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, Touitou *et al.* (7) screened MEFV mutations in a panel of BD patients and found that the M694V, 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 10^4 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

1. SOHAR E, GAFNI J, PRAS M, HELLER H: Familial Mediterranean fever: a survey of 470 cases and review of the literature. *Am J Med* 1967; 43: 227-53.
2. SAATÇI U, BAKKALOGLU A, OZEN S, BESBAS N: Familial Mediterranean Fever and amyloidosis in children. *Acta Paediatrica* 1993; 81: 705-6.
3. OZDOGAN H,ARISOY N, KASAPÇOPUR Ö, *et al.*: Vasculitis in familial Mediterranean fever. *J Rheumatol* 1997; 24: 323-7.
4. LIVNEH A, LANGEVITZ P, ZEMER D, *et al.*: Criteria for the diagnosis of familial Mediterranean fever. *Arthritis Rheum* 1997; 40:1884-90.
5. INTERNATIONAL STUDY GROUP FOR BEHÇET'S DISEASE: Criteria for the diagnosis of Behçet's disease. *Lancet* 1990; 335: 1078-80.
6. SCHWARTZ T, LANGEVITZ P, ZEMER D, GAZIT E, PRAS M, LIVNEH A: Behçet's disease in familial Meditearranean fever:Characterization of the association between the two diseases. *Semin Arthritis Rheum* 2000; 29: 286-95.
7. TOUITOU I, MAGNE X, MOLINARI N, *et al.*: MEFV mutations in Behçet's disease. *Human Mutation* 2000; 16: 271-2.
8. HAN K, SIEGEL R, PANTUCK AJ, GAZI MA, BURNO DK, WEISS RE: Behçet's syndrome with left ventricular aneurysm and ruptured renal artery pseudoaneurysm. *Urology* 1999; 54: 162.
9. SUEYOSHI E, SAKAMOTO I, HAYASHI N, *et al.*: Ruptured renal artery aneurysm due to Behçet's syndrome. *Abdom Imaging* 1996; 21: 166-7.
10. DÜNDARSV, ÜNAL S, SIVRI B, *et al.*: Behçet's disease in Turkish population:analysis of 200 cases. *In:* LEHNER T, BARNES GG (Eds.): *Recent Advances in Behçet's Disease*. London, Royal Society of Medicine Services International Congress and Symposium series No. 103, 1986; 219-21.
11. HAMURYUDAN V, YURDAKUL S, MORAL F, *et al.*: Pulmonary arterial aneurysms in Behçet's syndrome: a report of 24 cases. *Br J Rheumatol* 1994; 33: 48-51.
12. LIGHTFOOT RM, MICHET BA, BLOCH DA, *et al.*: The American College of Rheumatology 1990 criteria for the classification of polyarteritis nodosa. *Arthritis Rheum* 1990; 33: 1088-93.
13. OZEN S, BESBAS N, SAATÇI U, BAKKALOGLU A: Diagnostic criteria for PAN in childhood. *J Pediatr* 1992; 120: 206-9.
14. GLIKSON M, GALUN E, SCHLESINGER M, *et al.*: Polyarteritis nodosa and familial Mediterranean fever: A report of 2 cases and review of the literature. J Rheumatol 1989; 16: 536-39.

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 Behcet'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 ganrenosum, erythema nodosum and arthralgias was established 20 years ago. The treatment regiment 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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References

1. BEN-CHETRIT E, LEVY M: Colchicine: 1998 update. *Semin Arthritis Rheum* 1998; 28: 48-59.
2. MOCHIDA K, TERAMASE H, HAMADA T: Fixed drug eruption due to colchicine. *Der - matology* 1996; 192: 61.