This study was carried out on 70 diabetic men (and 16 normal men as a control) who were attending the diabetic outpatient clinic of Kasr-Al-Aini University hospital, Diabetes Institute, and T.V. Medical Center since 1997. On the basis of history taking ،clinical examination, and laboratory investigation, they proved to be free from hepatorenal disorders, schistosomiasis and infectious diseases. They were under treatment [diet, oral hypoglycemic agents (gliclazide = diamicron), or insulin].

Diabetes Mellitus was diagnosed on the following criteria:

(1) History: previous history of polyuria, polydepsia, and polyphagia, history of loss of weight, family history suggestive of diabetes.

(2) Urine examination: some patients showed glycosuria.

(3) Two hours oral glucose tolerance test using 75 gm glucose orally.

Diabetic patients were divided to two subgroups according to age and onset of the disease:

A) Type I: Insulin-Dependent Diabetes Mellitus (I.D.D.M.): Juvenile-Type (25 patients)

B) Type II: Non-Insulin Dependent Diabetes Mellitus (N.I.D.D.M): Adult-Type (45 patients)

NIDDM was further subdivided into patients taking and responding well to oral hypoglycemic drugs according to their fasting and post-prandial blood glucose (21 patients), and those not responding well to oral hypoglycemic drugs and taking insulin (24 patients).

-The control group comprised 16 healthy male volunteers.

The diabetic patients and control were screened for the following laboratory investigations:

(1)Fasting and two hours blood glucose.

(2)Glycosylated hemoglobin HbA1c.

(3) Serum C-peptide.

(4) Serum autoantibodies against Islet Cell Antigens (ICA).

(5) Serum autoantibodies against Glutamic Acid Decarboxylase (GAD).

(6) Serum Vascular Cell Adhesion Molecule-1 (VCAM-1).

(7) Serum E-Selectin.

Diabetic patients were further classified into:

Diabetic patients free of GAD and ICA autoantibodies (n= 26 patients) and diabetic patients proved to have GAD and ICA autoantibodies (n= 27 patients).

**The Prevalence of Islet Cell autoantibodies (ICA) in Type I & II Diabetes Mellitus**

The prevalence of Islet Cell autoantibodies (ICA) in IDDM group was (60%) which was higher than that of NIDDM group on insulin treatment (45.8%) and higher than of NIDDM group on oral treatment (19%). All controls had no ICA in their sera. In NIDDM patients with circulating ICA represented an early of a disease process culminating in IDDM. ICA might serve as an early indicator of the disease. An early detection of ICA is important in order to identify the individuals in general population who are at a high risk of developing the disease.

**The Prevalence of Glutamic Acid Decarboxylase autoantibodies (GAD) in Type I & II Diabetes Mellitus**

In the present study, the prevalence of Glutamic Acid Decarboxylase autoantibodies (GAD) in IDDM group was (72 %) which was higher than that of NIDDM group on insulin treatment (66.7%) and higher than that of NIDDM group on oral treatment (38.1%). GAD could not be detected in the control group. IDDM is an autoimmune disease in which the immune system mistakenly attacked and destroyed the beta cells of the pancreas. GAD could be of help in familial screening of type I. It has a better predictive value than ICA . GAD could also detect insulin dependency in NIDDM adult patients.

## Correlation Studies:

(1) There was a significant positive correlation between Islet Cell autoantibodies (ICA) and Glutamic Acid Decarboxylase autoantibodies (GAD) in NIDDM (oral, insulin) and IDDM groups.

(2) There was no significant correlation between Islet Cell autoantibodies (ICA) and C-peptide levels in NIDDM (on oral therapy) group but there were significant negative correlations between Islet Cell autoantibodies (ICA) and C-peptide levels in NIDDM (on insulin therapy) and IDDM groups. The presence of autoantibodies to the beta cells of the pancreas led to their destruction and loss of beta-reserve capacity and decrease in C-peptide level. C-peptide level had decreased significantly in ICA (+ve) patients but not in ICA (-ve) patients.

(3) There was no significant correlation between Islet Cell autoantibodies (ICA) and soluble Vascular Cell Adhesion Molecule-1 (sVCAM-1) in NIDDM (on oral therapy) group, but there was a significant positive correlation between Islet Cell

autoantibodies (ICA) and soluble Vascular Cell Adhesion Molecule-1 (sVCAM-1) in NIDDM (on insulin therapy) group, as well as in IDDM group. Adhesion molecules mediated leucocyte adhesion to the endothelium in the atherosclerosis process. The elevated levels of the soluble adhesion molecules in diabetic patients could be of pathogenic importance for the developing of atherosclerosis.

(4) There was no significant correlation between Islet Cell autoantibodies (ICA) and soluble E-Selectin (sE-Selectin) levels in NIDDM (oral therapy, insulin) and IDDM groups. Elevated levels of E-Selectin occurred independently of ICA which probably reflected ongoing immune response in diabetic patients.

(5) There was no significant correlation between Islet Cell autoantibodies (ICA) and glycosylated hemoglobin (HbA1c) levels in NIDDM (on oral and insulin therapy) and IDDM groups.

(6)There was no significant correlation between Islet Cell autoantibodies (ICA) and fasting blood glucose (FBG) levels in NIDDM (on oral and insulin therapy) and IDDM groups.

(7)There was no significant correlation between Islet Cell autoantibodies (ICA) and postprandial blood glucose (PPBG) levels in NIDDM (on oral and insulin therapy) and IDDM groups.

(8) There was no significant correlation between Glutamic Acid Decarboxylase autoantibodies (GAD) and C-peptide levels in NIDDM (on oral therapy) group, but there was a significant negative correlation between Glutamic Acid

Decarboxylase autoantibodies (GAD) and C-peptide levels in NIDDM (on insulin therapy) group as well as IDDM group. Differentiation between type I and II diabetes mellitus in adults was difficult only GADA was signed for insulin dependency in type II patients with lower C-peptide levels.

(9)There was no significant correlation between Glutamic Acid Decarboxylase autoantibodies (GAD) and soluble Vascular Cell Adhesion Molecule-1 (sVCAM-1) levels in NIDDM (on oral therapy) group but there was a significant positive correlation between Glutamic Acid Decarboxylase autoantibodies (GAD) and soluble Vascular Cell Adhesion Molecule-1 sVCAM-1) in NIDDM (on insulin therapy) group as well as IDDM group. Elevated sVCAM-1 levels in IDDM and NIDDM patients occurred in diabetic patients with vascular complications.

(10) There was no significant correlation between Clutamic Acid Decarboxylase autoantibodies (GAD) and soluble E-Selectin (sE-Selectin) levels in NIDDM (oral, insulin therapy) and IDDM groups. Increased levels of E-Selectin concentration occurred in patients with IDDM and NIDDM. E-Selectin was related to vascular complications in diabetic patients and concentration of E-Selectin might be related to metabolic control.

(11) There was no significant correlation between Glutamic Acid Decarboxylase autoantibodies GAD and glycosylated hemoglobin (HbA1c) levels in NIDDM (on oral and insulin therapy) and IDDM groups.

(12) There was no significant correlation between Glutamic Acid Decarboxylase autoantibodies (GAD) and fasting blood glucose (FBG) levels in NIDDM (oral, insulin therapy) and IDDM groups.

(13) There was no significant correlation between Glutamic Acid Decarboxylase autoantibodies (GAD) and postprandial blood glucose (PPBG) levels in NIDDM (oral, insulin therapy) and IDDM groups.

(14) Serum C-peptide levels in NIDDM on oral (Gliclazide=diamicron), insulin treatment and IDDM groups were significantly lower than that of control group. C-peptide had a discriminative power between type I and II diabetic patients and could be recommended as a classification tool.

(15) There was a significant negative correlation between C-peptide and soluble Vascular Cell Adhesion Molecule-1 (sVCAM-) levels in NIDDM (oral and insulin therapy) and IDDM groups.

(16) There was no significant correlation between C-peptide and soluble E-Selectin in NIDDM (oral, insulin) and IDDM groups.

(17) There was no significant correlation between C-peptide and glycosylated hemoglobin (HbA1c) in NIDDM (oral, insulin) and IDDM groups.

(18) There was no significant correlation between C-peptide and fasting blood glucose (FBG) in NIDDM (oral, insulin) and IDDM groups.

(19) There was no significant correlation between C-peptide and postprandial blood glucose (PPBG) level in NIDDM (oral, insulin) and IDDM groups.

(20) Soluble Vascular Cell Adhesion Molecule-1 (sVCAM-1) in NIDDM (oral gliclazide, insulin therapy) and IDDM groups were significantly higher than that of control group. sVCAM-1 had been associated with early stages of atherosclerosis and might reflect early vascular perturbation on diabetic vasulopathy and indicator of ongoing cellular dysfunction in diabetic patients and a marker of atherosclerotic lesions in diabetic patients.

(21) There was no significant correlation between soluble Vascular Cell Adhesion Molecule-1 (sVCAM-1) levels and soluble E-Selectin in NIDDM (oral, insulin) and IDDM groups.

(22) There was no significant correlation between soluble Vascular Cell Adhesion Molecule and glycosylated hemoglobin (HbA1c) in NIDDM (oral, insulin) and IDDM groups.

(23) There was no significant correlation between soluble Vascular Cell Adhesion Molecule (sVCAM-1) and fasting blood glucose (FBG) in NIDDM (oral, insulin) and IDDM groups.

(24) There was no significant correlation between soluble Vascular Cell Adhesion Molecule (sVCAM-1) and postprandial blood glucose levels (PPBG) in NIDDM (oral, insulin) and IDDM groups.

(25) sE-Selectin levels in NIDDM (oral gliclazide and insulin therapy) and IDDM groups were significantly higher than that of control group.

(26) There was no significant correlation between soluble E-Selectin and glycosylated hemoglobin (HbA1c) levels in IDDM, NIDDM (oral and insulin) and control groups.

(27) There was no significant correlation between soluble E-Selectin and fasting blood glucose levels (FBG) in NIDDM (oral, insulin) and IDDM groups.

(28) There was no significant correlation between soluble E-Selectin and postprandial blood glucose (PPBG) levels in NIDDM (oral, insulin)and IDDM groups.

## Conclusion:

(1) In NIDDM patients with circulating ICA represented an early of a disease process culminating in IDDM. The frequency of positive antibodies declined with duration of the disease. ICA might serve as an early indicator of the disease. An early detection of ICA was important in order to identify the individuals in general population who are at a high risk of developing the disease.

(2) GAD could be of help in familial screening of type one, better predictive value than ICA and IAA. GAD could also detect insulin dependency in NIDDM adult patients.

(3) C-peptide had decreased significantly in ICA (+ve) patients but not in ICA (-ve) patients. C-peptide levels were inversely correlated with ICA level. C-peptide had a discriminative power between type I and II and could be recommended as a classification tool.

(4) Adhesion molecules mediated leucocyte adhesion to the endothelium in the atherosclorosis process. The elevated levels of the soluble adhesion molecules in diabetic patients could be of pathogenic importance for the developing of atherosclorosis. Elevated levels of E-Selectin and sVCAM-1 in IDDM and NIDDM might occurred in diabetic patients with vascular complications.