

Gastroduodenoscopic findings and *Helicobacter pylori* in patients with rheumatoid arthritis.

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Objective: Patients with rheumatoid arthritis(RA) have an increased risk of peptic ulcer disease(PUD). The aim of this study is to investigate gastroduodenoscopic findings, presence of *Helicobacter pylori*(H. pylori) and peptic ulcer in patients with RA compared with patients with osteoarthritis(OA).

Methods: Gastroduodenal endoscopy was performed, regardless of gastrointestinal symptoms, on 38 RA patients and 24 OA patients as controls. Gastroduodenal injury was graded on a 0-3 scale according to the modified Lanza score and H. pylori infection was investigated by using histologic examination or CLO test.

Results: The incidences of gastroduodenal lesions were similar in both diseases, being 20(52.7%) of RA patients compared with 12(50%) of OA patients. However there were differences in type of gastroduodenoscopic lesions between the groups; Peptic ulcer was found only on 5(13.1%) of RA patients(4 gastric ulcer, 1 duodenal ulcer). In RA patients 14(36.8%) had gastric erosions and 1(2.6%) had chronic gastric atrophy and in OA patients, 6(25%) and 6(25%) respectively($p=0.04$). The mean scores of the modified Lanza mucosa injury system were 1.16 ± 0.19 in RA patients and 0.83 ± 0.19 in OA patients($p = 0.08$). Nonsteroidal antiinflammatory drugs(NSAID) and prednisolone were being taken more frequently in RA patients than in OA patients(97.4% and 97.4% vs 75% and 4.2%, respectively)($p < 0.001$), but H. pylori was present more frequently in OA patients than in RA patients(83.3% vs 47.4%, $p = 0.01$).

Conclusion: RA patients had more PUD compared with OA patients. This may be due to increased use of NSAID and steroid, but not H. pylori infection.

ENHANCED T CELL PROLIFERATIVE RESPONSE TO NATIVE BOVINE TYPE II COLLAGEN AND ITS IMMUNODOMINANT EPITOPE (CII 255-274) IN PATIENTS WITH RHEUMATOID ARTHRITIS.

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To define the specific immune recognition to native type II collagen (nCII) in patients with rheumatoid arthritis (RA), T cell proliferative responses to nCII and its immunodominant synthetic peptide ²⁵⁵TGEBGIAGFKGEQGPKEG²⁷⁴(CII 255-274) were performed in peripheral blood mononuclear cells (PBMC) ($n=57$), synovial fluid mononuclear cell (SFMC) ($n=20$) from patients with RA and PBMC from healthy control ($n=20$). Irradiated non-T cells separated immunomagnetically, were used as antigen presenting cells for T cell proliferation. Culture supernatants were collected to measure Th1 and Th2 cytokines (IL-2, IL-4, IL-10 and INF- γ) by sandwich ELISA. Positive T cell responses in PBMC (defined as stimulation index (SI) >2 and $\Delta\text{cpm}>1000$) to CII or CII 255-274 were found in 57.8% of RA and only 14.3% of normal controls ($p<0.001$), and mean SI (background cpm = 14033 ± 4044) was also higher in RA than normal (CII: 1.90 ± 0.56 vs. 1.46 ± 0.29 , CII 255-274: 1.86 ± 0.55 vs. 1.46 ± 0.37 , $p<0.001$). Proliferative response to CII was closely correlated with that to CII 255-274 ($r=0.533$). There were no differences in T cell proliferative response to CII or CII 255-274 between patients with antibody to CII ($n=32$) and without ($n=25$). Particularly, proliferative responses to CII 255-274 were more frequent and higher in SFMC ($n=20$) than in PBMC ($n=57$) (75.0% vs. 45.6%, $p<0.05$; SI= 2.22 ± 0.66 vs. 1.86 ± 0.54 , $p<0.01$). In culture supernatants of PBMC, the levels of IL-2, INF- γ and ratio of INF- γ /IL-4 were higher in RA ($p<0.001$), while the levels of IL-10 were lower in RA than normal. The level of IL-2 and INF- γ were positively correlated with SI ($r=0.387$, $r=0.329$). Taken together, CII 255-274 as well as CII could be recognized as immunogenic antigens by T cells, particularly recruited in synovial cavity in RA. These results are consistent with the possibility that CII may be involved in the pathogenesis of RA as an autoantigen.