High prevalence of small intestinal bacterial overgrowth (SIBO) in spondyloarthropathy

Sirs,

The concept of spondyloarthropathy (SpA) gathers together a group of chronic diseases with common clinical, biological, genetic and therapeutic characteristics: ankylosing spondylitis, reactive arthritis, psoriatic arthritis and arthritis in patients with inflammatory bowel disease. Spondyloarthropathies and Crohn's disease are multifactorial diseases with genetic and environmental backgrounds; a key role of intestinal microbiota is suspected in both diseases (1-3, 8). Rather than one pathogenic bacteria, it could result from an imbalance in the intestinal microbiota as observed in small intestinal bacterial overgrowth (SIBO) (5).

The aim of this study was to investigate the prevalence of small intestinal bacterial overgrowth using lactulose breath hydrogen test in adult patients with spondyloarthropathy, and the impact of long-term treatment on SIBO development.

One hundred and thirty-four rheumatologic patients were evaluated retrospectively. Among them, 73 were diagnosed SpA according to the AMOR, ESSG and ASAS criteria, and 61 with other rheumatologic pathologies were considered as controls. Long-term treatment at the time of the study by salazopyrin, non-steroidal anti-inflammatory drugs (NSAIDs) and/or proton-pump inhibitors (PPI) was described.

SIBO was diagnosed using hydrogen breath test as previously described, with kinetic measurement of hydrogen concentration in expired alveolar air sample using a gas chromatograph (Model DP, Quintron Instrument Co) (4, 6). The hydrogen produced after lactulose ingestion derives entirely from intestinal bacterial fermentation.

Table 1 describes the clinical characteristics (diagnosis, digestive symptoms and long-term treatment) of the SpA (n=73) and control (n=61) patients, and SIBO results.

The prevalence of SIBO was significantly higher in SpA patients (63% - 46/73) than in control group (5% - 3/61) ($p<0.0001$, χ^2, Table I). SIBO prevalence did not significantly differ in HLA-B27+ positive patients (25/38) as compared to HLA-B27 negative patients (21/35) ($p=0.6087$, χ^2).

SIBO prevalence was not significantly different in patients with or without salazopyrin ($p=1.0$, Fisher test), IPP or NSAIDS therapies ($p=0.3925$ and $p=0.1895$, respectively, χ^2 test). More than half of the SpA patients with PPI therapy (59%) and NSAID therapy (61%) had SIBO whereas only 6% of patients with PPI and/or NSAID therapy had SIBO in control group.

In this study, we show a high prevalence of SIBO in patients with SpA significantly higher than that of a control population of patients with other rheumatologic diseases. SIBO in SpA patients does not seem to be related to the long-term treatment with salazopyrin, NSAID or PPI.

SIBO development could be the result of alterations of the external environmental milieu (variation in the intestinal microbiota), of a genetic factor through innate immunity (role of Toll-like receptors) or of certain dietary intolerance (as in coeliac disease).

In reactive arthritis, a direct link has been observed between bacterial intestinal invasion and subsequent clinical manifestations. However, increase prevalence of SIBO rather suggests a key role for an imbalance of the intestinal bacterial flora which may disturb the biotope/intestinal interface (10). Although this study did not evaluate the efficacy of SIBO treatment upon symptomatology, it is interesting to note that 77% (33/43) of patients reported a quite satisfactory amelioration of their symptoms (fatigue, pain) after dual treatment with amoxicillin-clavulanic acid and metronidazole, alone or in combination.

In conclusion, this work illustrates the high prevalence of SIBO in spondyloarthropathies. The dysbiosis and the chronic bacterial colonisation of the small intestine would have a role in the physiopathology of SpA. It would be useful to evaluate the impact of healing (restoring intestinal bacterial flora balance – wide spectrum or local action antibiotics therapies such as amoxicillin-clavulanic acid association, ciprofloxacin, metronidazole or rifaximin) (7) and preventive (preserving it, pre- or probiotics, nutritional modulation) (9) therapies on the clinical evolution of spondyloarthropathy patients.

Table I. Control and SpA groups characteristics and SIBO results.

	Control group	SpA group
number of patients	61	73
median age	56 (18-79)	51 (17-84)
sex ratio (M/F)	0.09 (5/56)	0.52 (25/48)
"principal" pathologies	osteoarthritis (n=20), rachialgia (n=11), polyalgic syndrome (n=8), polyarthralgia (n=6)	spondyloarthritis (n=36), oligoarthritis (n=9), psoriatic rheumatism (n=16), spondyloarthropathy (n=12)
HLA B27+	14 (23%)	38 (52%)
Digestive symptoms	45 (74%)	42 (58%)
Salazopyrine treatment	0	12 (16%)
IPP treatment	19 (31%)	44 (60%)
NSAI treatment	25 (41%)	56 (77%)
SIBO	5% (3/61)	63 (46/73)

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