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A STUDY ON THE STAGE OF PRESENTATION OF ACUTE KIDNEY INJURIES TO CLINICS IN WESTERN NIGERIA

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ABSTRACT

Aim: The research was primarily set to assess the stages at which acute kidney injuries (AKI) are presented for clinical appraisal.

Methods: The research was carried out in southwest Nigeria. A total of 151 participants were included, 101 were AKI patients. The remaining 50 were apparently healthy individuals who had no history of AKI, who served as control. Estimation of plasma creatinine was carried out on the subjects' blood sample. Values were then subjected to statistical analysis using SPSS 17. The diagnosis and staging of AKI was based on history taken and a rise in plasma creatinine to set cut off points. AKI patients were therefore grouped into three with respect to the plasma creatinine content of the first blood sample collected on presentation. The three stages were titled 1, 2, and 3; 1 and 3 being mild and severe respectively.

Result: A significant rise in plasma creatinine was observed when the AKI patients were compared with control. The frequencies of patients at each AKI stage were 17, 5, and 79 for stages 1, 2, and 3 respectively. The age groups and frequency of AKI were: up to 10 years (31), 11 to 20years (6), 21 to 30 years (27), 31 to 4 years (33), 41 to 50 years (4), 51 to 60 years (5), and 60 years and above (6).

Conclusion: It appears that in the absence of metabolic diseases, the frequency of AKI is higher in children and between the age group of 21 to 40.

Key words: Acute kidney injury, Staging, Creatinine, Nephrotoxic substances

INTRODUCTION

The kidney is a vital organ. In order for the human system to function normally and for man to be relatively healthy, an inner chemical balance is necessary. This balance is primarily regulated by a pair of kidneys. The kidney clears the body wastes and maintains homeostasis with respect to water, electrolytes and pH (Bolarin, 2010). Summarily, the kidney can be said to perform three important functions. The first is the excretion of waste products and the preservation of essential solutes (Heusel et al., 1999). The second function is the regulation of water (Reeves et al., 1996; Maunsback et al., 1996), acid-base (Vanholder et al., 1995) and, electrolyte homeostasis (Newman and Price, 1999).The third is the endocrine function (Fine et al., 1991; Brown et al., 1992; Jelkman, 1992).The functional unit of the kidney is the nephron.The optimal functioning of the kidney requires an adequate number of structurally and functionally intact nephrons (Whitby et al., 1989). Kidney injury is said to occur when the kidneys are unable to perform their functions efficiently. Kidney injury could be acute or chronic. Whitby et al., (1989) defined acute kidney injury (AKI) as a renal disease of acute onset, but severe enough to cause failure of kidney function. In practice, acute kidney injury is diagnosed using the estimation of plasma creatinine levels but according to the Kidney Disease: Improving Global Outcomes (KDIGO) work group, the diagnosis and grading of acute kidney injury is based on either plasma creatinine level or the estimation of urine output. In this research, the use of plasma creatinine level is the assessment method of

choice since the assessment based on urine output in these patients is somehow severe and cumbersome (Leung et al., 2013). Stated below are the peculiarities of the KDIGO RIFLE classification method (Bellomo et al., 2013). The risk stage is diagnosed upon the finding of one of the following (i) a 1.5 fold increase in serum creatinine level,(ii) a more than 25% decrease in GFR or (iii) a urine output less than 0.5 ml/kg per h for 6 h. The Injury stage is diagnosed upon the finding of at least one of the following:(i) a two fold increase in serum creatinine level,(ii) a greater than 50% decrease in GFR or (iii) a urine output less than 0.5 ml/kg per hour for 12 hours. The third (failure) stage is diagnosed upon the finding of a threefold increase in serum creatinine level, or a 75%decrease in GFR, or an increase in serum creatinine level ≥4 mg/dl (≥354 µmol/l) with an acute plasma creatinine increase ≥0.5 mg/dl (≥44 µmol/l) or, Anuria for 12 hours. The Loss stage is diagnosed on the finding of a Persistent AKI with complete loss of renal function for more than four weeks. End-stage renal disease is diagnosed when AKI persists with complete loss of renal function for more than 3 months. As there is an increase in hospital attendance due to kidney related clinical conditions (Akpa et al., 2013), this research is designed to look at how early acute kidney injury cases are presented to the hospitals and, also to discuss the factors responsible for its development and progression through a toxicological point of view.

MATERIALS AND METHODS

Sources of samples

A total of 101 samples were collected from acute kidney injury (AKI) patients. Blood samples were collected upon the patients' first presentation in the hospital.

Inclusion and exclusion criteria

All subjects in this study were those who according to histor, developed the classical signs of AKI for not more than 3 months. All patients with history of hypertension and diabetes mellitus were excluded. All participants in this survey were therefore subjects known not be patients of metabolic diseases.

Determination of plasma creatinine was done using the Jaffe (1886) reaction based kinetic method as explained by Vaishya et al., (2010)

Determination of baseline plasma creatinine level. The mean plasma creatinine level of control subjects in this investigation was taken as the baseline plasma creatinine level. Stage 1 (Risk) <184 µmol/L; Stage 2 (Injury) ≥184 µmol/L but<276 µmol/L; Stage 3 (Failure, Loss of function and ESRD) >276µmol/L

RESULTS

Table 1. The mean, standard deviation (SD), Student's t when the creatinine levels of AKI patients were compared with the control

	Mean (µmol/L)	SD (µmol/L)	Student's t	Remarks
AKI	869	691	8.0	Significant
Control	92	12		

Table 2. Frequency of each AKI stage

Stage	1(Risk)	2(Injury)	3(Failure to ESRD)
Frequency	17	5	79

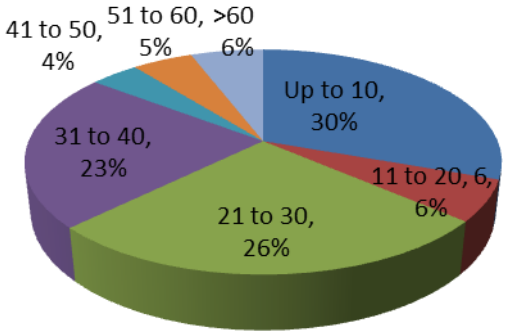

Fig 1 Distribution of AKI according to age

DISCUSSION

Plasma creatinine is a classical analyte used in the assessment of renal function (Burtis and Ashwood, 1999). It increases in plasma following renal injury. A significant variation was observed when the mean creatinine levels of AKI patients were compared with that of control (Table 1). There was also a significant variation when the mean creatinine levels of control were compared with that of Stages 1, 2, and 3 respectively. Table 2 shows a pattern of late presentation of AKI cases for clinical appraisal. Humans are exposed to a variety of potential nephrotoxic substances on a rather frequent basis (Guyton and Arthur, 1991; Isnard et al., 2004; Parazella, 2005; Schetz et al., 2005). Several therapeutic agents have known nephrotoxic potentials; classical examples include anti-microbial agents (Rougier et al, 2003, Izzedine et al., 2001), chemotherapeutic agents (Kintzel PE, 2001; Lamierre et al., 2005), analgesics (Elsevier and Debroe, 1999), and immunosuppressive agents (Gambaro et al.,

2003). Some of these drugs are prescription drugs while majority are over-the-counter drugs. A similar point is that Nigerians believe in the efficacy of alternative/complimentary therapy, herbal concoctions, infusions and mixes, maybe because it is most often relatively cheap, and more accessible. Moreover, Africans are faced with the menace of exorbitant cost of "western drugs". Though it is not the duty of the investigators in this research to doubt the efficacy of alternative therapy, we stress that more harm than good may be done if wrong diagnoses are made, or if the treatment is ineffective. A worst case scenario would be a situation in which one or more ingredients of the therapy are potential nephrotoxic phytochemicals. Similar circumstances are cases of product preservation and adulteration or substitution of a key ingredient (Isnard et al., 2004; Blowey, 2005), most probably to maximize profits. An increasing nephrotoxic concern is lead exposure, even at levels that are considered safe and acceptable to governmental agencies (Navas-acien et al., 2004). Other peculiarities and patterns could be unraveled if a critical look is taken at Fig I. The age brackets of up to 10, 21 – 30, and 31 -40 are the highest sufferers of AKI and the points are not farfetched, Parental carelessness and negligence or exacerbated susceptibility to AKI will likely be the reasons for the age bracket of 10 years and below, since this age range will solely depend on parental guidance and judgment. From a different perspective, the reason for an upsurge in AKI in the 21 – 40 years group could be due to the effect of self medication, riotous lifestyles, youthful exuberance, and indiscriminate use of performance enhancing or club drugs (Blowey, 2005). Recreational drugs and their metabolites have been known to have adverse effects on the kidney, some of them being chronic and irreversible but occasionally acute and reversible (Crowe et al., 2001). This observed trend disagrees with the work of Lewington and Kanagasundaram (2011), where AKI predominates in the elderly. This may be due to the fact that patients known to be suffering from hypertension and other diseases of the elderly that could have adverse effects on the kidney were excluded. The nephron dose of an individual is the number of nephrons an individual is born with, and it has been calculated to be in the range of 400000 to 800000 (Nyengard and Beenstein, 1992). The nephron dose has been proposed to be a determinant in the susceptibility of the individual to renal injury (Newman and price, 1999). If a nephron is lost, it can never be replaced. Since the functional unit of the kidney (The nephrons) is not regenerative in nature (Newman and price, 1999). The reason for the kidney being easily challenged or injured is not farfetched: The susceptibility of renal tissues to the toxic effects of potentially harmful chemicals can be attributed in part to the peculiarities in the anatomic and physiologic features of this organ. For instance, under resting state, the renal tissue receives about 20-25% of the resting cardiac output (Raina and Hamid, 2013). Therefore, any drug or chemical in the systemic circulation will be delivered to this organ in relatively high amounts; consequently, non-toxic concentrations of a chemical in the plasma or other tissues may reach toxic concentration in the kidney (Raina and Hamid, 2013).

CONCLUSION

It appears that in the absence of metabolic diseases and diseases of the elderly such as hypertension and diabetes mellitus, the frequency of AKI is higher in children and between the age group of 21 to 40.

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