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Desferal prevents cardiac fibrosis following diabetes mellitus I

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Introduction: Diabetic cardiomyopathy, a life threatening morbidity of diabetes mellitus (DM) is defined as combination of heart hemodynamic dysfunction and cardiac tissue fibrosis. Insulin therapy is the treatment of choice for DM type I, but usually unable to control diabetic cardiomyopathy. As an inflammatory process, intracellular iron overload can be the reason of heart oxidative stress and fibrosis production. Therefore we tried to compare desferal (iron chelating agent) with insulin the DMTI classic treatment, to prevent diabetic cardiomyopathy.

Material and Method: 60 Male 200-250 g, Wistar rats living in standard situation for water, food, circadian cycle and temperature were used. They were divided to two groups: DM and intact. Each group was subdivided to insulin (I) or desferal (D) or saline (S) treated. To induce DM, single dose of STZ, (60 mg/kg) was injected IP. One week later, rats with BS higher than 200 mg/100 were considered as diabetic. Then treatments started, daily for 8 continuous weeks. At the end of 8th week rats were sacrificed. Cardiac evaluations included: tissue IL-6 concentration level, Masson's trichrome to differentiate collagen, H&E to evaluate cellular morphology, Perl's staining for hemosiderin particles and tunnel test at last to evaluate apoptosis.

Results: Our study showed a high concentrations of collagen fibers, in DM+(I), compared with DM+(D)group ($P<0.01$), both H&E and tunnel confirmed higher ranges of pyknosis and nuclear fragmentation and apoptosis in DM+(I)group, compared with DM+(D) ($P<0.001$). Perl's staining showed no meaningful difference between hemosiderin in DM+(D) group compared with intact+(S). At last, IL-6 both in plasma and tissue was lower in desferal treated diabetic groups ($p<0.1$)

Conclusion: Our finding showed a decrease in fibrotic tissue formation following desferal therapy compared with insulin in DM, compared with insulin therapy. Therefore we can propose desferal as a cardio protective agent in DM and possibly a precious candidate to prevent diabetic cardiomyopathy.

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