KSN 2020
FULLY VIRTUAL MEETING

Abstract Type : Oral
Abstract Submission No. : OR-1017

Type II Diabetic induced oxidative stress and proinflammatory cytokines in renal cells leading to Acute Kidney Injury (AKI).

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Objectives: Acute Kidney Injury (AKI), biochemically characterized as abnormality in kidney function test which causes accumulation of creatinine and blood urea and functionally by a rapid decline in the glomerular filtration rate (GFR). Oxidative stress plays an important role in the development vascular complications in type 2 diabetes. Oxidant derived tissue injury occurs when production of oxidants or reactive oxygen species (ROS) exceeds local antioxidant capacity. Inflammatory cytokines such as tumor necrosis factor-alpha (TNF-a) and interleukin (IL-6) and various growth factors in renal cells modulate the local response are responsible for AKI.

Methods: 10 ml of fasting venous blood was collected from the anticubital vein in a plain, fluoride and EDTA vacutainers. The blood sample was centrifuged and stored at 4^0 C for biochemical and immunological investigations. The study group consisted of n=50 healthy individuals (Group I), n=25 Type II Diabetic without AKI (Group II), n=25 Type II diabetic with AKI (Group III) of either sex aged between 50-65 years. Type II Diabetic presented with clinical signs and symptoms of Acute Kidney Injury without Nephropathy. Serum levels of inflammatory markers (IL-6 & TNF-a), antioxidants (Glutathione reductase), plasma malondialdehyde (MDA), hs-CRP were estimated.

Results: Concentration of inflammatory molecules such as TNF-a 9.32±1.08, 14.04±1.42 and 36.56±10.50; IL-6 9.24±1.20, 14.14±1.50 and 36.76±11.56; hs-CRP 0.90±1.10, 1.96±0.50 and 2.18±0.90 was significantly elevated in Group III. GSH were significantly lower in both the groups of Diabetic with and without AKI when compared to controls. 7.10±0.58, 6.90±0.70 and 5.80±0.80. Mean value of total MDA 2.32±0.98, 8.68±2.50 and 9.80±2.72 was significantly more in Group III as compared to Group I and Group II.

Conclusions: Results of the present study indicates that inflammatory markers and oxidative stress are increased with decreased antioxidant defense levels in patients with AKI due to DM induced oxidative stress.

Table-1 Biochemical parameters in AKI

TABLE I- BIOCHEMICAL PARAMETERS IN NON-DIABETIC, DIABETIC WITH AKI AND WITHOUT AKI

INDIVIDUALS	FBS (mg/dl)	HbA1c (%)	Microalbumin (mg/g)	Cystatin-C (mg/L)	Urea (mg/dl)	Uric Acid (mg/dl)	Creatinine (mg/dl)
Normal n=50 (Group I)	91.60±10.44	6.28 ± 1.42	11.28 ± 3.22	2.10±1.13	30.50 ± 3.34	5.00±1.00	0.7±0.12
Diabetes without AKI n=25 (Group II)	180.88±20.54	8.98 ± 2.21	27.18 ± 4.32	4.88±2.32	45.48 ± 4.90	5.88±1.09	0.90±0.30
Diabetes with AKI n=25 (Group III)	253.30±28.12**	11.00 ± 2.06**	275.10±30.92**	5.25±2.50**	87.32 ± 10.29**	7.40±1.01**	2.50±0.44**

The data were expressed as mean ± SD. The data was analyzed using the student's t- test. * indicates p<0.05 and statistically significant, **indicates p<0.001 and statistically highly significant.

Table-2 Oxidative stress level in AKI

TABLE II- INFLAMMATORY AND OXIDATIVE STRESS LEVEL IN NON-DIABETIC, DIABETIC WITH AKI AND WITHOUT AKI

INDIVIDUALS	TNF-α (pg/ml)	IL-6 (pg/ml)	GSH (mg/dl)	MDA (µmol/L)	hs-CRP (mg/L)
Normal n=50 (Group I)	9.32±1.08	9.24±1.20	7.10±0.58	2.32±0.98	0.90±1.10
Diabetes without AKI n=25 (Group II)	14.04±1.42*	14.14±1.50*	6.90±0.70	8.68±2.50	1.96±0.50*
Diabetes with AKI n=25 (Group III)	36.56±10.50**	36.76±11.56**	5.80±0.80*	9.80±2.72**	2.18±0.90*

The data were expressed as mean ± SD. The data was analyzed using the student's t-test. * indicates p<0.05 and statistically significant, **indicates p<0.001 and statistically highly significant