various programs, as part of the training schedule or as a warming-down/relaxation procedure after an exercise session. Along with walking, warm water activities are best tolerated, but there are no contra-indications to endurance activities.

Compliance has been described as one of the major problems in exercise programs for FM patients. An inactive lifestyle is associated with poor physical fitness, and pain may further discourage participation in physical activities. If exercises are started in the deconditioned body, the unaccustomed physical activity may lead to more complaints and to further avoidance of physical activity. A vicious circle leading to less and less activity may ensue. This vicious circle is probably one of the reasons of low rates of volunteering, poor compliance with the program and/or program follow-up prescriptions, or drop-outs.

Self-management based programs will be illustrated with examples drawn from the literature and from our experience in Geneva.

Review session N° 3: Pregnancy and R. D. – Friday 15 June, 10.00-11.45/South Hall

SP0068 PREGNANCY AND RHEUMATOID ARTHRITIS

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10.1136/annrheumdis-2001.22

Women will hardly ever get their first signs of rheumatoid arthritis (RA) during pregnancy, whereas the onset of RA seems to cluster in the postpartum period. This clinical and epidemiological observation may be explained by a postponement of the onset of symptomatic RA by pregnancy, which is in line with the observed amelioration of disease activity in women with established RA. Up to 75% of women with RA improve during pregnancy, starting in the first or second trimester with maximum improvement during the third trimester of pregnancy. More than 90% of these patients will experience a flare of their disease within two to six months after delivery. Women who have ever been pregnant have a lower risk of developing RA, particularly when they had their first pregnancy at an early age. Several biological mechanisms have been put forward to explain the favourable effect of pregnancy on the onset and course of RA, but there is still no fully satisfactory explanation. The profound hormonal shifts (e.g. increased estrogens, progesterone and corticosteroids) have been implicated in the favourable effect of pregnancy through their effect on the immune system. Pregnancy is an immunological challenge requiring immunological protection or tolerance for the partly non-self fetus. Cellular immunity is decreased during pregnancy, which may play a role in the positive effect of pregnancy in RA. Pregnancy is associated with decreased production of TH1 associated cytokines (IL2, interferon gamma), increased production of TH2 associated cytokines (IL-4, IL-10), and decreased production of proinflammatory cytokines (TNF alpha, IL-12). Alterations in the glycosylation of IgG have also been put forward as a possible explanation. The question why not all women improve during pregnancy is still not answered by the above mechanisms. There is some evidence that HLA disparity between mother and fetus will play a role in the suppression of autoimmunity, which will differ between different pregnancies. A better understanding in

how pregnancy exerts its effect on the onset and course of RA will help to unravel possible immunological mechanisms in RA and may lead to more effective treatments.

SP0069 PREGNANCY OUTCOME AND ANTI-RO/SSA ANTIBODIES

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10.1136/annrheumdis-2001.23

Autoimmune phenomena seem to play a major role in the pathogenesis of unexplained pregnancy loss. The strong association of antibodies against ribonucleoproteins and neonatal lupus syndrome became soon apparent. Neonatal lupus syndrome, a classical model of passively acquired autoimmunity, results from the transplacental transfer of IgG anti-Ro/SSA between the 20th and the 24th week of pregnancy causing histopathological damage on developing tissues. It consists of congenital heart block (CHB), non-scarring cutaneous lesions, hepatitis, haemolytic anaemia and thrombocytopenia. A recent report supports that a significantly greater number of mothers whose children had CHB were asymptomatic compared with mothers of children with cutaneous disease. Specific maternal MHC class II genes seem to correlate with specific outcomes in neonatal lupus syndrome. A Japanese study suggests that certain maternal specific haplotypes were associated with CHB and cutaneous lesions.

The exact mechanism through which antibodies against ribonucleoproteins affect the fetal conductive system remains largely unknown. Maternal IgG anti-Ro/SSA and anti-La/SSB antibodies have been isolated from affected fetal hearts, while studies on human and animal models revealed possible arrhythmogenic effects of these antibodies. Apoptosis is proposed as a mechanism of translocation of intracellular Ro/SSA and La/SSB antigens to the cell surface of fetal cardiac myocytes, where they can be bound by cognate autoantibodies. In addition, it was shown experimentally that embryonic expression of certain constituents of the Ro/SSA molecule occurred some weeks before the clinical detection of CHB. Particularly, hYRNAs molecules between the 6th and 16th week of gestation, while an alternative 52b protein, recognised by the sera of mothers whose children had CHB, between 14–16 weeks. Finally, antibodies against a human endogenous retrovirus (ERV-3), which is expressed in fetal tissues particularly in the heart, were found to be elevated in mothers of children with CHB. IgG antibodies against a 57 kd protein and calreticulin are also proposed as additional risk factors in the pathogenesis of neonatal lupus syndromes.

Based on the hypothesis that unexplained pregnancy loss in autoimmune patients may represent intrauterine expression of neonatal lupus, several studies explored the possible association of anti-Ro/SSA autoantibodies and pregnancy loss in autoimmune patients. In initial studies such association was confirmed only among black women with SLE. Subsequently, in a large cohort study it was revealed that although the presence of anti-Ro/SSA antibodies did not affect the pregnancy outcome in SLE patients, it was strongly correlated with recurrent pregnancy loss in non-SLE patients, including patients with Sjögren's syndrome, rheumatoid arthritis and scleroderma. In a further study, a clear gradation risk between recurrent pregnancy loss and the number of different Ro/SSA peptides (Ro52, Ro60, p57) recognised by the sera of autoimmune patients was demonstrated. It is of interest that the association of these peptides with adverse pregnancy outcome did not alter by the presence of other autoimmune