omitted because of compromised LEVF. Twenty six pts completed all protocol treatments (chemotherapy and surgery). Forty-one pts completed preoperative chemotherapy and underwent surgery. In 5 pts surgery was palliative, for unresectable or unsuspected metastatic disease. Reasons for not completing treatment were: disease progression (10 pts), postoperative complications (3 pts), prolonged neutropenia (1 pt), febrile neutropenia with septic shock and prolonged UCI stay (1 pt) and diverticulitis (1 pt). Dose reduction was required in 9 pts, due to diarrhea (3 pts), neutropenia (2 pts), neurotoxicity (1 pt), weight loss (1 pt), catheter complications (2 pt). Chemotherapy delay occurred in 23 pts (minimum 7 days − maximum 14 days), and the main causes were: delay in admission, neutropenia, diarrhea, and postoperative complication. With a medium follow up of 37 months, 7 pts died of progressive disease, two of them had completed full treatment.
Conclusion: Completion of all planned perioperative chemotherapy was observed in 71% of patients. Disease progression was the main reason to stop chemotherapy and although some pts were not able to receive post-operative treatment, tolerability was reasonable.

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Chromogranine a (CG-A) Plus Vascular Endothelial Growth Factor (VEGF) as Predicting Factors (PF) of Sorafenib (SFB) Treatment of Multifocal Hepatocellular Carcinoma (M-HCC) in Elderly Patients

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Background: To date HCC accounts for approximately 90% of all primary liver cancers, this is the fifth most common cancer in the world with very poor prognosis. Despite treatment M-HCC outcomes are very discouraging and only sorafenib one of new TKI has demonstrated better effectiveness. Furthermore, till now we can only monitoring therapy effectiveness and prognosis with CT-Scan or Liver-Sonography or MRI.
Based on preliminary findings, authors will investigate if Cg-A and VEGF work as predicting factors of Sorafenib treatment's outcomes.
Material and Methods: 27 patients, mean age 68.6 (65−85) with M-HCC were observed and enrolled in this study in the last 24 months. Serum Cg-A (Chromogranin A ELISA), VEGF (Human VEGF ELISA) and αFP were evaluated at baseline and after end of treatment. SFB was delivered at standard dose of 400 mg p.o. bid and no one patients discontinued treatment for HFS and G.I.tract toxicity. Clinical response (RECIST), Comprehensive Geriatric Assessment and PFS were considered as well.
Results: Cg-A mean value was: (baseline) 78.7±9.0 ng/ml (after treatment) 25.59±9.0 ng/ml. VEGF levels was: (baseline) 65.8±8.7 pg/ml (after treatment) 48.33±8.7 pg/ml. Hand-Foot Syndrome and G.I. tract toxicity (grade 3−4) was not observed in these pts. Clinical benefit correlated with lower values of CgA and VEGF.
Conclusions: Both Cg-A and VEGF seem to be in reciprocal relation with response to SFB therapy in elderly with M-HCC patients. Further, a correlation between CT-Scan, Sonography and MRI of liver with prognosis of M-HCC was shown. A large number of patients are enrolling to convalidate these findings.

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Correlation of PFS With Early Response of Chromogranin A and 5-hydroxyindoleacetic Acid Levels in Pts With Advanced Neuroendocrine Tumours: Phase III RADIANT-2 Study Results

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Background: In the phase III RADIANT-2 trial (NCT00412061; ESMO 2010 Abstract LBA8), everolimus 10 mg/d, an oral mTOR inhibitor, + octreotide LAR 30 mg IM q28 days (E+O) demonstrated a clinically meaningful 5.1-mo increase in median progression-free survival (PFS) compared with placebo + octreotide LAR (P+O) in pts with low- or intermediate-grade advanced NET and a history of flushing and diarrhea. Chromogranin A (CgA) and 5-hydroxyindoleacetic acid (5-HIAA) are important biomarkers of tumour burden and carcinoid syndrome in NET. Elevated vs nonelevated baseline CgA (11.3 vs 26.8 mo; HR, 0.45; $P < 0.001$) and 5-HIAA (13.6 vs 15.4 mo; HR, 0.79; $P = 0.130$) have been shown to be associated with shorter median PFS (WCGI 2011). This analysis examined the effect of baseline and early CgA and 5-HIAA response on PFS in the RADIANT-2 trial.
Materials and Methods: Pts with low- or intermediate-grade advanced NET were randomly assigned to E+O (n = 216) or P+O (n = 213). Primary endpoint was PFS (RECIST v1.0). Serum CgA and 24-h urinary 5-HIAA were collected at baseline and on day 1 of each cycle. Early CgA and 5-HIAA responses, defined as ⩾50% reductions at week 4, were correlated with PFS using a Cox-proportional hazards model.
Results: At baseline, 71.7% of E+O pts and 62.5% of P+O pts had elevated CgA (>2×ULN); elevated (>2×ULN) 5-HIAA was observed in 67.9% and 66.0%, respectively. The number of pts with early CgA (24.5% vs 16.5%) and 5-HIAA (24.0% vs 17.5%) responses were greater with E+O than P+O. Median PFS was significantly longer among pts with an early CgA response (27.3 vs 10.6 mo; HR, 0.35; $P < 0.001$) vs those without early response, regardless of treatment. Patients with early 5-HIAA response also had a longer median PFS than those without, but it did not reach statistical significance (18.3 vs 13.6 mo; HR, 0.71; $P = 0.139$).
Conclusions: In the large population of pts from the phase III RADIANT-2 trial, early CgA responses were associated with significantly improved PFS regardless of treatment, suggesting that early CgA response may serve as a surrogate for favorable PFS.
Study supported by Novartis.

Early response	Median PFS		Hazard Ratio	*P*
	E+O (N = 216)	P+O (N = 213)	E+O vs P+O	
CgA				
Yes	27.3 (13.9, NR) n = 34	27.8 (13.0, 30.4) n = 20	0.79 (0.4, 1.8)	0.283
No	13.7 (10.6, 16.1) n = 105	8.3 (5.9, 9.4) n = 101	0.54 (0.4, 0.8)	<0.001
5-HIAA				
Yes	18.3 (8.57, NR) n = 24	16.9 (8.4, 30.4) n = 18	0.81 (0.4, 1.8)	0.229
No	18.6 (13.6, 24.8) n = 76	8.4 (8.1, 13.6) n = 85	0.54 (0.3, 0.8)	0.003

NR, not reached.

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Androgens in the Etiology of Esophageal Adenocarcinoma − a Population-based Cohort Study on Prostate Cancer Patients in Sweden 1961 to 2008

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Background: The incidence of esophageal adenocarcinoma is continuing to rise in western populations. There is a major male predominance, unexplained by sex differences in the distribution of known risk factors. Androgens may play a role in the etiology of esophageal adenocarcinoma and anti-androgen treatment might thus protect against the development of this tumour.
Material and Methods: The nationwide Swedish Cancer Register was used to identify a cohort of men diagnosed with a first malignant primary of prostate cancer, an androgen sensitive tumour often receiving hormonal therapy. All participants in the cohort were followed until a diagnosis of a second primary cancer, death, loss to follow-up or end of the study period. Age- and period adjusted standardized incidence ratios (SIR) with 95% confidence interval (CI) were calculated as an estimation of relative risk for a second malignant primary tumour of the esophagus.
Results: Between 1961 and 2008 190,497 patients developed cancer of the prostate as a primary malignant tumour in Sweden. Following exclusion for a follow-up period of less than one year 162,771 patients were eligible for study, contributing 712,496 person-years of follow-up. In total, 60 primary esophageal adenocarcinoma were observed in the cohort as compared to 62.1 expected, resulting in a SIR of 0.97 (95% CI 0.74−1.24).
Conclusions: The risk of developing esophageal adenocarcinoma following a diagnosis and treatment of a primary prostate cancer was no different than expected in the general population. Despite a large cohort our study was hampered by a small number of cases of esophageal adenocarcinoma and of misclassification of anti-androgen exposure diluting