Anti-tumor necrosis factor-? therapy augments dipeptidyl peptidase IV activity and decreases autoantibodies to GRP78/BIP and phosphoglucose isomerase in patients with rheumatoid arthritis

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Objective. To assess the enzymatic activity and biochemical status of dipeptidyl peptidase IV (DPP IV), an enzyme that participates in the degradation of proinflammatory molecules, in sera from a group of patients with rheumatoid arthritis (RA; n = 15) treated with a human anti-tumor necrosis factor-? (anti-TNF-?) antibody (adalimumab) for 32 weeks. IgG antibody titers against chaperone Bip (GRP78), phosphoglucose isomerase (PGI), lactate dehydrogenase (LDH), fibronectin (FN), and actin were also studied. Methods. DPP IV activity was measured in sera using Gly-Pro-p-nitroanilide as substrate. The biochemical profile of circulating DPP IV glycoforms was assessed by isoelectric focusing gel electrophoresis. All IgG autoantibody titers and their sialylation levels were determined by ELISA. Results. Patients showed significant increases in serum DPP IV enzymatic activity from basal values (3.554 ± 1.096) with respect to those obtained at 32 weeks (4.787 ± 0.953; $p < 0.05$). Changes in the b