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OP001 DIAGNOSTIC YIELD OF "ARTIFICIAL INTELLIGENCE"-ASSISTED ENDOCYTOSCOPY FOR COLORECTAL POLYPS: A PROSPECTIVE STUDY

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Introduction: Computer-aided diagnosis (CAD) powered by artificial intelligence is attracting increased attention as an option to improve the performance of optical biopsy for evaluating colorectal polyps [1]. Although positive preliminary data have been shown for applying CAD to endocytoscopy (EC) (500-fold ultra-magnifying endoscopy; Olympus Corp., Tokyo, Japan) [2, 3], no prospective studies have been reported.
Aims & Methods: The present study is an initial prospective trial to validate the feasibility of applying CAD to endocytoscopy in a routine colonoscopy practice. A total of 88 patients (38 women, 50 men; mean age 64 years) in whom colorectal polyps had been detected using EC for colonoscopy were prospectively enrolled in the study between January and March 2017. When a polyp was detected, an on-site endoscopist predicted the polyp pathology using the CAD system [2], which was designed to output the predicted pathology of the target lesion—whether neoplastic or non-neoplastic—together with the probability of the diagnosis (0–100%) immediately after obtaining a methylene blue-stained EC image. The endoscopists obtained as many images as they thought were needed, each of which was evaluated using image-based analysis. The diagnostic ability of the CAD for each image was assessed with reference to the final pathology of the resected specimen. The main outcome measures were diagnostic sensitivity specificity, accuracy, positive predictive value, and negative predictive value of the CAD system for identifying neoplastic change with high confidence (probability ≥90%). Prior to initiating the trial, 13,861 EC images were used for machine-learning the CAD model.
Results: Overall, 126 lesions (62 neoplastic lesions, 64 non-neoplastic lesions; mean size 6 mm) were detected, all of which were successfully analyzed using the CAD system. A total of 1014 EC images of neoplastic lesions and 1480 EC images of non-neoplastic lesions were obtained during the colonoscopies of these patients. Among them, 55% (1378/2494) were diagnosed with high confidence (CAD probability was ≥90%). The sensitivity, specificity, accuracy, positive predictive value, and negative predictive value of the CAD system in identifying neoplastic change with high confidence were 97%, 67%, 83%, 78%, and 95%, respectively (Table). No complications occurred during the study.

		pathology neoplastic	non-neoplastic
Diagnosis of CAD with high confidence	neoplastic	733	208
	non-neoplastic	20	417

Conclusion: This prospective trial revealed that applying CAD to EC was feasible, with a negative predictive value of >90%, which is likely to meet the threshold required for optical biopsy of colorectal polyps. Our next goal is to increase the proportion of high confidence diagnoses, which is currently limited to 55%. (This study is registered as UMIN Clinical Trial Registry No. 000013917 and supported by Grants-in-Aid for Scientific Research No. 15K19351 from the Japan Society for the Promotion of Science.)
Disclosure of Interest: All authors have declared no conflicts of interest.

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OP002 GENOME-WIDE ASSOCIATION STUDY IDENTIFIES INVERSION IN THE CTRB1-CTRB2 LOCUS TO MODIFY RISK FOR ALCOHOLIC AND NON-ALCOHOLIC CHRONIC PANCREATITIS

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Introduction: Alcohol-related pancreatitis is associated with a disproportionately large number of hospitalizations among gastrointestinal disorders. Despite its clinical importance, genetic susceptibility to alcoholic pancreatitis is poorly characterized. Our aim was to identify risk genes for alcoholic chronic pancreatitis (CP) and to evaluate their relevance in non-alcoholic CP. Therefore, we performed a genome-wide association study and functional characterization of a new pancreatitis locus.
Aims & Methods: Patients with CP were collected according to a standardized protocol throughout Europe. DNA extracted from peripheral blood samples was used for genetic investigations with Illumina technology, PCR, and melting curve assays. In total, 1959 European alcoholic CP patients and population-based controls from the KORA, LIFE and INCIPE study (n = 4708) as well as chronic alcoholics from the GESGA consortium (n = 1332) were investigated. Replication was performed in three European cohorts comprising 1650 patients with non-alcoholic CP and 6695 controls originating from the same countries.
Results: We replicated previously reported risk loci PRSS1-PRSS2, CLDN2-MORC4, SPINK1 and CTRC in alcoholic CP patients. We identified CTRB1-CTRB2 (chymotrypsin B1 and B2) as a new risk locus with lead SNP rs8055167 (odds ratio, 1.35 [95% CI 1.23 to 1.6]; $P = 4.2 \times 10^{-9}$). We found that a 16.6 kb inversion in the CTRB1-CTRB2 locus was in linkage disequilibrium with the CP-associated SNPs and best tagged by rs8048956. The association was also successfully replicated in three independent European non-alcoholic CP cohorts of 1, 650 patients and 6695 controls (odds ratio, 1.62 [95% CI 1.42 to 1.86]; $P = 1.64 \times 10^{-12}$). The inversion changes the expression ratio of CTRB1 and CTRB2 isoforms and thereby affects protective trypsinogen degradation and ultimately pancreatitis risk.
Conclusion: Our GWAS identified *CTRB1-CTRB2* as a new risk locus for ACP and NACP. The association within the *CTRB1-CTRB2* locus was linked to a 16.6 kb inversion that altered CTRB1/CTRB2 expression thereby affecting protective trypsinogen degradation. Furthermore, we confirmed association of ACP with the *PRSS1-PRSS2*, *CLDN2-MORC4*, *CTRC* and *SPINK1* loci. Taken together, the identified risk variants explained about 18% of the variance in ACP. Our discovery provides strong evidence for common pathogenic mechanisms underlying the complex etiology of ACP and NACP.
Disclosure of Interest: All authors have declared no conflicts of interest.