Letters to the Editor

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Familial adenomatous polyposis coli associated arthritis and vasculitis

Sirs.

The association between arthritis, mainly peripheral (20%) but also axial (10%), vasculitis and inflammatory bowel diseases (IBD) is well known and frequently reported in the literature (1, 2). To our knowledge, an association between arthritis, vasculitis and FAP has never been recognized. Here we illustrate a case in which these features were all present, as well as a precise coincidence of timing between polyposis relapses after endoscopic removal and arthritis outbreaks.

A 56-year-old woman came to our attention because of fever, arthritis of the wrists and hands, and vesiculopapular eruptions on the back and limbs. There was a family history of colorectal carcinoma and adenomatous polyposis, with a sister also affected by polyps in the residual rectum, laparoscopic and endoscopically excluded. A colonoscopy was performed and revealed widespread colic polyposis. The patient was treated with 6-methylprednisolone therapy and pouchitis after proctocolectomy for inflammatory diseases of the bowel and for FAPs described; the suggested mechanism is the persistence of abnormally permeable intestinal membrane in the site because of phlogosis. The described clinical manifestations are confined to arthralgias, not arthritis (7, 8).

In this case, cutaneous biopsy showed a perivascular infiltrate mainly composed of neutrophils (and their fragments) and lymphocytes, and fibrinoid degeneration of the wall of some vessels, leading to a diagnosis of necrotizing venulitis. A paraneoplastic expression of collagen colon carcinoma, which is rarely manifested in an in situ colon carcinoma, was considered, but the second and the third outbreaks of cutaneous lesions occurred without evidence of neoplastic degeneration. Erythema elevatum diutinum and Sweet’s syndrome were clinically and histologically excluded. Moreover, the precise coincidence of timing between the polyposis relapses and arthritis/vasculitis outbreaks was very striking; this could reasonably suggest a correlation between altered intestinal permeability and the development of phlogosis, perhaps mediated by inflammation triggers. An additional interesting reflection could be made regarding the clinical case of the patient’s sister, who was in turn affected by FAP and developed arthritis at the same time and with the same characteristics. This finding could suggest a common pathological and genetic mechanism.

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References


Fig. 1. X-ray showing non-erosive arthritis of the wrist and hands.