JOINING THE IMMUNOLOGICAL DOTS IN RECURRENT MISCARRIAGE

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ABSTRACT

While raised cellular immunity mediated by T helper (Th) 1 type cells may be harmful for the developing embryo/foetus, it is likely that Th2 type immunity may be helpful. The role of natural killer (NK) cells is presently underestimated, although they are clearly important in angiogenesis and the coordinated invasion of the decidua by the trophoblast. Deficient T regulatory cell (Treg) function is evident in women with recurrent miscarriage particularly when this occurs in early pregnancy. The role of the pro-inflammatory Th17 cells is presently unclear. However, early evidence suggests that excessive Th17 activity may promote miscarriage and preterm delivery.

This may relate to the ability of these cells to produce those cytokines that encourage Th1 and NK cell activity. As such recurrent miscarriage may be caused not only by chromosomal abnormalities, autoimmunity and uterine abnormalities but also by subclinical uterine infection and inflammation which by stimulating interleukin 6 favours Th17 development over Tregs. This review examines the role of these different cells in early pregnancy and suggests a schema that may join the dots of the immunological puzzle called pregnancy. Finally, suggestions are made as to how inappropriate immunity in recurrent miscarriage may be down-regulated using currently available therapies.

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