Desferrioxamine, a metal chelating agent with high affinity for iron, has been found to suppress tissue injury in animal models of inflammation. Few studies have been performed in humans, and these have given conflicting results. Giordano et al showed an improvement of clinical indices and a significant increase of serum iron and haemoglobin with a marked progressive decrease of serum ferritin in patients with RA with hyposideraemic anaemia in a short desferri- oxamine treatment. Fudman et al by intrar- ticular administration of desferrioxamine noted predominantly systemic effects with decreased serum ferritin and decreased serum concentrations of lipid peroxidation pro- ducts.10 They treated patients with RA with larger doses of desferrioxamine and found serious side effects consisting of loss of consciousness and of pigmentary reti- pathy.11 Pall et al observed ocular toxicity after a low dose of desferrioxamine.12 Poisson et al found no significant changes in rheuma- tological indices or in immunological markers of disease activity of patients with RA re- fractory to conventional treatment and receiv- ing desferrioxamine for six months.13

Recent studies have shown that desfer- oxi-oxamine affects lymphocyte function. It can inhibit proliferation of human lymphocytes, promote the secretion of lymphokine, re- ductase and DNA synthesis.14 It is found that desferrioxamine treatment impairs the expres- sion of interleukin 2 binding receptors on lymphoid cells in response to mitogen15 and markedly reduces interleukin 2 production by mitogen stimulated cells.15

In this study we noted a statistically signifi- cant improvement of clinical indices in the patients with RA at the 28th day. Patients had no side effects. A notable increase of serum ferritin, microglobulin concentrations was noted in all patients at the 14th day followed by a statistically significant decrease at the 28th day in patients with more active disease. This increase in ferritin, microglobulin at the end of desferrioxamine administration (14th day) without a worsening in clinical indices has not been explained. It may be due to a polyclonal B lymphocyte response or a treatment effect in these patients determined by desferrioxamine displacement of iron or other metals. Polyclonal B lymphocyte activators are mitogens that non-specifically stimulate lymphocytes to secrete immuno- globulins, and both groups of patients had significantly increased concentrations of imm- unoglobulins after desferrioxamine treat- ment.

High serum ferritin concentrations seem to correlate with the severity of arthritis involve- ment. In this study patients with more active RA had higher serum ferritin concentrations than patients with less active disease. On the other hand, low serum ferritin concentrations may be connected with iron deficiency and in this study patients with RA and low serum ferritin concentrations had lower serum iron and higher transferrin and iron binding capacity than patients with higher serum ferritin.

In patients with severe active RA the as- sociation of raised concentrations of ferritin and β2 microglobulin suggests that conspicuous iron deposits may play a part in the stimu- lation of lymphoid cells. Desferrioxamine seems to reduce lymphocyte activation and function, probably by inhibiting DNA syn- thesis and interleukin 2 action in these pa- tients. In patients with less active RA and low or normal serum ferritin concentrations the desferrioxamine effects do not seem to influence lymphocyte function.

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