Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Results: Fever, dyspnea, loss of smell/taste/weight, diarrhea, myalgia, arthralgia and asthenia were reported by more than 40% of patients during the acute phase. Abdominal pain/discomfort, diarrhea/incontinence and gastroesophageal reflux disease/regurgitation symptoms persisted after SARS-CoV-2 infection, but with very low severity; the relative increase on the mean score of each domain was minimal (score difference up to +0.16). Compared with controls, adjusted RRs for loose stools, chronic fatigue and somatization were increased after infection: 1.88 (95% CI 0.99–3.54), 2.24 (95% CI 1.48–3.37), 3.62 (95% CI 1.01–6.23) respectively. The prevalence of IBS and HADS scores tended to be greater in patients than in controls. Gastrointestinal sequelae were greater in patients with diarrhea during the acute phase.

Conclusions: Mild gastroenterological symptoms persist five months after SARS-CoV-2 infection, in particular in patients reporting diarrhea in the acute phase. Infected patients are at increased risk of chronic fatigue and somatoform disorders, thus supporting the hypothesis that both functional gastrointestinal and somatoform disorders may have a common biological origin.

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: 0.11–0.78; p=0.0123) 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 and the expression/release of specific damage-associated molecular patterns (DAMPs). Unfortunately, a 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 eIF2 α phosphorylation and autophagy induction), we investigated whether rafoxanide could promote ICD of CRC cells.

Materials and methods: The expression of p-eIF2 α 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-eIF2 α 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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