Fetuin was first isolated in 1944 by **Pedersen K. O.** as a major plasma protein in fetus**.** Fetuin is produced by multiple organs (including liver, kidney and brain) during fetal development. It is more abundant in fetal blood, hence the name fetuin *(from Latin word fetus)*. Fetal calf serum contains more fetuin than albumin, while adult serum contains more albumin than fetuin.

Fetuin-A acts as inhibitor of vascular calcification; it reversibly complexes with calcium and phosphorus and increases their respective serum solubility in a mechanism reminiscent to that by which apolipoproteins solubilize lipids.

The aim of this work was to evaluate the circulating levels of fetuin-A, a well-described as possible inhibitor of calcification in chronic kidney diseases.

Vascular calcification is a very common complication in dialysis patients and this induces the development of cardiovascular disease.

The very high risk of cardiovascular death in patients with end-stage renal disease (ESRD) has become increasingly apparent over the last several years

Despite advanced dialysis techniques, patients with end stage renal disease (ESRD) frequently have complications of cardiovascular diseases.

The calcification process as seen in calcifying atherosclerosis is an active cellular process, which is controlled by calcification inhibitors and inducers **.** Physiological inhibitors of calcification (fetuin-A, osteoprotegerin

(OPG) and undercarboxylated- matrix-carboxyglutamic acid protein) may play a role in preventing the development and progression of ectopic calcification.

This study was carried out on 40 patients (29 males & 11 females) with chronic renal disease, their ages ranged from 25 to 56 years old. Age and sex matched 20 healthy individuals (12 males & 8 females) were included as the control group. Patients and control were classified according to estimation of GFR by Modification of Diet in Renal Disease (MDRD).

Group I: Included 20 healthy individual age and sex matched to patient groups, 12 males and 8 females, their ages ranged from 25-50 years.

Group II: Included 20 patients with chronic renal disease (stage 4) without dialysis. They were 14 males and 6 females, their ages ranged from 33-50 years.

Group III: Included 20 patients with end stage renal disease (stage 5) under regular dialysis. They were 15 males and 5 females, their ages ranged from 35-56 years. Patients were sustained by maintenance hemodialysis thrice weekly for at least 6 months before included in the study.

Patients were referred from the nephrology outpatient and hemodialysis unit of Benha University Hospital and blood samples were collected and subjected to the following investigations (GFR, blood hemoglobin , calcium , sodium , albumin , urea nitrogen , creatinine , uric acid , phosphorus , potassium , highly sensetive CRP , Cholesterol , LDL-

cholesterol , HDL-cholesterol and serum fetuin) which were done in Department of Medical Biochemistry, Faculty of medicine, Benha universty.

Our results showed significant decrease ($p<0.05$) of GFR, blood hemoglobin , calcium , sodium and albumin in both groups of CRF patients (group II & III.) compared with healthy control persons (group I).

Also, the results showed significant increase ($p<0.05$) of blood urea nitrogen , serum creatinine , uric acid , phosphorus , potassium and highly sensetive CRP in both groups of CRF patients (group II & III.) compared with healthy control persons (group I).

Cholesterol and LDL-cholesterol increased slightly in both groups of CRF patients , HDL-cholesterol was significantly increased in group II of CRF patients

Serum level of fetuin A was significantly decreased in the two diseased groups (58.7 and 53.5 mg/dl) as compared to the control group (70.1 mg/dl). On the other hand, serum phosphorous was increased in the two diseased groups (4.6 and 5.31 mg/dl). Therefore, the lower level of fetuin A in the patients will be associated with increased ectopic calcification and vascular calcification. The higher levels of phosphorous enhance phenotypic transition of vascular smooth muscle cells to chondrocytes or osteoblasts causing more vascular calcifications.

Conclusion : vascular calcification in CRD is enhanced by the low levels of serum fetuin A and predisposes to the high mortality.

Therfore serum fetuin-A is used as biochemical parameter in chronic renal failure patients on hemodialysis as inhibitor of vascular calcification.

Recommendations: Genetic elements are important as evidenced in the fetuin A polymorphism which are associated with calcification may indicate other lines of treatment.