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Gastroduodenoscopic findings and Helicobactor 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 Helicobactor 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 Lansa 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.

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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 ^{255}TGEBGIAGFKGEQGPKGEBG^{274}(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 seperated immunomagnetically, were used as antigen presenting cells for T cell proliferation. Culture supernants were collected to measure Th1 and Th2 cytokines (IL-2, IL-4, IL-10 and INF-γ) by sandwitch ELISA. Positive T cell responses in PBMC (defined as stimulation index (SI) >2 and $\triangle$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$\pm$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 supernants of PBMC, the levels of IL-2, INF-γ and ratio of IFN-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.