BD have also been described in the same patients (6). Whether there is an association between these two diseases or the occurrence is due to the high prevalence of these 2 diseases in the same area is not clear. Recently, Toutou et al. (7) screened MEFV mutations in a panel of BD patients and found that the M94V, V726A, and E148Q mutations tended to be more frequent in definite BD (2.6%, 2.6% and 5.2% respectively) than in controls (0%, 0% and 2.2% respectively), and suggested that they may act as additional susceptibility factor in BD. Schwartz et al. (6) showed that the prevalence of BD was higher in FMF than in populations rich in BD (e.g., 16 per 4000 in FMF compared with 1 per 104 in Japan).

In our case, it may be difficult to make a diagnosis of PAN, as perirenal and retroperitoneal haematoma secondary to renal arterial aneurysm has been reported in two patients with BD to date (8, 9). Therefore, angiographic findings and retroperitoneal haematoma may be due to BD in this case. However, thrombophlebitis and venous thrombosis are often seen at or around the same time as arterial aneurysms in patients with BD (10, 11), and since our patient neither had any history of these nor was observed to have such manifestations during the follow-up period, we can conclude that the angiographic findings and retroperitoneal haematoma may also be due to PAN. Our case met the ACR criteria (12) and the criteria suggested by us (13) for the classification of PAN. Furthermore, the prevalence of PAN in FMF is 1% (3), and perirenal haematoma is seen in almost half the patients with FMF who develop PAN (14).

The relationship between these diseases requires further investigation. There are some clinical and laboratory similarities between them (6). Additional genetic and/or environmental factors may predispose an FMF patient to a more persistent inflammation in the relevant pathway, which may manifest itself in the form of vasculitis. Further studies will enlighten the association of these diseases and shed light on the pathogenesis of inflammation.

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References

Letters to the Editor

Reporting a desensitization protocol for colchicine treatment

Sirs,
The development of toxicity and hypersensitivity to colchicine therapy is a well-known entity in patients with familial Mediterranean fever (FMF) and Behçet’s disease (BD) (1, 2). On the other hand amyloidosis is a known sequel of untreated patients affecting mainly patients with FMF, and therefore there is a need to treat such patients with colchicine to prevent this complication.

We recently examined a 47-year-old female with FMF in whom the diagnosis of BD manifesting as recurrent oral and vaginal ulcers, pyoderma gangrenosum, erythema nodosum and arthralgias was established 20 years ago. The treatment regimen prescribed for BD as well as FMF included methotrexate, cyclophosphamide and colchicine which led to partial remission of both diseases.

The patient developed an allergic skin reaction to colchicine, proven by an oral challenge test. Despite the allergic reaction that erupted as a maculopapular rash evident on the lower limbs, it was necessary to continue the colchicine therapy because of its prominent role in preventing the development of amyloidosis.

Desensitization was achieved with the oral administration of increasing doses of colchicine. Colchicine 0.5 mg was dissolved in 500 cc of glucose 5%. On the first day of treatment 0.5 cc was administered, and the dose was doubled each day until a dose of 250 cc (0.25 mg colchicine) was reached on the tenth and last day of treatment. No adverse effects were observed during the 10 day desensitization period. At the present time, two months after the desensitization process, the patient is receiving 1 mg colchicine/day, with no evidence of an allergic reaction.

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