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High Incidence of Acute Renal Failure with Oxidative Stress in Older Female

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ABSTRACT

Advance age, underlying as risk factor for the development of acute renal failure. Acute renal failure is a loss of renal function over a period of hours to days, as reflected in the glomerular filtration rate and it is usually considered a disease of the hospitalized patients. Reactive oxygen species are constantly formed within the body from oxidation-reduction reactions due to incomplete reduction of molecular oxygen. Superoxide anion radicals (O2·-), hydrogen peroxide (H2O2) and the highly reactive hydroxyl radical (OH·) are responsible for many of the biological effects of free radicals within the human body. Acute renal failure are associated with increased serum potassium, creatinine, urea and plasma malondialdehyde and hyponatremia in older age and decreased antioxidants enzymes are also associated with the disease.

KEYWORDS : Acute renal failure, Antioxidants, Oxidative stress

INTRODUCTION

Acute renal failure is a loss of renal function over a period of hours to days, as reflected in glomerular filtration rate[1] and it is usually considered a disease of the hospitalized patients.[2] Acute renal failure is a relatively common problem, affecting up to 5% of hospitalized patients and it is calculated approximately 1% of patients presenting to hospital–based emergency department.[3] Although this is often true, acute renal failure may occur in the outpatients setting. The likely cause of this condition differs significantly for patients in these two groups, so they are considered separately. Acute renal failure in the hospital setting is often iatrogenic a result of medical intervention. Advance age, liver disease, underlying renal insufficiency and diabetes (especially the presence of vascular disease) have been implicated as risk factor for the development of acute renal failure.[2] The most common causes of intrinsic acute renal failure in these patients were drug–related nephrotoxicity and infection, although renal vein thrombosis and cholesterol immobilization were also noted.

Pre-renal renal failure refers to origin of pathogenic process i.e. before the kidney. Hypovolumia due to inadequate circulating blood volume is the most common cause of pre-renal azotemia (Nitrogen substances in the blood that accumulate due to impaired elimination by the kidney).[4] Elderly patients are particularly susceptible to pre-renal azotemia because of their predisposition to hypovolumia and high prevalence of renal-artery atherosclerotic disease. The combination of angiotensin converting enzyme inhibitors and diuretics can cause pre-renal azotemia in patients with large vessel or small vessel renal vascular disease.[5,6,7] Patients with diminished renal perfusion, NSAIDs can precipitate pre-renal azotemia.[8,9,10] Acute tubular necrosis is the most common cause of intrinsic acute renal failure in hospitalized patients. Ischemia or toxins usually induce this condition. Ischemic acute tubular necrosis is frequently reversible, but if the ischemia is severe enough to cause cortical necrosis, irreversible renal failure can occur.[11,12] Acute tubular necrosis may be prevented by promptly treating patients with reversible causes of ischemic or pre-renal acute renal failure and by maintaining appropriate hydration in patients who are receiving nephrotoxins.[13]

Obstruction processes are the most common post-renal renal failure causes[4] and might result from blockage of the renal tubules as by acyclovir or uric acid crystallization or from blockage of the renal pelvis by stone or necrotic papillae (papillary necrosis).[3] Findings on examination may include hypertension, an abdominal mass representing a distended bladder, hydronephrotic kidney or obstructive tumor. The pathophysiology of post-renal acute renal failure starts with increased in the renal tubules because of the downstream obstruction. This in turn, leads to decreased glomerular filtration rate through several mechanism including direct hydrostatic effects on the glomerular as well as indirect effects on the renal circulation.[14]

MATERIALS AND METHODS

The clinical material for present study comprised 58 acute renal failure female patients attended in medicine ward S. G. M. Hospital, S. S. Medical College, Rewa (M.P.), India. The age range was taken from 30-80 years. Blood samples were collected from the patients at the time of admission. Clinical investigations were performed in the Clinical Biochemistry Section, Central Pathology Laboratory, Department of Pathology, S. S. Medical College, Rewa (M. P.), India. Serum glucose, protein (T), creatinine, urea, and superoxide dismutase were estimated by GOD-POD, biuret, jaffe's, diacetyl monoxime, and Misra H P et. al. methods respectively. Plasma malondialdehyde and haemolysate glutathione reductase, glutathione peroxidase and catalase were estimated by Jean C D et. al. method (1983), Horn H D (1963), Hafeman D G method (1974), and Asror K sinha method (1972) respectively. Serum electrolytes were estimated by flame photometric method. Obtained data were analyzed statistically by using student "t" test.

OBSERVATIONS

Table 1: Mean ± SD value and significant test between age group (30–50 year) and age group (51–80 year) in acute renal failure female patients

S. No.	Particulars	Mean ± SD (30–50 years) (n=30)	Mean ± SD (51–80 years) (n=28)	t-test	P-value
Electrolyte					
1	Serum Sodium ions (mEq/L)	127.07 ± 3.05	123.07 ± 3.24	4.843	< 0.001
2	Serum Potassium ions (mEq/L)	6.13 ± 0.28	6.73 ± 0.39	6.765	< 0.001
Biochemical Parameters					
3	Serum Glucose (mg/dl)	119.87 ± 3.84	121.36 ± 3.53	1.535	0.130
4	Serum Protein (Total) (gm/dl)	6.14 ± 0.21	6.09 ± 0.23	0.865	0.390
5	Serum Creatinine (mg/dl)	3.05 ± 0.35	3.28 ± 0.34	2.536	< 0.05
6	Serum Urea (mg/dl)	44.97 ± 1.30	48.89 ± 2.51	7.542	< 0.001
Antioxidant / Oxidant product					

7	Superoxide dismutase (EU/mg protein/ml)	9.50 ± 0.29	8.93 ± 0.45	5.773	< 0.001
8	Glutathione reductase (EU/gm protein)	17.99 ± 0.06	17.71 ± 0.05	19.233	< 0.001
9	Glutathione peroxidase (EU/mg % Hb)	7.52 ± 0.08	6.99 ± 0.06	28.383	< 0.001
10	Catalase (EU/mg protein/ml)	4.79 ± 0.06	4.33 ± 0.09	23.047	< 0.001
11	Plasma Malondialdehyde (nano mol/ml)	9.55 ± 0.28	9.91 ± 0.25	5.151	< 0.001

RESULTS

Results of this table revealed that serum creatinine was increased significantly (P < 0.05) in the age group 51 – 80 years of acute renal failure female patients. Serum potassium ions, urea and plasma malondialdehyde were increased significantly (P<0.001) in the age group 51–80 years of acute renal failure female patients.

Serum sodium ions, superoxide dismutase, glutathione reductase, glutathione peroxidase and catalase were decreased significantly (P<0.001) in the age group 51 – 80 years of acute renal failure female patients.

DISCUSSION AND CONCLUSION

Acute renal failure is characterized by tubular dysfunction with impaired sodium and water reabsorption and it has associated with the shedding and excretion of proximal tubule brush border membranes and epithelial tubules cells into the urine.[15] An evolving understanding of epidemiology and pathophysiology of acute organ dysfunction in the setting of critical illness has give rise to new concepts and terminology for a syndrome once known as either acute tubular necrosis or acute renal failure.[16] Acute renal failure most commonly in older adults and it is associated with significant mortality and morbidly, with death rates among hospitalized patients ranging from 25-70 %.[17,18]

Affects potassium excretion due to reduced nephron mass (number of functioning collecting ducts) and intrinsic impairment of active potassium secretion, because the number of collecting ducts is directly related to the glomerular filtration rate, renal failure whether acute or chronic, leads to impaired renal potassium secretion.[19] With acute renal failure, sustained decline in the glomerular filtration rate, which leads to accumulation of nitrogenous waste products and uremic toxin.[20]

The results presented in this study also demonstrated that significant (P<0.05) elevated concentration of serum creatinine and significantly (P<0.001) increased levels of potassium ions, urea and plasma malondialdehyde in female (51–80 years) have supported by some authors.[21,22] Hyperkalemia could be caused by an overall excess of body potassium or by a shift from inside to outside cells or hyperkalemia could be caused by the sudden release of potassium ions from muscle into the surrounding fluids.[23]

Reduced levels of sodium, superoxide dismutase, glutathione reductase, glutathione peroxidase, and catalase are explained significantly (P<0.001) in female (51–80 years) when compared to female (30–50 years). Reactive oxygen species are constantly formed within the body from oxidation-reduction reactions due to incomplete reduction of molecular oxygen, such as the superoxide radicals, hydrogen peroxide and highly reactive hydroxyl radical are responsible for many of the biological effects of free radicals within the human body.[24] Acute renal failure can be triggered or aggravated by reactive oxygen species but established acute renal failure per se might also affects the antioxidant defense mechanisms of the organism.[25] Superoxide dismutase is generally thought to play a central role because it scavenges superoxide anion at the initial step of the radical chain reaction.[26] Therefore, from above study we are concluding that acute renal failure is associated with increased serum potassium, creatinine, urea, plasma malondialdehyde and hyponatremia in older age and decreased antioxidants enzymes are also associated with the disease.

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