Small intestinal bacterial overgrowth in patients with rheumatoid arthritis

I read with interest the paper by Henriksson et al., in which the authors demonstrated an altered character in the small bowel’s bacterial flora in patients with rheumatoid arthritis. However, the authors did not demonstrate the presence of bacterial overgrowth. The syndrome of bacterial overgrowth is characterised by diarrhoea (a luminal flora) and not by diarrhea with malabsorption. The mechanism by which the bacterial overgrowth causes symptoms is not known. These symptoms are related to an increase in the luminal bacterial flora and the presence of obligate anaerobes (colonic luminal flora) as judged by jejunal fluid culture or breath tests. The patients in the study did not have any evidence of malabsorption or diarrhoea.

The investigations used, apart from the bile acid deconjugation test, had not been previously validated in patients with bacterial overgrowth. Quantitative culture was not done; the bacterial flora was of oral type, not colonic. The presence of enterobacteriaceae (a facultative anaerobe), tryptic activity, gas production (both not specific to obligate anaerobes) and the use of jejunal biopsy culture (biopsies culture produces different flora than fluid culture) are not proven features of bacterial overgrowth of the small bowel. The controls used did not have the syndrome of bacterial overgrowth as they were clearly stated.

We feel that the two concepts bacterial overgrowth and bacterial overgrowth syndrome are sometimes confused. We used the concept bacterial overgrowth to designate an altered microflora in the upper small intestine in patients with RA, and the criteria were clearly stated in the article. The bacterial overgrowth syndrome, also known as blind loop or stagnant loop syndrome is characterised by steatorrhea and other signs of malabsorption. We agree that our patients with RA did not have the bacterial overgrowth syndrome.

Dr Lewis, however, seems to presume that only an altered small intestinal microflora complicated by the bacterial overgrowth syndrome is of interest in patients with RA. This view has to be questioned as we still do not know what types or quantities of microbes demonstrated in the upper small intestine are of interest in these patients.

The aim of our study was not to describe a bacterial overgrowth syndrome in patients with RA, but rather to denote an altered small intestinal microflora. A combination of techniques was preferred to get a comprehensive picture of small intestinal microflora, as it has been shown that different tests may be necessary to detect bacterial overgrowth in the upper small intestine. It has been suggested that a qualitative change may be more important than a quantitative change. We used the growth of Enterobacteriaceae (Escherichia coli or Klebsiella) without counting the microorganisms as one of the criteria of bacterial overgrowth. These species originate from the colonic and not from the oral microflora.

The clinical significance of our findings is still uncertain. The RA patients with signs of bacterial overgrowth had significantly higher rheumatic factor titres as well as rheumatoid factor titre. Dr Lewis’s suggestion that the changes in the small bowel bacterial flora may be secondary to multi-system involvement of RA certainly cannot be excluded. As for his contentions that treatment with proton pump inhibitors or antibiotics would confirm our hypothesis, it should be noted that the small intestinal bacterial overgrowth in these patients was not only related to lack of gastric acid and that we know very little about the types of microorganisms involved; severe flare ups after treatment with antibiotics have been observed in patients with RA, possibly by changing the intestinal microflora in the ‘wrong’ direction.

Relapsing polychondritis as a secondary phenomenon of primary systemic vasculitis

Papo et al recently reported that in 33 patients with relapsing polychondritis (RP) (three women, four men) the diagnosis of RP was either made historically or according to the diagnostic criteria established by McAdam et al. In six patients, RP presented as a secondary phenomenon during an active phase of a primary vasculitic entity. Autoantibody screening revealed cANCA (PR3 ANCA +) in two patients, pANCA in one patient and autoantibodies to native collagen type II in four patients. According to the ACR Classification-Criteria for PSV 1990, the underlying PSV in these six patients was historically proven (PR3 ANCA +) in two cases, microscopic polyarthritis in three cases (one pANCA +), classic polyarthritis nodosa in one case (table).

We think that polychondritis can be diagnosed more often as a secondary phenomenon in PSV than has previously been recognised. The diagnosis of PSV has been considerably improved by the detection of ANCA and the establishment of ACR Classification-Criteria for Primary Systemic Vasculitis, Definition of PSV. Subsequently, PSV with secondary polychondritis seems to be recognised more often. Although there are only very few case reports on the association of RP with recognised, classified PSV, the coincidence of RP with unclassified vasculitic symptoms is said to be about 10%. The occurrence of glomerulonephritis in RP seems to be even more frequent (29 of 112 patients). These vascular symptoms may be attributed to an underlying PSV, that has not been previously recognised as such. We conclude that the occurrence of ANCA in RP should encourage thorough investigation for the presence of PSV, in which the polychondritis may be a secondary phenomenon. This is important, as the vasculitis determines the therapy and prognosis of the disease.
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