added to his treatment, as with Case 1.

Case 3 (37/M) referred to us for cutaneous vasculitis, confirmed by the skin biopsy. He had a history of recurrent fever, abdominal and chest pain, and erysipelas-like erythema since he was 3. He was diagnosed with FMF at the age of 27 and colchicine was administered at the dosage of 0.5-1 mg/day. Since case 3 had a cutaneous vasculitis, I administered PRD and AZA in the aforementioned dosages in addition to colchicine (2 mg a day), enalapril maleat (10-20 mg/day) immediately after the diagnosis of AA amyloidosis and vasculitis. The dosage of PRD and AZA was adjusted as in cases 1 and 2.

Laboratory findings for the patients obtained during the follow-up period are presented in Table I.

Three cases with NRP secondary to FMF amyloidosis responded markedly to AZA and PRD while still receiving colchicine. It is possible for FMF patients to suffer from non-amyloid renal involvements, such as glomerulonephritis (3), and to respond favourably to immunosuppressants (3). To be frank, I could not exclude the possibility of glomerulonephritis or other glomerulopathies in my cases due to the failure to perform a renal biopsy. It is well documented that FMF patients with glomerulonephritis do suffer from hematuria and leukocyturia (3). However, my cases were determined to have neither of these urinary abnormalities during the follow-up period. Based on this result, I think the chances for the presence of such possibilities, as glomerulonephritis are low with my cases.

Although the first two cases showed a mild stabilization of NRPbefore the start of AZA and PRD, a noticeable improvement was determined after the start of AZAand PRD. This can best be seen in case 3, who showed the earliest noticeable improvement in NRP because of receiving the combination of AZAand PRD and colchicine upon presentation. At this point, it could be argued that addition of the AZA and PRD was made fairly early when taking into account that one year may not be a sufficient period for the effect of colchicine to begin for cases 1 and 2. However, it should be noted that the optimum length of time needed for the effect of the colchicine to begin has not be determined precisely thus far. The natural duration between the emergence of proteinuria and ESRF has been determined to range from 2 to 13 years (4).It is well known that long-term proteinuria due to other diseases might lead to tubulointerstitial nephritis (5). Also, histological evidence of tubulointerstitial injury has been found to be a better predictor of impaired renal function than glomerular injury (5). Moreover, it has been reported that tubulointerstitial injury is also a predictor for the poor outcome of FMF amyloidosis (6). Based on the data above, early interventions for reducing NRP could help renal prognosis in a positive way. The dramatic decrease in the levels of proteinuria following the commencement of the combination of AZA and PRD seems to be suggestive of its workability.

There exist only a few studies into endogenous cortisol levels in FMF patients. Early blunted cortisol response was reported in FMF patients without amyloidosis in the face of stressful situation during attack-free periods (7).Sub clinical adrenal insufficiency was reported in most FMF patients with AAamyloidosis (8).Based on these data, it could be argued that small dosages of steroids may compensate for the possible cortisol deficiency in FMF patients complicated by amyloidosis. Steroids are also used to suppress inflammation and prevent interstitial inflammation in the kidney likely to arise during long-term proteinuria (9).

In conclusion, I suggest that a combination of AZA and PRD in addition to colchicine could be useful in regression of NRPin selected cases with FMF complicated by AA amyloidosis. I also suggest that further controlled studies be conducted for a better emphasis of this aspect.

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ed to a tertiary referral centre where he received thalidomide for 6 months with significant improvement in clinical features as well as neutrophil count. However he developed peripheral neuropathy, which prompted immediate discontinuation of Thalidomide therapy.

His clinical feature of intermittent orogenital ulcerations continues with Neutropaenia. His disease has proven difficult to treat and currently anti-TNF therapy is being considered.

We have described an Englishman who was diagnosed with Behçet's disease (BD) associated with myelodysplastic syndrome (MDS) (1). Our literature search reveals 14 cases of Behçet's disease (BD) associated with MDS, mostly reported from Japan. (2-4). There is no report of such a case in a Caucasian patient from Great Britain.

A strong association has been shown between BD and HLA B51 in ethnic groups extending from Middle East to Japan (5). It is interesting to note that HLAtyping of our patient was negative for HLAB51.

In a study of 10 patients with MDS and BD the production of reactive oxygen species by neutrophils was investigated by luminal enhanced chemo-luminescence assay and the authors concluded that Trisomy 8 predisposes to BD in patients with MDS (2). Cytogenetic analysis of our patient revealed Trisomy 8.

Although BD is characterised by a dramatic response to steroids, our patient had only a modest response. Thalidomide was tried with success although it had to be withdrawn due to peripheral neuropathy. In a study conducted by the University of Istanbul, thalidomide was effective in resistant cases of BD (6). In 2001 and 2003 two similar cases were reported, one associated with Leishmaniasis and the other was again in a Japanese patient with elevated cytokines (7, 8).

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