Introduction and Aim of the work

Insulin dependent diabetes mellitus is an autoimmune disease caused by a selective autoimmune process that gradually destroys the insulin producing B cells (Rossini et al., 1991).

Genetic suscptibility and environmental factors seem to be important causal factors. The presence of autoantibodies in the sera of diabetic patients such as islet cell cytoplasmic antibodies (ICA) and islet cell surface antibodies (ICSA) has been reported (Srikanta et al., 1986). and Palmer et al., (1993).

In addition, an increasing number of other autoantibodies and abnormalities of cell mediated immunity are frequently observed in type 1 diabetic patients (Lorini et al., 1993).

Anticardiolipin antibodies (ACL) directed against negativily charged phospholipids are frequently found in sera of S.L patients and appear to predispose to many complications of this disease especially thromboembolic complications (Sammaritano et al., 1990).

A number of proposals have been reported to explain thrombosis associated with APL syndrom including inhibition of prostacyclin generation by endothelial cells, decreased activity of protein C system, impaired fibrinolysis and inhibition of beta 2 Glycoprotein 1 (Shortell et al., 1992).

Anticardiolipin antibodies (ACL) may be primary or secondary to auto immune disorders, drugs or certain infections (Fong & Boey, 1992). Anticardiolipin are also found in other autoimmune diseases even in children (Ravelli et al., 1990).

Anticardiolipin have been recently found in mice with streptozocin induced diabetes (Anzai et al., 1993). Appear with hyperglycaemia, increased and the decreased.

Recently *Lorini et al.*, 1995 reported high incidance of ACL IgG in diabetic patients no correlation with HLA typing.

The Aim of the work:

The aim of the work was to evaluate ACL IgG and IgM in type I diabetic patients and to study the possible correlation with duration of the disease, other autoantibodies and presence of microangiopathic complications.