

ABSTRACT

Serum electrolytes disorders in HIV patients in addition to resulting from disease induced fluids losses or accumulation could be attributed to a wide range of structural defects of cellular apparatus, tissue or organs of regulation. Most routine clinical investigation of impaired serum electrolytes in HIV infection limit attribution to body fluids charges and to primary organs of regulations. Such investigations do not address the likelihood of existence of multiple regulatory organs defects, the contribution of secondary regulatory organs and of non elemental electro chemical forces in establishing observed serum electrolytes states in HIV infection. This study investigated the association of electrolytes levels with kidney and liver functions in HIV infection in order to explore the extent of contribution of the renal and gastrointestinal primary regulatory organs to existing serum electrolytes disorders in light of the extended range of HIV impact on multiple organs of electrolytes regulation. This was a hospital based cross sectional study enrolling consecutive attendants of the PSC at Jaramogi Oginga Odinga Teaching and Referral Hospital. 800 HIV-infected and 406 seronegative controls were enrolled. Biochemical analysis was done of serum levels of major electrolytes (Na⁺, K⁺ and Cl⁻), markers of kidney function (creatinine and urea) and liver pathology (bilirubin, albumin, total protein and enzymes) and related body fluids parameters (osmolality and pressure). Frequency counts and measures of central tendency and dispersion around normal reference values were used to assess the distribution of analytes in the population. Associations of HIV status, CD4 count, ARV use, age and gender with electrolytes and fluid parameters were tested using, t-tests and Chi-square and regression logistics (r and r²) and significance levels assigned using $\alpha = 0.05$. Female gender, increasing age and CD4<200cells/mm³ emerged as determinants of occurrence of kidney disorders which were observed in 54% of the seropositive individuals. HIV infection conduced significant reduction in mean eGFR (88.1mls/min v/s 95.5mls/min, t=3.1, p=0.001). Therefore creatinine and urea imbalance were more prevalent in seropositive than healthy controls (26.1% vs11.8%; p<0.0001, 4.4% vs 0.5%, p<0.0001). liver function indicators; albumin, total proteins, bilirubin and enzymes (AST and ALT), were significantly impaired in seropositive than seronegative individuals; (32.8g/l vs 34.5g/l, t=5.3,p<0.0001); 64g/l vs 67.1g/l, t=6.7,p<0.0001); (6.2vs5, t=5.7,p<0.0001) AST;45.1U/l vs 36.9U/l, t=10.3,p<0.0001 and ALT;36.5U/l vs 30.7U/l, t=7.2,p<0.0001). Serum Na⁺, K⁺ and Cl⁻ ion imbalance were observed in 26.1%, 27.4% and 17.3% of HIV+ individuals respectively with only the prevalence of sodium imbalance being significantly more in HIV+ than HIV- individuals (26.1% vs 17.7%, $\chi^2 = 10.6$, p = 0.001). Using ARVs was accompanied with significant reduction in prevalence of Na⁺ ion imbalance (24.6%vs32.3%, $\chi^2 = 3.98$, p=0.046). Rates of electrolytes imbalance in HIV+ infection did not differ with or without kidney disorders and only 0.1% Na⁺, 0.01 % K⁺ and 0.3 % Cl⁻ imbalance were attributable to kidney defects. Lower prevalence of K⁺ imbalance (OR=0.6, $\chi^2 = 10.5$,p=0.001; OR=0.6, $\chi^2 = 6.7$,p=0.01) were associated with albumin and total protein depletion while higher rates of Na⁺ (55.5%vs25.4%, $\chi^2 = 8.3$,p=0.004) and K⁺(38.9% vs 16.8%, $\chi^2 = 6$,p=0.01) imbalance were associated with hyperbilirubinemia. Nonetheless co-variation between electrolytes imbalance and liver markers were minimal. Thus impaired liver and kidney functions did not sufficiently explain occurrence of the multiple electrolytes imbalance in HIV infection. Therefore diagnostic and management practices of electrolytes disorders in HIV-infected individuals need to expand to include comprehensive biochemical assessment of probable causes to reach as many as the likely elemental contributors as possible.