Annals of the Rheumatic Diseases, 1988; **47**, 350–352

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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 Suchenwirth[5] 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

fibrin and a radially arranged pallisade of mononuclear cells. A diagnosis of definite RA was made. He was unable to tolerate an adequate dosage of different non-steroidal anti-inflammatory drugs because of gastric complaints (he had a history of gastric bleeding in 1970 and 1977) but responded rapidly to intramuscular gold. The number of tender and swollen joints decreased; his ESR was 24 mm/h in December 1985 and 12 mm/h in March 1986, and he gained weight. In June 1986 he started to complain of fatigue and further weight loss. Physical examination showed normal joints and no other abnormalities. In the following months his general condition deteriorated, but his RA remained in remission. He continued to lose weight, developed fevers, and felt worse every day. Further review disclosed hepatosplenomegaly and enlarged lymph nodes in both axillae. He had a candida stomatitis, and serological tests showed evidence of an old hepatitis B as well as a cytomegalovirus infection and recent (early antibodies) Epstein-Barr virus infection. He developed livedo reticularis and skin discoloration with painless nodules on both legs, resembling Kaposi's sarcoma; this was proved by biopsy. Antibodies to HIV were detectable in the enzyme linked immunosorbent assay (ELISA) and in the immunoblotting test. The diagnosis of acquired immune deficiency syndrome (AIDS) was thus established.

Of course the remission of RA just before the onset of AIDS could be fortuitous. Alternatively, latent HIV infection might have reduced the severity of his RA. Unfortunately no serum is available from the onset of the arthritis to check this hypothesis. Theoretically it is possible that RA will regress in patients developing AIDS. We wonder whether others have made similar observations.

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Book review

CIBA Foundation Symposium 129: Autoimmunity and Autoimmune Disease. Edited by Ian Mackay. Pp. 278. £28·95. John Wiley: Chichester, Sussex. 1987.

This symposium was held at the Ciba Foundation between 30 September and 2 October 1986, at the suggestion of Ian Mackay, to take stock of current understanding of the immune response and its regulation, so as to consider whether more specific and rational immunotherapy can be developed for autoimmune disease.

The participants' presentations are a reasonable sample from the field of autoimmunity, given the limits of a small symposium: autoimmune myocarditis (Rose), nuclear autoantigens (Tan), cell surface carbohydrates (Feizi), tolerance (Nossal), experimental interstitial nephritis (Neilson), HLA class II expression (Feldmann), idiotype connectivity (Kearney) and cross reactivity in rheumatoid factors (Carson), immunodeficiency (Rosen), complement abnormalities (Lachmann), interferons (Revel) and immunotherapy by means of anti-Ia antibody (McDevitt), antibodies to T cell subsets (Waldmann), idiotype/anti-idiotypes (Roitt), and immunoabsorption (Lockwood, presented by Savage). A reader of five to 10 years ago would not find a radical change in the views of autoimmunity expressed here; however, there comes across from the papers and their discussion a sense of more convincing evidence for the multiplicity of underlying mechanisms and clearer, but even more intriguing, hints about the nature of autoantigens driving the autoimmune process, notably in the connective tissue diseases; also that immunotherapy is a more complex undertaking than thought previously, but still desirable and feasible.

Papers and discussions are both furnished with references (a useful feature) and the volume is well indexed. The reader who is not actively engaged in one of the relevant areas of research will find it helpful in keeping up to date with the field. It is worth a place on the bookshelf.

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Spondyloarthropathies: Involvement of the Gut. Eds. H Mielants, E M Veys. Pp. 456. Dfl 275.00. Elsevier: Amsterdam. 1986.

In September 1986 there were 47 participants at the first conference on spondyloarthropathies in Ghent. It was obviously a friendly meeting and of an ideal size for informal discussion, all of which has been recorded faithfully. The purpose was an exchange of ideas between representatives of different disciplines, considering together work which had been published previously. This volume stands as a useful record of the formal presentations and the discussion sessions.

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