Depletion of Plasma Total Antioxidant Status in Marked in Diarrhoel Diseases of Acquired Immunodeficiency Syndrome

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Persistent diarrhoea in some cases, is a prominent feature of the acquired immunodeficiency syndrome (AIDS). But its cause and effect on the total antioxidant status (TAS) of patients with AIDS requires wider investigation and elucidation of plasma total antioxidant status as a factor in the pathogenesis of human immunodeficiency virus (HIV) infection. We studied 151 AIDS patients with diarrhoeal diseases and 120 others without diarrhoeal diseases fro levels of TAS by standard methods (Randox, UK). AIDS patients with diarrhoeal diseases had TAS level of 0.23±1.1 mmol/L and those without diarrhoea the levels were 0.37±0.19 mmol/L. the difference in the results of the two groups was statistically significant, $p<0.05$. Possibly depletion of plasma total antioxidant status is more expressed in diarrhoeal molecules may be beneficial to IDS patients with diarrhoeal diseases.

Keywords: AIDS, Diarrhoeal diseases, Total antioxidant status, Depletion.

Introduction

Repeated episodes of diarrhoea often lead to increasingly severe protein-energy malnutrition, specific micronutrient deficiency e.g., zinc, vitamin A, or a combination of the two. This is especially so when nutrient intake is limited[1]. Independent of human immunodeficiency virus

(HIV) infection, malnutrition impairs tissue repair, reduces immunologic function, and may inhibit host defenses, ultimately compromising the ability to resolve acute diarrhoea[2]. The impact of HIV infection in Sub-Saharan Africa is immense[3], with about 22.58 million (68 percent of world total) reported to be living in the sub-region. When excessive reactive oxygen species produced in patients infected with HIV is not appropriately compensated by antioxidant molecules, an oxidative stress may occur which could play an important role in the pathogenesis of HIV infection through various mechanisms[4, 5].

Diarrhoea is a frequently reported cause of death among some groups of patients with HIV infection[6]. It also causes great morbidity and mortality in African children with human immunodeficiency virus type-I (HIV-I) infection, accounting for up to 60% of the recorded illnesses in a country like Rwanda[7], and afflicting 80% of HIV-I infected infants in the second year of life in Zambia[8]. The risk factors for diarrhoea and its natural history in patients with HIV infection demands wider investigation. This report describes the results of plasma total antioxidant status in AIDS patients with diarrhoeal diseases and AIDS patients without diarrhoeal diseases in Maiduguri and environs, Nigeria.

Materials and Methods

Subjects

Adult patients (151) with gastrointestinal complaints and diarrhoeal diseases of AIDS, and 120 other AIDS patients without diarrhoeal diseases who attended the University of Maiduguri Teaching Hospital, Maiduguri Nigeria, and the state government hospitals at Auno and Mafa districts were studied. All medical care and assessments for clinical status were provided by that attending clinicians according to standard protocol[9, 10]. By this, acute diarrhoea was defined as a change in normal stool pattern, characterized by at least one day of increased frequency, at 14 days were considered to be persistent diarrhoea.

Methods

After counseling and informed consent in each case, blood was collected by venepuncture using a sterile hypodermic syringe from the ante-cubital vein into plain and heparinized containers. HIV status was determined by Genescreen ELISA (Biorad, France). HIV antibody positive individuals were confirmed using Immunocomb II HIV-I and 2 confirmed kits (Orgenics, Israel) on a fresh sample. Plasma total antioxidant (TAS)

levels were determined using a commercial kit (Randox, UK). The CD4+ T cell count was determined by a standard commercial method (Dynabeads, France).

The results were analysed using Epilinfo 6.04 statistical package. The data were summarized as means ±SD and compared using Mann Whitney U test for non-normally distributed data. Differences between the mean results for the groups was inferred at $p<0.05$.

Results

AIDS patients (151) with diarrhoeal diseases and AIDS patients without diarrhoeal diseases (120) were studied. The patients with diarrhoeal diseases had mean plasma total antioxidant status of 0.23±0.11 mmol/L and mean CD4+ count of ≤180±2.3. The patients without diarrhoeal diseases had mean plasma TAS of 0.37±0.19 and CD4+ count of ≥193±3.5 (Table 1). The difference in the mean TAS results between the two groups was statistically significant, U=412.500, p=0.000.

TABLE 1
Plasma Total Antioxidant Status (TAS) and CD_4+ Count Results for the AIDS Patients Studied in Maiduguri and Environs

Group	n	TAS Value (mmol/L)	CD4+ Count (/mm^3)
Diarrhoeal AIDS patients	151	0.23±0.11	180.0±2.3
Non-diarrhoeal AIDS patients	120	0.37±0.19	193.0±3.5

U=412.500, p=0.000

Discussion

This study provides evidence that plasma TAS is depleted in AIDS patients with diarrhoeal diseases compared to those without diarrhoea. This finding is consistent with a previous cross-sectional data from a cohort study showing that diarrhoea was associated with severe malnutrition and HIV-I infection[11]. There is palpable poverty and poor socio-economic conditions in the area of this present study. The basic staple diet among the peasant inhabitants of the local communities are

mainly carbohydrate rice, maize and rarely beans. Some of the increased risk of death among the infants with HIV-I infection may be attributable to the effects of malnutrition.

Antioxidants are compounds with chemical affinity for free radicals[12] existing in abundance and bond with free radicals before they can cause damage. Antioxidants are reported[12], to be of five classes, i.e., enzymes such as glutathione; phenolic compounds like vitamin E and plant flavonoids; nitrogen compounds which includes various amino acids, and carotenoids, mostly beta-carotone. Antioxidants are thus part of nutrition.

The AIDS patients used in the study had low CD_4+ counts. Whether this increased immunosuppresion was due entirely to the effects of HIV infection itself or was by part due to the malnutrition is not clear.

However, the cyclic effects of diarrhoea, malnutrition and immune dysfunction can produce an accelerated downward course in patient[13-15]. It is recommended that new management strategies for AIDS patients recognize the importance of plasma total antioxidant status.

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REFERENCES

1. Keusch, G.T., (1990). Malnutrition, infection and immune function, Nestle' Nutrition, In: Suskind, R.M. and Le Winter-Suskind, Eds., *The Malnourished Child,* Vol. 19 of the Nestle Nutrition Workshop Series, Vevey, Switzerland, pp. 37-55.
2. Keusch, G.T., Wilson, C.S. and Waksal, S.D., (1983). Nutrition, host defenses, and the lymphoid system, In: Gallin, J.I. and Fauci, A.S., Eds. *Advances in Host Defense Mechanism,* Vol. 2, Lymphoid Cells, Raven Press, New York, pp. 275-359.
3. Ofosu-Barko, K., (1998). The current global status of the HIV/AIDS Epidemic: What are the driving forces, First National AIDS Conference, Abuja, December 15-18.
4. Baruchel, S. and Wainberg, M.A., (1992). The role of oxidative stress in disease progression in individuals infected by the human immunodeficiency virus, *T. Leukoc. Biol.,* 52, pp. 111-114 (Abstract).
5. Israel, N. and Gougerot-Pocidalo, M.A., (1997). Oxidative stress in human immunodeficiency virus infection, *Cell. Mol. Life Sci.,* 53, pp. 864-870.

6. Nsanga, B., Ryder, R., Nsuami, M., Matela, B. and Nsa, W., (1989). High HIV-associated infant mortality correlates with poor post-partum maternal health: 18 months follow-up of 477 children born to HIV(+) mothers in Zaire, In: *Proceeding of the Fifth International Conference on AIDS,* Montreal, June 4-9, p. 324 (Abstract).
7. Malek, A.N.A., Mukelabai, K. and Luo, N.P., (1989). HIV disease in infants and children: 22-month prospective study, In: *Proceedings of the 5th International Conference on AIDS,* Montreal, June 4-9, p. 392 (Abstract).
8. Hira, S.K., Kamanga, J. and Bhat, G.J., (1989). Perinatal transmission of HIV-I in Zambia, *British Medical Journal (BMJ)*, 299, pp. 2853-2859.
9. World Health Organization, (1984). Programme for control of diarrhoea diseases: a manual for the treatment of acute diarrhoea for use by physicians and other senior health workers, Geneva.
10. Centers for Disease Control, (1992/93). Revised classification system for HIV infection and expanded surveillance case definitions of AIDS among adolescents and adults, *Morbid. Mortal Weekly Report,* 41, (RR-17), pp. 1-19.
11. Pavia, A.T., Long, E.G. and Ryder, R.W., (1992). Diarrhoea among African children born to HIV-I infected mothers: clinical, microbiologic and epidemiologic features, *Pediatr. Infect. Dis. J.,* 11, pp. 996-1003.
12. Diplock, A., (1991). Antioxidant nutrients and disease prevention: An overview, *Am. J. Clin. Nutr.,* 53, pp. 189s-193s.
13. Sepulveda, J., Willett, W. and Munoz, A., (1988). Malnutrition and diarrhoea: A longitudinal study among urban Mexican children, *Am. J. Epidemiol.,* 127, pp. 365-376.
14. Black, R.E., Brown, K.H. and Becker, S., (1984). Malnutrition is a determining factor in diarrhoeal duration, but not incidence, among young children in a longitudinal study in rural Bangladesh, *Am. J. Clin. Nutr.,* 39, pp. 87-94.
15. Bairagi, R., Chowdhury, M.K., Kim, Y.J., Curlin, G.T. and Gray, R.H., (1987). The association between malnutrition and diarrhoea in rural Bangladesh, *Intl. J. Epidemiol.,* 16, pp. 477-481.