intratumoral heterogeneity with a minimal invasive approach and easy access. We aimed at assessing the feasibility and clinical utility of ctDNA genotyping for patients with advanced CCA in a group of referral Oncology centers across Spain.

Methods: We genotyped ctDNA from blood samples from patients with advanced CCA, treated between 2019 and 2021 at 9 Spanish University Hospitals. ctDNA sequencing was performed by an NGS-based comprehensive approach using Guardant360™ (G360).

Results: ctDNA sequencing data were available for 112 patients. The main clinical characteristics were as follow: median age was 62.5 years (range: 28-86), 62 patients (55.3%) were men, 65 patients (58%) had intrahepatic cholangiocarcinoma, 70 patients (62.5%) had stage IV disease at diagnosis and 98 patients (87.5%) had received cisplatin plus gemcitabine as first-line treatment for advanced disease. Determination of ctDNA was performed at baseline in 36.6% of patients and at the time of progression to first-line treatment in 48 patients (42.8%). 96% of patients had $\geq$ 1genomic alteration detected. Median number of alterations per patient was 2 and median VAF was 0.5%. 245 genomic alterations (GAs) have been identified among 35 different genes. 89% of GAs identified were SNVs, being the most frequent TP53 (33.9%), KRAS (9.8%), IDH1 (6.5%), ATM (6.5%), PIK3CA (4.9%) and ARID1A (4.5%). CNVs accounted for 7.7% of GA and consisted in amplifications of: EGFR (1.6%), MYC (1.6%), BRAF (1.2%) and ERBB2 (0.8%). FGFR2 and FGFR3 fusions were identified in 1.2% and 0.4% patients, respectively. High-microsatellite instability was identified in 1.2% of patients. These findings are similar to previously reported data on tissue molecular profiling in CCA. Importantly, sixty-seven patients (53%) had GA considered as actionable, including three patients with FGFR2 fusions, for which Pemigatinib is approved.

Conclusions: ctDNA genotyping for molecular profiling of advanced cholangiocarcinoma patients is feasible and a non-invasive procedure, especially relevant for CCA patients in which increase in actionable biomarkers and insufficient tissue availability is becoming a challenge.

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PD-11 In depth analysis of label-free infrared (IR) imaging-based microsatellite instability (MSI) classification in early colon cancer (CC) on samples from the AIO ColoPredictPlus 2.0 (CPP) registry trial

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Background: MSI occurs in 15-20% of all early CC due to a deficient mismatch repair system and testing is now considered standard for all colon cancers as MSI is of prognostic and predictive value. Presently, protein analysis using immunohistochemistry targeting the mismatch repair proteins is most frequently used in routine clinical pathology with high sensitivity. Alternatively, fragment length analysis of microsatellite loci or next-generation sequencing can be used. All the techniques referred to are time and tissue consuming. We utilize label-free quantum cascade laser (QCL) based IR imaging combined with artificial intelligence (AI) approaches to test unstained tissue samples for MSI. IR imaging is an emerging microscopic technique based on the interaction of electromagnetic waves with the molecules within the tissue creating molecular fingerprints. To establish the methodological robustness of our approach, we analyzed samples from the multicentric prospective AIO ColoPredict Plus (CPP) 2.0 registry trial.

Methods: IR imaging can analyze unstained paraffin-embedded tissue slides within an average of 30 min/slide. All tissue samples were obtained from the CPP registry trial. For development of the MSI/MSS classification model the cohort was split into a training, test, and validation set. The training set was utilized for the optimization of a modified VGG-16 convolutional neural network (CNN) with area under receiver operating characteristic (AUROC) and area under precision recall curve (AUPRC) evaluated on the test set as endpoints. The final classification model is validated on the validation set and in depth analyzed for sub-cohorts (BRAF, KRAS, UICC stage, grading).

Results: A cohort of 547 patients (training n=331 (43% MSI), test n=69 (43% MSI), validation n=147 (18% MSI)) was selected from CPP. Baseline characteristics including BRAF mutations, were balanced between training and test set, thus providing a balanced ground-truth for the training of the classifier. By contrast, the validation set (18% MSI) corresponds to the natural occurrence of MSI, showing a typically pattern for MSI and MSS with a higher proportion of female patients, more right-sided cases and more BRAF mutations for MSI-samples. The selected MSI/MSS classifier reached a validation AUROC of 0.90 (sensitivity 85%, specificity 84%) and AUPRC of 0.74. The AUROC of the sub-cohorts range between 0.78 and 0.99 (BRAF: 0.93 (wt), 0.78 (mt); KRAS: 0.84 (wt), 0.99 (mt); Grading: 0.93 (G2), 0.84 (G3); UICC Staging: 0.93 (II), 0.86 (III)).

Conclusions: AI integrated IR imaging demonstrates a reliable classification performance for MSI/MSS with an AUROC of 0.90 (sensitivity 85%, specificity 84%) and robustness over sub-cohorts e.g., BRAF, KRAS, or staging. It further approaches the performance of the present gold standard immunohistochemistry. The combination of spatial and biochemical information encoded in the IR images allows CNNs to track changes on the molecular level. Additionally, this method requires fewer samples than other AI approaches and maintains intact tissue for further molecular analysis. Overall, this method has the potential to become an applicable diagnostic tool beyond the scope of known biomarkers for tumor-agnostic assessments.

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PD-12 Characterization of best responder patients to oxaliplatin rechallenge in patients with refractory metastatic colorectal cancer (mCRC)

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Background: Oxaliplatin in combination with fluoropyrimidines constitute one of the most effective treatments in front-line mCRC patients. Rechallenge with oxaliplatin-based regimens in the refractory setting is practiced although based on scarce and heterogeneous evidence. Further clinical and molecular data to determine which patients do benefit from this treatment strategy are needed.

Methods: We analyzed all patients treated with oxaliplatin in a third- or fourth-line setting in our institution between 2015 and 2021. Outcomes were analyzed with overall response rate (ORR), disease control rate (DCR) and median progression free survival (mPFS). The best-responders group was defined-as those patients who achieved mPFS $>$ 6 months. As part of the descriptive analysis, we divided patients into three clinical groups according to the prognostic characteristics previously reported: Good Prognostic Characteristics (GPC) defined as having passed $\geq$18 months since metastatic disease debut, $<$ 3 metastatic sites and presence of liver metastasis, Best Prognostic Characteristics (BPC) defined as $\geq$18 months since metastatic disease debut, $<$ 3 metastatic sites and absence of liver metastases and Poor Prognostic Characteristics (PPC) defined as $<$ 18 months since metastatic disease debut and/or with $\geq$3 metastatic sites. An Amplicon-seq panel was used to analyze regions of interest in 61 genes using an Illumina sequencing platform.

Results: A total of 102 out of 735 mCRC patients (13,9%) were analyzed. Median age was 57.3 year and 55% were male. The outcomes were as follows: ORR 12%, DCR 39%, and mPFS 4.0 months (CI95% 3.29-5.03). Of note, 28 patients (27%) had a mPFS $>$ 6 months with rechallenge (range from 6.57 to 14.2 months) and constituted the best-responders group. Data of prognostic characteristics and molecular alterations are available for 19 of these patients. The prognostic subgroup classification was as follows: 9 patients (47.4%) PPC, 5 patients (26.3%) GPC, and 5 (26.3%) BPC. Most frequent molecular alterations were: APC (68.4%), TP53 (63%), RAS (47.4%), and PIK3CA (21%). Concomitant APC and TP53 mutations were detected in 47.4% of patients. No associations between prognostic characteristics and molecular alterations were observed.

Conclusions: This study suggests that rechallenge with oxaliplatin can achieve a clinically meaningful mPFS $>$ 6 months in 27% of patients. No enrichment in GPC and BPC was observed. APC, TP53 and RAS detected in this best-responders group are the major tumour genes which are frequently mutated in mCRC. A more extensive molecular analysis should be carried out to better characterise the patients who benefit the most from this treatment strategy.

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PD-13 Plasma RAS dynamics and efficacy of anti-EGFR rechallenge in patients with RAS/BRAF wild-type metastatic colorectal cancer: REMARRY and PURSUIT trials

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Background: Assessment of plasma RAS (pRAS) mutations in circulating-tumor DNA at 'just before' rechallenge with anti-EGFR monoclonal antibody (EGFR mAb) may predict efficacy for the rechallenge therapy in patients with RAS/BRAF V600E wild-type metastatic colorectal cancer (mCRC). However, the clinical impact of pRAS status at progression on prior EGFR mAb for the rechallenge therapy is unknown. The REMARRY trial is a prospective longitudinal study to investigate the pRAS dynamics, and PURSUIT trial is a phase II trial to investigate the efficacy of EGFR mAb rechallenge in patients with pRAS wild-type just before rechallenge therapy.

Methods: Eligibility criteria of REMARRY included RAS/BRAF V600E wild-type mCRC; ECOG PS 0-1; CR or PR during prior EGFR mAb; and progressed $\leq$ 2 months from the last administration of EGFR mAb. pRAS status by the BEAMing method (OncoBEAM RAS CRC Kit) was prospectively monitored at timepoints of progression on EGFR mAb and each subsequent therapy. Among participants of the REMARRY, patients who satisfied the following eligibility criteria were enrolled in PURSUIT; pRAS wild-type within 28 days prior to enrollment in PURSUIT; being refractory or intolerant to fluoropyrimidine, oxaliplatin, and irinotecan; and $\geq$ 4 months of EGFR mAb-free interval. Study treatment was rechallenge with panitumumab 6 mg/kg + irinotecan 150 mg/m2q2wks. Primary endpoint of PURSUIT was a confirmed objective response rate (ORR) according to RECIST v1.1. Biomarker analysis was performed for blood samples after disease progression on prior EGFR mAb, immediately prior to PURSUIT, and after disease progression in PURSUIT using plasma next generation sequencing (Guardant360). Plasma RAS, BRAF V600E, and EGFR extracellular domain mutations were defined as acquired resistances for EGFR mAb.

Results: Between May 2019 and May 2021, 183 patients were enrolled in REMARRY from 27 institutions, and 50 patients were enrolled in PURSUIT; median age, 68 years; left-sided primary, 44 patients; and prior EGFR mAb, 1st/2nd/$\geq$3rd lines in 28/6/16 patients. Confirmed ORR and disease control rate were 14% (90% CI, 7.8%—23.9%) and 80% (95% CI, 67.0%—88.8%), respectively. In addition, 4 patients showed an unconfirmed PR. Median progression-free survival (PFS) was 3.6 months (95% CI, 3.0—4.7 months). Among 31 patients with biomarker results after disease progression on prior EGFR mAb, ORR occurred in 5 of 21 patients (23.8%) with pRAS/BRAF/EGFR wild-type, whereas no responses occurred in patients whose tumors harbored any pRAS/BRAF/EGFR mutations (0/10) (p=0.092). Median PFS was 4.2 months and 2.8 months in patients without vs with pRAS/BRAF/EGFR mutations, respectively (p=0.06). In terms of pRAS/BRAF/EGFRstatus immediately prior to PURSUIT, 4 of 22 patients with wild-type responded to the study treatment (ORR, 18.2%), while one patient harboring pKRAS/EGFR co-mutations in 9 patients with any mutations also responded (ORR, 11.1%) (p=0.63). No trend in PFS was observed between the groups; median, 3.1 month and 3.3 months in patients with wild-type and those with any mutations (p=0.62).

Conclusions: Plasma RAS/BRAF/EGFR mutational status after progression on prior EGFR mAb may identify patients with RAS/BRAF V600E wild-type mCRC who could benefit from rechallenge with EGFR mAb.

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PD-14 Resectability, conversion, and resection rates with survival according to RAS and BRAF mutations in a prospective metastatic colorectal cancer study (liver-limited subgroup in the RAXO study)

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Background: RAS and BRAF mutations are associated with worse outcomes for patients with colorectal cancer (CRC) liver metastases, but little is known about their effects on resectability, conversion, and resection rates.