MATTERS ARISING

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 characterized 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 diarrhea.

The investigations used, apart from the bile acid deconjugation test, had not been previously validated in patients with bacterial overgrowth. Quantitative bacterial 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 (biopsy 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 elderly people, with dysmotility, which is associated with an altered and increased bacterial flora, does not necessarily cause symptomatic bacterial overgrowth. Thus only the three patients with positive bile acid deconjugation test could be considered to have investigational evidence of bacterial overgrowth. In view of previously reported changes seen in the small bowel’s bacterial flora of elderly and infirm people, perhaps the findings were not surprising considering the multi-system involvement of rheumatoid arthritis. To confirm their hypothesis that disease activity is due to and not causing the bacterial flora changes, the authors could have studied the influence of either proton pump inhibitors or antibiotics on disease activity.

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Relapsing polychondritis as a secondary phenomenon of primary systemic vasculitis

Papo et al recently reported that in 33 patients with relapsing polychondritis (RP) low titre cANCA were present in 24% of the cases. They concluded that low titres of cANCA therefore are not specific for Wegener’s granulomatosis (WG). We do not think that this line of reasoning is correct. We and others have earlier reported that cANCA is a highly specific and sensitive marker for WG. Thus we conclude that the presence of ANCA in RP, may instead indicate, that polychondritis occurs in the course of a primary systemic vasculitis (PSV), for example, WG.

We have seen such an association of PSV with polyarthritis in six of seven patients with 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 acute phase of a primary vasculitis entity. Autoantbody screening revealed cANCA (PR3 ANCA +) in two patients, pANCA in one patient and autoantibodies to native collagen type II in four patients.

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 PSV 1990, the underlying PSV in these six patients was: histologically proven WG (PR3 ANCA +) in two cases, microscopic polyangiitis in three cases (one pANCA +), classic polyarteritis nodosa in one case (table).

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