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men (Fig. 1). Clinical malnutrition was defined by a BMI <18.5 kg/m², while visceral obesity as a visceral fat area (VFA) ≥130 cm².

Results: Overall 17(42.5%) patients were sarcopenic. In detail, 14 out of 22 (63.6%) females and only 3 out of 18 (16.6%) males (p=0.04). The majority (65%) had a moderate-severe activity of inflammation based on Harvey Bradshaw index (HBI) ≥8 with a mean of HBI 9.2±1.6. Malnutrition occurred in 41.2% sarcopenic patients with a mean BMI of 16.5±3.75. A significant correlation was observed between BMI and sarcopenia (r=0.4, p=0.001). A total of 25 (62.5%) patients underwent surgery within one year. Among them, 40% patients were sarcopenic, while 60% non sarcopenic (p=0.7). In the total population the mean of VFA was 48.03±58.04 and the ratio between VFA and subcutaneous fat area (SFA) was 0.57±0.5. The correlation between SMI and VFA was significant (r=0.4, p=0.02), while it was not significant with VFA/SFA (p=0.7). For all IBD patients, univariate analysis revealed that female sex (p=0.002) and low BMI (p=0.003) were significantly associated with sarcopenia.

Conclusions: Approximately 42.5% CD patients were sarcopenic. Female sex and low BMI were significantly associated with sarcopenia but this latter did not correlate with the clinical outcome.

OC.07 LOWER GI

OC.07.1
INCREMENTSAL YIELD OF ARTIFICIAL INTELLIGENCE IN FOLLOW-UP SCREENING COLONOSCOPY: AN INTERIM ANALYSIS

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Background and aim: Although colonoscopy is the gold standard for detection of precancerous colonics lesions, they are still missed, and this is directly related with an increased risk of interval colorectal cancer. To improve the diagnostic performances of colonoscopy, novel technologies have been recently developed, such as Artificial Intelligence (AI). The objective of this study was to compare the diagnostic yield obtained by using the GI Genius (Medtronic, Minneapolis, USA) AI software during colonoscopy to that obtained by standard colonoscopy (SC).

Materials and methods: This is a single-center RCT evaluating consecutive patients undergoing follow-up screening colonoscopy at Fondazione Poliambulanza Istituto Ospedaliero, Brescia, Italy. Patients were randomly assigned to SC or AI arm. Subjects with ≤ 1 in any segment according to Boston Bowel Preparation Scale were excluded from analysis. Polyp Detection Rate (PDR), Adenoma Detection Rate (ADR), Serrated Detection Rate (SDR) patients with advanced adenomas (i.e., villous histology, high-grade dysplasia or low-grade dysplasia >1cm) and patients with ≥3 adenomas were compared between the group using χ²-test. P<0.05 were considered statistically significant.

Results: Results are summarized in Figure 1. Out of 708 expected, preliminary data from 392 patients (M:F = 221:171) were collected up to March 3rd 2021. Of those, 192 and 200 were assigned to AI and SC arm, respectively. 19 patients (8 AI and 11 SC) were excluded from final analysis due to inadequate bowel cleansing. Statistically significant improvement was shown in AI group in terms of patients with at least 3 adenomas (26.1% [48/184] vs 16.9% [32/189]; p=0.016), although increases were also shown for ADR (68.5% [126/184] vs 60.3% [114/189]; p=0.05), PDR (82.1% [151/184] vs 75.7% [143/189]; p=0.06), SDR (15.7% [29/184] vs 14.8% [28/189]; p=0.4) and patients with advanced adenomas (10.9% [20/184] vs 8.5% [16/189]; p=0.2).

Conclusions: These preliminary results suggest that AI can be a useful tool during screening colonoscopy, since it significantly improves the number of adenomas detected per patients. In this setting, AI can modify the intervals of endoscopic surveillance.
OC.07.3
RISKS FACTORS IN SYMPTOMATIC UNCOMPLICATED DIVERTICULAR DISEASE
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Background and aim: Clinical scenarios of diverticular disease (DD) are represented by asymptomatic diverticulosis, symptomatic uncomplicated (SUDD) and complicated DD. DD natural history is still not clear and the progression from one stage to another not mandatory. Numerous risk and protective factors for complicated DD have been identified but those regarding SUDD are more scarce. Aim was to search modifiable and not modifiable influencing factors that differ between SUDD and diverticulosis patients.

Materials and methods: Out-patients with SUDD or diverticulosis, referred to Sapienza University Gastroenterology Unit services, were enrolled in this observational study. The influence of modifiable and non-modifiable risk and protective factors were analyzed between the two groups. Continuous variables were expressed as median±standard deviation while categorical variables reported as %. Univariate analysis was performed by Student’s- and Chi square tests respectively. Odd ratio (OR) and 95% confidence interval were obtained by multivariable logistic regression analysis. p values <0.05 were considered statistically significant.

Results: 106 patients (pts) were included (53 with diverticulosis and 53 with SUDD), 64.3% female (85/132), mean age 70.9±11.6 ys, BMI 26.5±4.1 kg/m2. Among not-modifiable risks, female gender was more prevalent in SUDD than in diverticulosis (79.2 vs 52.8%) with a significant association (OR=3.40, 95% CI:1.44–8.01; p=0.0043). Concerning age, pts below mean age of 71 were more prevalent in SUDD than in diverticulosis (42 vs 25%). As regards to modifiable risk factors, BMI was significantly higher (27.2±4.0) in SUDD than in diverticulosis (25.6±3.5) as well as smoking and NSAIDs intake. Smoking was significantly associated with SUDD with an OR of 3.37 (95% CI: 0.11–7.8) while NSAIDs with OR of 5.29 (95% CI=1.63-17.16; p=0.0030). As regards to modifiable protective factors, fiber intake >20 g/day and use of statins were statistically lower in SUDD with respective OR of 2.76 (95% CI: 1.25–6.10; p=0.0114) and of 3.83 (95% CI=1.60-9.20; p=0.002). No differences were found in terms of comorbidities, use of other drugs and physical activity between the two groups.

Conclusions: The association of risk and protective factors known to be related to complicated DD results to influence also the SUDD clinical scenario. An increased awareness of these risks is warranted to prevent the occurrence of symptomatic DD.

OC.07.4
RAFOXANIDE INDUCES IMMUNOGENIC DEATH OF COLORECTAL CANCER CELLS
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Background and aim: Colorectal cancer (CRC) is a major cause of cancer-related death in the world, mainly due to the lack of effective treatment of advanced disease. The ability of cytotoxic therapies to promote tumor-reactive adaptive immune responses has recently emerged as a critical requirement underlying their clinical effectiveness. Antitumor immune response can be primed by immunogenic cell death (ICD), a form of apoptosis associated with endoplasmic reticulum stress (ERS) induction. Unfortunately, the limited number of ICD inducers have been identified so far. The anthelmintic drug rafoxanide has recently shown anti-neoplastic actions in different cancer types, including CRC. As some of these effects relied on pathways activated during ICD (i.e., ERS-mediated eIF2α phosphorylation and autophagy induction), we investigated whether rafoxanide could promote ICD of CRC cells.

Materials and methods: The expression of p-eIF2α and LC3-II markers of ERS and autophagy respectively was assessed by Western blotting in human CRC cells (i.e., HCT-116 and DLD1) treated or not with increasing doses of rafoxanide. The potential of rafoxanide to induce ICD-related DAMPs (i.e., ecto-calreticulin exposure, adenosine triphosphate (ATP)/high mobility group box 1 (HMGB1) release) was assessed by flow-cytometry, chemiluminescent assay and ELISA in HCT-116 and DLD1 cells as well as in the murine adenocarcinoma cell line CT26. Cell proliferation and death was assessed in rafoxanide-treated CT26 cells by BrdU assay and flow-cytometry respectively. To study the immunogenic potential of rafoxanide in a vaccination setting CT26 cells were treated with rafoxanide in vitro and then injected into the left flank of immunocompetent BALB/c mice. The mice were then rechallenged with live tumor cells injected into the right flank and monitored for tumor formation.

Results: Rafoxanide induced p-eIF2α and LC3-II expression in both HCT-116 and DLD1 cells. Such effects were associated with pre-mortem calreticulin exposition on the cell surface, ATP secretion and HMGB1 release. Rafoxanide also induced apoptosis and ICD-related DAMPs in CT26 cells. Finally, we observed a marked increase of tumor-free survival among immunocompetent mice immunized with rafoxanide-treated dying tumor cells as compared with sham.

Conclusions: Our data provide the first evidence that rafoxanide induces bona fide ICD, thus adding a new potential tool in the armamentarium of anti-cancer immunotherapy.

OC.07.5
NON-CURATIVE ENDOSCOPIC SUBMUCOSAL DISSECTION (ESD) FOR COLORECTAL CANCER: CLINICAL OUTCOMES AND PREDICTORS OF RECURRENCE
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