levels linked with vascular thrombosis. However, I have two important questions to the authors:

1. The first Behçet patient was treated with chloroquine, colchicine and warfarin. To our knowledge, chloroquine is used for neither the treatment nor management of Behçet’s disease and no study has found that chloroquine is effective on any aetiological factor related to the pathogenesis of the disease.1–4 Therefore, what was the indication of chloroquine for the treatment of Behçet’s disease? If there is still an indication for chloroquine, why did the second patient not receive it? I think the authors should first clarify these statements.

2. To my surprise, the disease was named as ‘Adamantiades–Behçet’s disease’. Many physicians before Hulusi Behçet (1889–1948) reported various findings of this disorder including Hippocrates BC, Janin (1772), Neumann (1895), Reis (1906), Blüthe (1908), Weve (1923), Grütz (1926), Kumer (1930), Adamantiades (1930), Whitwell (1934), Nishimura (1936) and Blobner (1937).6 However, none of these papers and many others, not detailed here, indicated a new or a single syndrome with a ‘classical triad’. However, Dr Hulusi Behçet was the first physician to recognize a set of dermatological, ophthalmic and orogenital lesions, to group all these manifestations into one disease, and to then describe the results between 1937 and 19407–9 as a ‘triple symptom complex’.

Therefore, the authors should address the reason for using the eponym ‘Adamantiades–Behçet’s’ in their paper. PubMed revealed more than 5703 articles and tens of thousands of citations that used the eponym ‘Behçet’s’. Similarly, JEADV has published just 14 papers on Behçet’s disease since 1998, two from us, with the eponym ‘Behçet’ without exception. Moreover, the American Behçet’s Disease Association, International Behçet’s Society, and Japan Behçet’s Disease Research Committee call this disease ‘Behçet’ to honour the first describer of ‘triple symptom complex’. Furthermore, classical textbooks of dermatology, rheumatology and ophthalmology, and international symposiums and congresses again call this entity Behçet’s disease, not Adamantiades–Behçet’s. Therefore, if honour is to be given to all authors who reported some findings on this disorder, the disease would be entitled as ‘Hippocrates–Janin–Neumann–Reis–Blüthe–Weve–Grütz–Kumer–Adamantiades–Whitwell–Nishimura–Blobner–Behçet disease’.6 Why should only Adamantiades be honoured among all these authors? On the contrary, Adamantiades himself named the disease as ‘Behçet’ in his one subsequent paper published just 12 years after the original article of Hulusi Behçet.10

People who have a good knowledge of historical facts and follow positive sciences use the eponym ‘Behçet’. Apart from of medicine itself, authors must have a well-rounded knowledge of things. It is time to abandon efforts to change the name of an old disorder well known for more than 65 years by every physician in the world, as well as by medical students and the public, as ‘Behçet’s disease’.

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Behçet’s disease associated with hidradenitis suppurativa

Editor

Behçet’s disease (BD) is an inflammatory systemic disorder of unknown origin, characterized by recurrent aphthosis.

This case was presented as a poster at the ‘14th EADV Congress’, 12–15 October 2005, London, UK.
genital ulcers, uveitis and skin lesions. Cutaneous manifestations may include neutrophilic dermatoses such as Sweet’s syndrome and pyoderma gangrenosum, although these are unusual in BD. Hidradenitis suppurativa (HS) is one of the less commonly appreciated complications. We describe here an unusual case of BD coexisting with HS.

A 24-year-old woman was admitted to our outpatient clinic complaining of erythematous, tender and edematous nodular lesions on her axillary region. Owing to a history of idiopathic anterior uveitis and the recurrent aphthosis the diagnosis of BD had previously been suggested and she had been given colchicin 500 mg BID for the past 2 years. At the time of admittance to our clinic she had no signs of active BD but did have signs of hidradenitis suppurativa. Her full blood count and chemical profiles were within normal limits. We administered doxycyclin 200 mg/day, after which she completely recovered.

Hidradenitis suppurativa (HS) is a relapsing, inflammatory disease originating in the apocrine gland follicles, which may become chronic and often indolent because of subcutaneous extension, with induration, scarring, destruction of skin appendages and sinus formation. It is characterized by the development of tender, red nodules, which at first are firm but later become fluctuant and painful. The initial event is follicular keratinization with resultant plugging of the apocrine duct, followed by a severe inflammatory response in the apocrine gland. On the other hand, BD is an idiopathic, multisystemic, inflammatory disorder that can affect major organs and consists of recurrent oral aphthous ulcers, recurrent genital ulcers, uveitis and skin lesions. The most common skin manifestations of BD are erythema nodosum on the lower legs, and pseudofolliculitis, papulopustular lesions and acneiform nodules on the back. Trombophlebitis, Sweet’s syndrome and pyoderma gangrenosum are more exceptional.

In vitro and in vivo studies have shown that overactive neutrophils play a key role in the pathophysiology of BD. The enhanced expression of adhesion molecules may be responsible for the tissue neutrophilia and may induce the dense infiltrate surrounding the sweat glands and/or hair follicles in patients with BD. Thus, these observations can explain the association between HS and BD.

To our knowledge, our case is the second case of HS with BD. We report this case to draw dermatologists’ attention to the possibility that HS is related to BD.

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A case of keratoacanthoma centrifugum marginatum with a curious mast cell accumulation at tumour sites

Editor
We report on a 60-year-old Caucasian man presenting with a 3-year-old lesion on the buttocks diagnosed as advanced keratoacanthoma centrifugum marginatum (KACM), which curiously was associated with mast cell (MC) accumulation. As far as we know, there is no report indicative of the presence of MC in keratoacanthoma lesions. A dermatological examination revealed the presence of a rounded lesion area about 40 cm in diameter, formed by dome-shaped erythematous multinodular plaques occupying most of the buttocks. Clinical and histopathological features were compatible with KACM.

Histopathological study, based on the content of sulphated glycosaminoglycans, showed two MC subtypes: mucosal (alcan-blue-positive), containing low levels, and connective tissue (safranin-positive), with high levels. In the normal tissue all MCs were alcan-blue-positive (fig. 1a), while in KACM lesions a large number of MCs was found located within the core of the tumour (fig. 1b,e), with 2% of MCs safranin-positive and 98% of MCs alcan-blue-positive (fig. 1b). Although we found few connective tissue MCs, the presence of this subtype might by strongly implied in KACM pathogenesis, as they contain secretory granules rich in heparin proteoglycans. Recent findings have shown that MC suppresses the growth of tumour cells through an indirect mechanism involving heparin and fibroblast adjacent to the tumour cells. In the present study, we propose that the MC accumulation in the