s ASCO meeting where the addition of 15 mg/m² of bevacizumab every 3 weeks during both chemotherapy and maintenance drug alone (total duration =15 months) prolonged PFS by 6 months (13.1 for arm 1 versus 19.1 for arm 3, HR=0.63, \(P<0.0001\)) (Abstract 5023). An update of a second front-line study using bevacizumab for 12 months at 7.5 mg/m² (ICON7) showed that it prolonged survival in the highest risk group of patients defined as those having large volume residual disease and stage IV (HR 0.64, 95% CI 0.48–0.85, \(p=0.002\)) (Abstract LBA 5006). This subset analysis was mandated by European regulators and survival data from the study as a whole are not yet mature.

Clearly, survival is a higher bar than response rate and time to progression when evaluating new agents and combinations. Thus, mature survival data were presented from the CALYPSO study (Abstract 5052) and OVA301 (Abstract 5046). Interestingly, the former study showed no improvement in survival in treating platinum sensitive recurrent ovarian cancer with carboplatin and PLD compared to carboplatin and paclitaxel (Median 31.5 months, 21.7 vs 19.1, HR=0.63, \(p=0.002\)). Updates of previously reported phase III studies were presented in poster form and a large trial of maintenance immunotherapy was negative.

**Table 3**

<table>
<thead>
<tr>
<th>Abs #</th>
<th>Agents</th>
<th>Mechanism</th>
<th>Type of patients</th>
<th>Results</th>
<th>HR</th>
<th>P Value</th>
<th>Major toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>5008</td>
<td>Daily cabozantinib vs placebo</td>
<td>Dual inhibition MET and VEGFR2</td>
<td>Primary ovary</td>
<td>PFS and PR ovary</td>
<td>ORR 24% (29%)</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>5017</td>
<td>Docetaxel &amp; afilbercept</td>
<td>Cytotoxic; high affinity binding for VEGF-A,-B, and PIGF</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>ORR 54%</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>5019</td>
<td>Sorafenib &amp; bevacizumab</td>
<td>VEGFR2/Raf kinase inhibition; anti-VEGF</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>CR 22%</td>
<td>SD 64%</td>
<td>–</td>
</tr>
<tr>
<td>5061</td>
<td>PLD &amp; carboplatin &amp; bevacizumab</td>
<td>Cytotoxic; anti-VEGF</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>ORR 72.2%</td>
<td>Median PFS 14 m</td>
<td>–</td>
</tr>
</tbody>
</table>

**PARP inhibitors**

<table>
<thead>
<tr>
<th>Abs #</th>
<th>Agents</th>
<th>Mechanism</th>
<th>Type of patients</th>
<th>Results</th>
<th>HR</th>
<th>P Value</th>
<th>Major toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>5003</td>
<td>Carboplatin plus bevacizumab &amp; iniparib q21d</td>
<td>Cytotoxic; PARPi</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>ORR 31.6%</td>
<td>PFS 5.9 mos</td>
<td>–</td>
</tr>
<tr>
<td>5004</td>
<td>Carboplatin plus gemcitabine &amp; iniparib q21d</td>
<td>Cytotoxic; PARPi</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>ORR 31.6%</td>
<td>PFS 5.9 mos</td>
<td>–</td>
</tr>
<tr>
<td>5005</td>
<td>Carboplatin plus gemcitabine &amp; iniparib q21d</td>
<td>Cytotoxic; PARPi</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>ORR 31.6%</td>
<td>PFS 5.9 mos</td>
<td>–</td>
</tr>
</tbody>
</table>

**Other novel agents**

<table>
<thead>
<tr>
<th>Abs #</th>
<th>Agents</th>
<th>Mechanism</th>
<th>Type of patients</th>
<th>Results</th>
<th>HR</th>
<th>P Value</th>
<th>Major toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>5010</td>
<td>Carboplatin plus paclitaxel with/iniparib</td>
<td>Cytotoxic; dual inhibition PKC(\beta)- and PI3/AKT</td>
<td>Primary ovary</td>
<td>PFS 18.9 vs 15.2 m</td>
<td>HR = 0.80</td>
<td>(P=0.37)</td>
<td>ANC, intestinal perforation ((n=1))</td>
</tr>
<tr>
<td>5045</td>
<td>EC145 plus PLD vs PLD</td>
<td>Follic acid conjugate; cytotoxic</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>PFS 21.7 vs 11.7 wk</td>
<td>HR = 0.626</td>
<td>(P=0.031)</td>
</tr>
<tr>
<td>5011</td>
<td>Carboplatin plus decitabine</td>
<td>Cytotoxic; hypomethylating agent</td>
<td>Phase II combinations</td>
<td>PRov</td>
<td>RR 35%</td>
<td>PFS</td>
<td>–</td>
</tr>
<tr>
<td>5090</td>
<td>Eribulin</td>
<td>Tubulin inhibitor</td>
<td>Phase II single agent</td>
<td>PRov</td>
<td>309 days</td>
<td>CR 19%</td>
<td>SD 54%</td>
</tr>
</tbody>
</table>

**References**

- Hypertension, d = diarrhea, HFS = hand foot syndrome.
- Neutropenia, An = anemia, PLTS = thrombocytopenia, PARPi = poly(ADP-ribose) polymerase inhibitor, n = nausea v = vomiting, f = fatigue, s = stomatitis, d = diarrhea, htn = hypertension, d = diabetes, HFS = hand foot syndrome.
HR = 0.987, P = .87) despite a statistically significant difference in PFS presented at ASCO 2009. However, the use of trabectedin and PLD prolonged survival compared to PLD alone in treating patients second line (adjusted HR = 0.82; 95% CI: 0.69, 0.98, P = 0.0285). The largest difference in survival was seen in the subset of women who recurred 6–12 months after front-line platinum based therapy.

Finally, in a disappointing placebo controlled maintenance trial of abagavomab, a murine monoclonal anti-idiotypic antibody directed against CA125, there was no difference in the primary endpoint of PFS among 888 enrolled subjects (P = 0.675) (Abstract LBA 5002).

Translational science in ovarian cancer

Gourley et al. studied microarray expression analysis in 363 formalin-fixed paraffin embedded epithelial ovarian cancer tissues with linked prospectively collected clinical data (Abstract 5000). The resulting molecular taxonomy contains clusters with differing survival with a serous subgroup defined by upregulation of multiple angiogenesis genes. A pro-angiogenic signature may explain the observed response and positive impact on PFS attributed to bevacizumab in GOG 218, ICON7, and most recently, OCEANS.

Lonning et al. analyzed WBC DNA from 899 ovarian cancer patients for BRCA1 promoter methylation and found a significantly increased risk of ovarian cancer in the cohort of patients with blood samples drawn at the time of (OR 4.765; CI 2.814–8.069) or prior to (OR 2.937; CI 1.476–5.845) diagnosis (Abstract 5029). The clinical implications suggest a potential role for hypomethylating agents, such as decitabine (Table 5), to restore wild-type gene function in patients at established genetic risk.

Human epididymis protein 4 (HE4) is the product of the WFDC2 (HE4) gene and is overexpressed in patients with ovarian carcinoma. Marinaccio et al. investigated the ability of HE4 to predict survival in 35 women with ovarian cancer (stage III, n = 28; stage 4, n = 7) (Abstract 5081). All patients with a HE4 >400 pM died within 2 years of diagnosis, while those with a reduced HE4 at both baseline and 3 months had the best overall survival.

Brief update

Alifrangis et al. from Charing Cross noted that OS for GTN following EMA/CO has improved significantly from 87% to 98%, partly due to the introduction of 2 cycles of low dose EP-induction chemotherapy (etoposide 100 mg/m² and cisplatin 20 mg/m² on days 1 and 2, repeated weekly x 2) before commencing EMA/CO (Abstract 5024). EP-induction given to high risk patients (FIGO score >8 and >6 metastases) was felt to minimize the risk of early deaths.

Conclusion

There are now three phase III randomized studies demonstrating a positive impact in PFS among women with advanced ovarian cancer who receive the anti-angiogenesis drug, bevacizumab. The latest study, OCEANS, demonstrated a 4-month improvement in PFS for women with essentially incurable platinum sensitive recurrent disease. The interim OS analysis from ICON 7 demonstrates improved survival associated with bevacizumab for frontline/maintenance treatment in a high risk subset, and the IRC of GOG 218 was consistent with the investigator’s assessment of response. The non-platinum chemotherapy doublet of PLD plus trabectedin can prolong survival. Based on recent phase II trials, PARPi(s) and mTORi(s) are emerging as drugs of interest in treating ovarian and endometrial cancer, respectively. Finally, the search for novel therapies for women with metastatic/recurrent cervical carcinoma continues.

Conflict of interest statement

Dr. Bradley Monk discloses that he has received research grants from GlaxoSmithKline, PharmaMar, Sanofi-Aventis, Merck and Novartis along with honoraria for speaker bureaus from GlaxoSmithKline, Roche and Johnson and Johnson. Additionally Dr. Monk has been a consultant for Qiagen, Roche, GlaxoSmithKline and Merck. Dr. Krishnansu Tewari discloses that he has received research grants from Precision Therapeutics, Amgen, Imclone, and Biogen Idec and honoraria for speakers bureaus from Genzyme, Vermillion, Qiagen, and Merck.

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