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<th>Surveillance colonoscopy in patients with serrated lesions at baseline</th>
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expression of TGF-β1/R in HCT116 cells. Addition of SB431542, a selective inhibitor of TGF-βRI, suppressed p38 elevation despite of TGF-β1 suppression. Consistently, suppression of TGF-β1/Ri inhibited p53 elevation and cell apoptosis when expression of TGF-β1 was suppressed in HCT116. Conclusion: The results indicate that in TGF-βRI-disfunctional colorectal cancer cells, endogenous TGF-β1/R has the potential to suppress p53 expression via reduced TGF-β1/R expression, leading to resistance to apoptosis.

Disclosure of Interest: None declared

P1655 SURVEILLANCE COLONOSCOPY IN PATIENTS WITH SERRATED LESIONS AT BASELINE
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Introduction: Serrated lesions of the colon comprise a group of heterogeneous lesions with distinct histological features. The large serrated polyps, in particular, are associated with advanced colorectal neoplasia and possibly higher risk of colorectal cancer.1 None declared However, there is a paucity of data on the optimal surveillance interval for patients with different lesions of the colon.

Aims & Methods: We aim to determine the polyp and adenoma detection rate on surveillance colonoscopy in patients with different serrated lesions.

We identified patients who had undergone surveillance colonoscopy in our hospital between January 2008 and June 2011. Patients with concurrent or past history of colorectal cancer were excluded. Patients were stratified according to their baseline lesions: serrated adenoma (SA, large (>10mm) serrated polyps (LSP) and medium-sized (5-9mm) hyperplastic polyps (MHP), and small number of patients in these groups. The proportion of patients and the time to recurrent colonic polyp/adenoma on surveillance colonoscopy between the two groups (SA + LSP vs MHP) were compared.

Results: A total of 98 patients (24 SA, 9 LSP and 65 MHP) were included for analysis. Surveillance colonoscopy was completed in 65 (66.4%) patients (20 SA, 7 LSP and 38 MHP) with a total of 75 colonoscopy performed. The median age of the patient in the SA + LSP group was significantly older than the MHP group (64.3 years, range 34-86 vs 55.8 years, range: 26-89; p = 0.023). The median time of surveillance colonoscopy was 40.7 and 44.3 months in SA + LSP group and MHP group respectively (p = 0.332). The proportions of patients with recurrent colorectal polyps (including serrated lesions and adenoma) on surveillance colonoscopy were 63.0% and 38.6%, respectively (SA + LSP group vs MHP group) (p = 0.038). There was also a significant difference on the time to polyps recurrence between the two groups, with a lower rate in the MHP group (p = 0.006). The adenoma detection rate on surveillance colonoscopy for SA + LSP group and MHP group were however comparable (29.6% and 28.9%, p = 0.952). Amongst those who have recurrent polyps, 41.2% and 35.7% of patients developed serrated lesions in SA + LSP group and MHP group respectively.

Conclusion: Patients with baseline SA and LSP have a significantly higher polyp detection rate on surveillance colonoscopy compared to those with baseline MHP. When deciding on the optimal screening interval for patients with different baseline serrated lesions, the adenoma detection rate on surveillance colonoscopy was similar. These findings provide new data on the relation between proximal colonic serrated lesions with distinct histological features. The large serrated polyps, in particular, are associated with advanced colorectal neoplasia and possibly higher risk of colorectal cancer. However, there is a paucity of data on the optimal surveillance interval for patients with different lesions of the colon.

References:

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P1656 MECHANISM OF PROKINETIN RECEPTOR 2 IN COLON CANCER
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Introduction: Prokinetin-1 (PROK1) is thought to be involved with cell invasion through interaction of prokinetin receptor 2 (PK-R2), that is one of the Prokinetin-receptor-1 receptors. We report a function via PROK1-PK-R2 signaling in vitro.

Aims & Methods: 1: Four colon cancer cell lines (DLD1, HCT116, SW620 and HT29) were analyzed for PROK1 and PK-R2 protein expressions. 2: Two colon cancer cell lines (DLD1 and HT116) stimulated with PROK1 protein, were observed for 24 hours using “Olympus Fluoview 10w”, for the presence of changes in cell movement. Furthermore, we measured the moving distance of five cells in non-stimulated group and PROK1 stimulated group, compared using the U test of Mann-Whitney. 3: The expression of PROK1 and PK-R2 protein expressions in 253 colorectal cancer tissues by immunohistochemical staining using anti-PROK1 antibody and anti-PK-R2 antibody. We investigated the relation between PROK1 and PK-R2 expression and clinicopathological factors of colorectal cancer patients. 4: Expression with several immunohistochemistry analyses of lymphatic invasion, venous invasion, lymph node metastasis, peritoneal metastasis, and hematogenous metastasis was assessed by cross-tabulation, and statistical significance was determined by the χ2 test. Life-table analysis and Kaplan-Meier technique and outcomes from different groups of patients were compared by the log rank test.

Results: 1: The expression of PROK1 and PK-R2 protein was detected in four colon cancer cell lines. 2: The PROK1 stimulated group, compared to non-stimulated group, showed a significant increase in the expression of PROK1. The average of the moving distance of the cells of DLD1 was 380.96 pixel in non-stimulated group, in PROK1 stimulated group was 581.50 pixel. The average of the moving distance of the cells of HCT116 was 266.29 pixel in non-stimulated group, in PROK1 stimulated group was 400.90 pixel. Both cell lines showed a significant extension of the moving distance of the cells in PROK1 stimulated group. 3: Group1, positive both PROK1 or PK-R2, was 117 cases. Group2, positive either PROK1 or PK-R2, but not both, was 109 cases. Group3, negative both PROK1 and PK-R2, was 99 cases. According to the clinicopathological examinations, the frequency of Group1 was significantly higher in cases with lymph node invasion, venous invasion, lymph node metastasis and hematogenous metastasis. The prognosis for patients with Group1 were significantly worse than the other groups.

Conclusion: Colon cancer cell lines stimulated with PROK1 protein, showed enhancement of moving distance and deformation and motility of cells. Signaling cascade thought PROK1/PK-R2 was suggested likely to be involved in metastasis, especially cell invasion.

Disclosure of Interest: None declared