Correspondence

Behçet's disease and pregnancy

SIR, At the 3rd International Conference on Behçet's Disease it was proposed that hormonal factors may influence the pathogenesis of this disease.¹ A variable influence of pregnancy has been reported. Madkour and Kudwah reported exacerbation of mucocutaneous and arthritic manifestations in four women,² and Plouvier and Devulder,³ Ferraro *et al.*⁴ and Suchenwirth⁵ have reported remission during pregnancies.

We studied 21 pregnancies in eight women with Behçet's disease, who each had at least three major criteria for diagnosis. The disease was classified as mucocutaneous in five, arthritic in one, and of the ocular type in two.⁶ Clinical manifestations were oral ulceration (eight patients), genital ulceration (eight), erythema nodosum (six), positive pathergy test (five), and uveitis (two). The histocompatibility antigen HLA-B5 was present in five of six patients tested.

Table Influence of pregnancy on Behçet's disease

Patient No	Pregnancy (n=21)	Pregnancy remission (n=12)	Pregnancy exacerbation (n=9)	Aggravation, clinical features
1	4	3	1	EN
2	1	1	0	
3	3	3	0	
4	2	2	0	
5	3	1	2	GA, EN, NPF
6	2	1	1	BA, GA, EN
7	4	0	4	BA, GA
8	2	1	1	GA

EN=erythema nodosum; GA=genital aphthosis; NPF=necrotic pseudofolliculitis; BA=buccal aphthosis.

The influence of pregnancy on the course of the disease varied between patients and during different pregnancies in the same patient (see Table). Remission without any treatment occurred during 12 pregnancies while exacerbation of disease occurred in nine pregnancies despite prednisone 10-15 mg daily. These exacerbations were usually of painful genital ulceration during the third trimester.

Behçet's disease did not influence any pregnancy, there being no abortions, prematurity, or perinatal deaths.

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Does AIDS 'cure' rheumatoid arthritis?

Rheumatoid arthritis (RA) is a chronic, symmetrical, inflammatory polyarthritis of unknown cause. In the aetiopathogenesis of RA, cell-mediated immunity involving T lymphocytes has a pivotal role. One of the early findings in RA is the perivascular accumulation of T lymphocytes of the helper-inducer phenotype (CD4 positive) in the synovium. The number of suppressor-cytotoxic T lymphocytes (CD8 positive), however, is relatively decreased. It is possible that local inadequate T lymphocyte suppression encourages a chronic, self perpetuating inflammatory response in the joints.¹ It is also well known that immunosuppressive treatment can be effective in RA. One can speculate on the effects of human immunodeficiency virus (HIV) on RA. The main target of the HIV is the CD4 positive T lymphocyte, which has been shown to bind the envelope glycoprotein of HIV. The ensuing destruction of CD4 bearing lymphocytes probably accounts for the immunosuppressive effect of the virus.² If the aetiopathogenesis of RA is as stated, RA activity should decrease after infection with HIV. We observed a patient in whom this might have happened.

In July 1985 a 60 year old, single man was admitted with severe polyarthritis of two months' duration. He had morning stiffness exceeding two hours, weight loss (8 kg), and was unable to take care of himself. On examination there was symmetrical arthritis of the second and third metacarpophalangeal joints, both wrists, both knees, and some metatarsal joints, and subcutaneous nodules at both elbows. His erythrocyte sedimentation rate (ESR) was 51 mm/h; he had a normochromic, normocytic anaemia, thrombocytosis, and an increase in total serum proteins, especially gammaglobulins. Liver function tests were normal. Rheumatoid factor was negative; x rays of hands and feet were normal. Histology of a subcutaneous nodule showed an inner necrotic core with destroyed collagen and