ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 8, Issue, 11, pp.42328-42330, November, 2016

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

SERUM ZINC LEVEL IN CHILDREN WITH RELAPSING NEPHROTIC SYNDROME

***Qasim M H Almosawi**

Department of Pediatrics, College of Medicine, University of Kufa, AlZahraa teaching hospital, Najaf, Iraq

ARTICLE INFO

Article History:
Received 19th August, 2016
Received in revised form
09th September, 2016
Accepted 15th October, 2016
Published online 30th November, 2016

Key words:

Frequent relapses,
Nephrotic syndrome,
Zinc.

ABSTRACT

Background: Zinc as a second trace element of human body plays an essential role in numerous function. Abnormality in the metabolism of zinc in renal problem especially nephrotic syndrome is well documented. We aim in this research to measure the serum zinc level in patients with relapsing nephrotic syndrome.
Patient and method: A hospital based case control study that conducted at nephrology clinic at Al-Sadder and AL-Zahra teaching Hospitals for period between 1stJanuary 2013 to end of October 2013. A total of 60 pediatric patients with relapsing nephrotic syndrome were enrolled in this research. They were divided in two groups, (30) patients constituent of group A (patients with infrequent relapsing) and (30) patients constituent group B (patients with frequent relapsing).Control group consist of 32 healthy children. Serum zinc was measured by atomic absorption spectrophotometery.
Results: Patients aged 2-14 years, boys were 40 and girls were 20.The mean age of patients was 7.5 years. The Mean serum zinc level in group B(frequentrelapse) (58.45 µg/dl) that was significantly lower than that of group A (infrequent relapse) (61.58/dl) and control group (89.64µg/dl) respectively. P-value <0.001. There was no significant difference between patients of both groups and control in the mean of serum zinc level and sex of patients.
Conclusion*:* Hypozincemia can occur in chronic renal problem like nephrotic syndrome. The low level of serum zinc mainly found in those with frequent relapses Routine follow up of serum zinc level and other possible causes of hypozenicemia should be studied before giving zinc to the patients.

Citation: Qasim M Almosawi, 2016. "Serum zinc level in children with relapsing nephrotic syndrome", *International Journal of Current Research*, 8, (11), 42328-42330.

INTRODUCTION

Nephrotic syndrome was mainly a disease of children because it is 15 times morecommon in children than adults. The incidence was 2-3/100,000 children/year; and most of them are minimal changedisease. (Ciark and Barratt, 1999) The nephrotic syndrome was caused by renal diseases that increase thepermeability across the glomerular filtration barrier. It have the followingfeatures:

1. Proteinuria (Urinary protein excretion greater than 50mg/kg per day) or 40 mg/m^2 per hour.
2. Hypoalbuminemia(Serum albumin concentration less than 2.5 g/dL(25g/L))
3. Edema
4. Hyperlipidemia (Ciark and Barratt, 1999)

Conditions that increase the risk of infection in patient with nephrotic syndrome (McKinney *et al*., 2001)

1. Cold and infections. Both are very important cause of relapse in nephrotic syndrome. Children patients. Children are more liable for infection with various microorganism such as viruses, bacteria and protozoa; and all of these infection may cause relapse.
2. Poor compliance. It is very difficult to ensure that the child take his medicine at proper time and dose.

Hormones and immunosuppressantcan control the disease manifestation but cannot solve the disease pathology, that is why patients often become drug dependent and any missing or decreasing the dose may cause relapse. (VandeWalle *et al*., 1995) Infection is an important cause of morbidity andmortality in nephrotic children. Patients with steroid sensitive nephrotic syndrome (SSNS) have increased susceptibility to bacterial infections and various infections may result in relapses or steroid resistance or may trigger theonset of disease (Sleiman *et al*., 2007). Relapses in SSNS often follow infections of upperairway or gastrointestinal tract and cellulitis. It isestimated that 52–70% of relapses amongchildren in developing countries chiefly follow theupper respiratory tract infection (The primary nephrotic syndrome in children, 1981; White *et al*., 1970). Commoninfections associated with either onset of diseaseor in the course of disease are acute upper andlower respiratory infections (ARI) includingpneumonia with or without empyema, skininfections including impetigo and cellulitis, acutewatery or invasive diarrhea, urinary

Corresponding author: Qasim M H Almosawi,
Department of Pediatrics, College of Medicine, University of Kufa, AlZahraa teaching hospital, Najaf, Iraq.

tractinfections (UTI) and primary peritonitis (Barrat *et al*., 1994). Studies have shown that use of prophylacticantibiotics, immunoglobulins replacementtherapy, vaccination against streptococcip neumoniae, thymosin as immuno-modulatingagent, use of Chinese medicinal herb (TIAOJINING) and zinc supplements may have arole in prevention of these infections (Lonnerdal, 2000). However, in a recent Cochrane Database ofSystemic Review, it has been concluded thatthere is no strong evidence for any of aboveinterventions for prevention of infection innephrotic syndrome (Houser, 1984). Though pneumococcal peritonitis and cellulitisare decreased with use of pneumococcalvaccine and antibiotics but these infections arestill responsible for 1.4-10% of mortality andrepeated relapses in more than 80% of cases, requiring high dose steroids andhospitalization (The primary nephrotic syndrome in children, 1981). A high frequency of infections in children withnephrotic syndrome (38–83%) has been reportedfrom developing countries like India, Pakistan & Bangladesh in different studies (Houser *et al*., 1986). Studies fromdeveloping countries have also suggested thatincreasing the maintenance dose of steroid fromalternate day in a child with remission to dailyduring the episode of mild infections can preventrelapse (Houser *et al*., 1986; Barrat *et al*., 1994). Thus a strong suspicion regardinginfections in a nephrotic child is important notonly for treatment but also to prevent infectionassociated relapse. Zinc was an essential trace element. Protein increase zinc absorption so when there is deficiency in protein intake it may cause zinc deficiency. (Hambidge *et al*., 1986) The daily cause of zincwas 4, 14 mg; therecommended dietary allowance (RDA) was 8 mg/day for children ages 9 to 11years; (Stec *et al*., 1989). The essential sources of zinc include animal products such as meat, seafood, and milk.Ready-to-eat cereal contains the greatest amount of zincconsumed from plant products (Subar *et al*., 1998). About 10 - 40 % of dietary zinc was absorbed in the small intestine;absorption was decreased by the presence of phytates and fiber in the food that bindto zinc (Rahi *et al*., 2009). About 0.5 - 1.0 mg/day was secreted in the biliary system and discharged in the stool. Zinc circulates at a serum level of of 70 - 120 mcg/dL with 60 % bound to albumin and 30 % bound to macroglobulin. Urinaryexcretion about 0.5 - 0.8 mg/day. The main stores of zincare hepatic and renal. Most of the body zinc are stored inside the cell where zinc was bound to metalloproteins. (Subar *et al*., 1998) In nephrotic syndrome of steroid-sensitive the relapsesusually occur after upper respiratory tract infections and gastroenteritis. Some research suggest that zinc may decrease the risk of infections, we assess the role of such supplements in reducing relapse rates in nephrotic syndrome patients. (Tumer *et al*., 1989)

MATERIALS AND METHODS

This was a hospital based case control study on samples at nephrology clinic at Al-Sadderand AL-Zahra teaching Hospitals for period between 1st January 2013 to end of October 2013 as following.

Sample Size

A total of 60 pediatric patients age 2-14 years with relapsing nephrotic syndrome were included in this study, male were 40 and female were 20. They were separated into two groups: Group A(infrequent relapsing), 30 children, where there were 20 males and 10 females (relapse once time during 6 months since diagnosis of disease). Group B (frequent relapsing), 30 children, where there were 20 males and 10 females (relapse two or more during 6 months since diagnosis of disease). These patients were compared with 32 healthy children called"control group", where there were 17 males and 15 females. For both groups and control, blood samples were collected to measure the serum zinc concentration level by using Spectrophotometer. Zinc Measurement. This occur by spectrophotometry which measure color intensity that formed from reaction between zinc and chromogen. We draw a three ml of blood from each nephrotic patients and control. Then we centrifuge it at 3000 rpm for 10 minutes, the serum were kept at -70°C. Serum zinc was measured by atomic absorption device model Carl Zeiss Jena (Jena, Germany) Model AAS3 flame atomic absorption spectrometer. Serum zinc level less than 70µg/dl wasconsidered as hypozincemia. Data analysis was done by SPSS version 20 from IBM. P value less than 0.05 was considered as significant. This is blind study so the laboratory personnel did not know the patient and control sample. The study was approved by the local research and the ethics committee in the hospital and the college, parents consent was taken.

RESULTS

We made the statistical analyses to correlate the different conditions groups (Control, A, and B). These different conditions were divided according to the historical background of the patients (frequent and infrequent relapsing) in the mentioned hospitalsduring the lifetime of the disease (nephrotic syndrome). Table (1) shows the samples sizes for each group.

Table 1. The Samples Sizes for Each Group

Variables	Group Symbol	Sample Size		Total
		Male	Female	
Control	control	17	15	32
Infrequent Relapsing	A	20	10	30
Frequent Relapsing	B	20	10	30

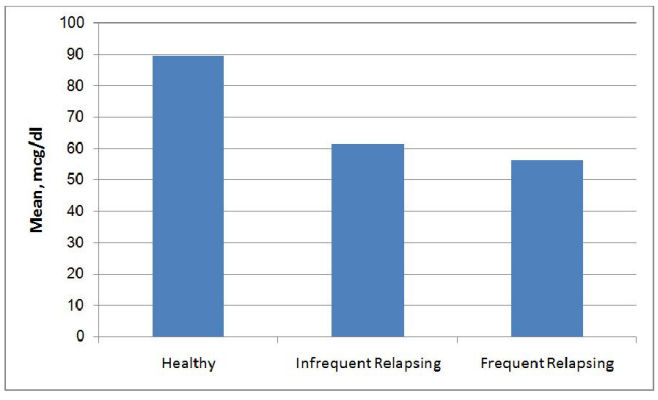

Figure 2. Mean serum zinc concentration for in all groups studied

Table 3. Mean Serum Zinc Concentration for Males and Females in all groups studied

Groups	Sex	Mean, mcg/dl	Standard Deviation	Standard Error	P-Value
Control	Male	92.159	17.25	4.18	*0.209
	Female	86.787	19.46	5.025	
A	Male	62.095	4.07	0.91	**0.214
	Female	60.55	5.26	1.66	
B	Male	58.45	6.60	1.47	***0.498
	Female	58.46	5.97	1.888	

Table 4. Statistical Analyses to Correlate the Different Groups

Variables	Sample Size	Mean	Standard Deviation	P-Value
Control	32	89.64	18.223	<0.001
A	30	61.58	4.469	
Control	32	89.64	18.223	<0.001
B	30	58.45	6.286	
A	30	61.58	4.469	0.028426
B	30	58.45	6.286	

DISCUSSION

In this study The Mean serum zinc level in group B (frequent relapse) (58.45 µg/dl) that was significantly lower than that of group A (infrequent relapse) (61.58/dl) and control group (89.64 µg/dl) respectively. P-value <0.001 these finding can be explained by Mild zinc deficiency is believed to result in a reduced production of Th1 cytokines, resulting in Th2 cytokine bias (Shankar *et al*., 1998; Prasad *et al*., 1988). In contrast, zinc intake is thought to increase gene expression for IL-2 and IFN-γ, so it result in augmentation of the Th1 immune response [(15)]. Since the Th1–Th2 cytokine imbalance is also believed to result in relapses of SSNS. So this results support the findings that suggest the patients with SSNS who take RDA of zinc show a less relapses and higher possibility of remission. The response was better in the frequent relapsers (Bovio *et al*., 2007; Reimold, 1980). The Mean serum zinc level of group A (infrequent relapse) is also low (61.58/dl), this finding can explained by The increase urinary zinc excretion in children with NS (whether in relapse or in remission) was attributed as a cause for low serum zinc level by many authors who reported a positive correlation between urinary zinc and protein excretion in their studies (Tumer *et al*., 1989; Mumtaz *et al*., 2011; Perrone *et al*., 1990; Stec *et al*., 1989). Also Several studies demonstrated low blood or serum zinc levels among children with NS compared to that of the control groups (Bovio *et al*., 2007; Tumer *et al*., 1989). This study certifies insignificant difference corresponding to sex in all groups of patients studied. This result was also stated by Rahi *et al*. 2009, with M:F ratio of 1.8:1. This result moreover was stated by Sarker *et al*., 2012, with M:F ratio of 2:1. (Sarker *et al*., 2012)

Conclusion

Hypozincemia can occur in chronic renal problem like nephrotic syndrome. The low level of serum zinc mainly found in those with frequent relapses there is no effect of sex on mean serum level in all group studied

REFERENCES

Bao B, Prasad AS, Beck FW, Godmere M 2003. Zinc modulates mRNA levels of cytokines. *Am J Physiol Endocrinol Metab.,* 285:1095–1102

Barrat, T.M., J.M Beattic, JS Dossetor Janet Colmen, 1994. Consensusstatement on management and potential for steroid responsive nephroticsyndrome. *Archif. Dis. In Childhood*, 70:151-157.

Bovio G, Piazza V, Ronchi A, *et al*. 2007. Trace element levelsin adult patients with proteinuria. *Minerva Gastroenterol Dietol*., Dec;53(4):329-36.

Ciark AG. and Barratt TM. 1999. Steroid responsive nephrotic syndrome in pediatric nephrology Barratt TM, MB. FRCS, Avner ED,MD Harmon WE, MD, Awoltersklumer campany 4th ed, 731.147

Hambidge, KM, Casey, CE, Krebs, NF. 1986. Zinc in trace elements. In:Human and animal nutrition, vol 2, 5th edition, Mertz, W (Ed), AcademicPress, Orlando., p.1.

Houser, M. 1984. Assessment of proteinuria using random urine samples. *J Pediatr*, 104:845.

Houser, MT, Jahn, MF, Kobayashi, A, Walburn, J. 1986. Assessment ofurinary protein excretion in the adolescent: effect of body position andexercise. *J Pediatr*., 109:556.

Lonnerdal, B. 2000. Dietary factors influencing zinc absorption. *J Nutr*., 130:1378S.

McKinney, PA, Feltbower, RG, Brocklebank, JT, Fitzpatrick, MM. 2001. Time trendsand ethnic patterns of childhood nephrotic syndrome in Yorkshire, UK. Pediatnephrol, 16:1040

Mumtaz A, Anees M, Fatima S, *et al*. 2011. Serum zinc andcopper levels in nephrotic syndrome patients. *Pak J MedSci*., October-December;27(5):1173-6.

Perrone L, Gialanella G, Giordano V, *et al*. 19903 Impairedzinc metabolic status in children affected by idiopathicnephrotic syndrome. *Eur J Pediatrics*, 149:438-40.

Prasad, AS, Meftah, S, Abdallah, J. 1988. Serum thymulin in human zinc deficiency. *J Clin Invest*, 82:1202.

Rahi, Kasim, Al-Badri, Abdel Salam, Salih, BushraJalil, and Hasan, FaleehaObaid, 2009. "Childhood Nephrotic Syndrome, Frequent and Infrequent Relapses and Risk Factors for Relapses", *The Iraqi Pstgraduate Medical Journal*, Vol. 8, No. 3, Baghdad, Iraq.

Reimold, E.W. 1980. Changes in zinc metabolism during thecourse of the nephrotic syndrome. *Am J Dis Child.,* 134:46–50

Sarker. MN., MMSU Islam, T Saad, FN Shoma, LS Sharmin, HA Khan, F Afrooz, LE Fatmi, A Alam, ASM Salimullah, MR Uddin, T. *SahaFaridpur Med. Coll. J*., 2012;7(1): 18-22.

Shankar, AH, Prasad, AS. 1998. Zinc and immune function: The biological basis of altered resistance to infection. *Am J ClinNutr*., 68:447S.

Sleiman, JN, D'Angelo, A, Hammerschlag, MR. 2007. Spontaneous Escherichia coli cellulitis in a children with nephrotic syndrome. *Pediat infect Dis J*., 26:266.

Stec J, Podracká L, Pavkovceková O. 1989. Renal excretion ofzinc in children with kidney diseases. *Cesk Pediatr*, Dec;44(12):705-7.

Subar, AF, Krebs-Smith, SM, Cook, A, Kahle, LL. 1998. Dietary sources of nutrients among US children, 1989-1991. *Pediatrics*., 102:913.

The primary nephrotic syndrome in children. Identification of patients withminimal change nephrotic syndrome from initial response to prednisone. Areport of the International Study of Kidney Disease in Children. *J Pediatr*., 98:561.

Tumer N, Baskan S, Arcasoy A, Cavdar AO, Ekim M 1989. Zincmetabolism in nephrotic syndrome. *Nephron,* 52:95.

VandeWalle, JG, Donckerwolcke, RA, van Isselt, JW, *et al*. 1995. Volumeregulation in children with early relapse of minimal-change nephrosis with or without hypovolaemic symptoms. *Lancet*, 346:148.

White, RH, Glasgow, EF, Mills, RJ.1970. Clinicopathological study of nephroticsyndrome in childhood. *Lancet*, 1:1353.

****