Pearl Research Journals

Journal of Medical and Biological Science Research
Vol. 3 (3), pp. 19-23, July, 2017
ISSN: 2449-1810
Full Length Research Paper
http://pearlresearchjournals.org/journals/jmbsr/index.html

Measurement of serum Zinc and Magnesium in Sudanese Patients with Metabolic Syndrome

Aseil Abdalla Khalid Yousif [1], Abdelrahman Ahmed Mohammed Elamin Zayed[2], Amna O.M.Elzein[2] and Nassr Eldin M.A. Shrif [2]*

Accepted 19 June 2017

[1]Department of Clinical Chemistry, Faculty of Medical Laboratory Sciences, Alneelain University, Khartoum, Sudan.
[2]Department of Clinical Chemistry, Faculty of Medical Laboratory Sciences, Alzaeim Alazhary University, Sudan.

ABSTRACT

Metabolic syndrome may have a potential association with the functions of zinc and magnesium. The study aim to find the association between serum zinc, magnesium levels and metabolic syndrome. This Cross sectional study was conducted during the period of September 2016 to February 2017 in Diabetes and endocrine glands hospitals in Khartoum state, Sudan. Hundred individuals was enrolled in the study classified into two groups, 50 diabetic patients as case group and 50 healthy individuals as control group, Blood sample was taken and serum was used for estimation of zinc and magnesium by using atomic absorption spectroscopy. All data analyzed by using the statistical package for Social Sciences (SPSS). Means of serum zinc and magnesium levels in patient with metabolic syndrome were 0.20±0.13 mg/L and 14.9±5.2 mg/L, respectively, Means of serum zinc and magnesium levels in patient without metabolic syndrome were 0.67±0.34 mg/L and 18.9±2.5 mg/L, respectively, this study revealed that there were significant differences in serum zinc levels, magnesium levels, triglyceride, BMI, HDL cholesterol and systolic blood pressure between case and control ($p<0.05$), but insignificant differences in diastolic blood pressure ($p=0.228$).These findings suggest that serum zinc and magnesium levels might be associated with metabolic syndrome or metabolic risk factors.

Key words: Metabolic Syndrome, Zinc, Magnesium and Sudanese.

*Corresponding author. E-mail: aboamr124@hotmail.com.

INTRODUCTION

The cluster of metabolic abnormalities that predisposed an individual to develop Type 2 Diabetes (T2D) and Cardiovascular Disease (CVD) has been called Metabolic Syndrome (MetS) Morimoto-(Yamashita et al., 2012). The World Health Organization announced the first formal definition of MetS, including the following clinical parameters: Impaired Glucose Tolerance (IGT), changes in Impaired Fasting Glucose (IFG) or diabetes mellitus and/or resistance to insulin, plus two more additional components: hypertension (blood pressure ≥160/90 mm Hg), high triglycerides in plasma (≥150 mg/dl) and/or low levels of HDL-cholesterol (HDL-c 30 mg/dL or waist circumference/hips index >0.90 for men and >0.85 for women) and micro albuminuria (Anjani et al., 2013; Rajhans et al., 2011). Trace elements form part of daily diet, which are well known to play vitally important roles in the maintenance of health. Moreover, composition of daily diet has been changed considerably, which causes great increased incidence of many different diseases such as diabetes mellitus (DM) (Thomas et al., 2010). Zinc is known to be an essential trace element and a component of hundreds of enzymes (Jayawardena et al., 2012), and is involved in the synthesis, storage, and release of insulin. Many studies have documented that plasma zinc levels are lower in obese individuals (Jansen et al., 2009). Furthermore, zinc deficiency may predispose glucose intolerance and insulin resistance, diabetes mellitus, and coronary artery disease (Kiilerich et al., 1990).

The role of Zn in the development of diabetes is controversial (Viktorínová et al., 2009), but there is evidence that increases insulin secretion (Fawcett et al., 1999; Jahnen-Dechent and Ketteler,2012). Zn interacts with insulin to form hexamers and guarantees its biological function. This oligo element plays an important role in the regulation of glucose in blood through insulin (Sales and Pedrosa, 2006). Many studies showed that higher zinc

Table 1. The comparison of mean (SD) of zinc and magnesium between case and control.

Status	Case	Control	P-value
	Mean(SD)		
Magnesium (mg/L)	14.9(5.2)	18.9(2.5)	0.006
Zinc (mg/L)	0.20(0.13)	0.67(0.34)	0.003
Triglyceride (mg/dL)	258(64)	145(27)	0.000
HDL cholesterol (mg/dL)	70(20)	65(13)	0.014
Dystonic Blood pressure (mmHg)	82(6.0)	80(5.0)	0.228
Systolic blood pressure (mmHg)	127(13)	122(4.9)	0.000
Body Mass Index (kg/m^2)	27.9(4.2)	21.4(2.5)	0.003

Independent t test was used to calculate p value.

intakes are associated with a lower risk of type 2 diabetes in U.S. women (Jahnen-Dechent and Ketteler,2012) and other studies have shown that zinc supplementation increases high density lipoprotein (HDL) cholesterol and reduces triglyceride (TG) in type 2 diabetes patients (Jansen et al., 2009; Kiilerich et al., 1990). Furthermore, supplemental zinc (20 mg per day) significantly decreased insulin resistance in obese children with metabolic syndrome (Ebel and Gunther, 1980; He et al., 2006). However, relationships between zinc status and insulin resistance/metabolic risk factors are more controversial among non-diabetic obese and non-obese subject (He et al., 2006). A strong association between Mg, T2D and hypertension has been described Deficiency of Mg exacerbates insulin resistance and T2D, and predisposes the subjects to CVD (Javed et al., 2013). On the other hand, it has been shown that oral supplementation with Mg improves T2D control. Hence, these data support the idea that Mg supplements may be particularly beneficial in diabetic and hypertensive subjects (Al-Daghri et al., 2013). However, in a project of the Atherosclerosis Risk in Communities Study (ARIC) it was found that low dietetic intake of Mg does not confer risk for T2D in middle-aged population (Chambers et al., 2011). There was, nonetheless, a clear inverse correlation between Mg concentrations in serum and the T2D incidence in white population, but not in Afro-American population (Rajhans et al., 2011; Dubinsky et al., 2006). Epidemiologic studies have shown a high prevalence of hypomagnesemia in type 2 diabetic subjects with poor glycemic control so increased Mg intake may be associated with reduced risk of developing the metabolic syndrome (Rajhans et al., 2011;).

MATERIALS AND METHODS

This is a cross-sectional study conducted in diabetic centers in Khartoum State, Sudan during the period of September 2016 to February 2017. After writing an informed consent blood samples were collected in heparinized containers from 50 diabetic patients as case group and 50 healthy individuals as control group and then samples were centrifuged at 3000 rpm for 3 min to obtain plasma. Hemolyzed samples were excluded and 3.0 ml plasma samples were preserved at -20°C until analysis for estimation of zinc and magnesium by using atomic absorption spectroscopy. Patients with other chronic medical disorders were excluded (renal failure, liver failure, thyroid disorder). All data analyzed by using the statistical package for Social Sciences (SPSS) two independent t –test and Bivariate Pearson`s correlation considering confidence Interval of 95%, so P value < 0.05 considered significant.

RESULTS AND DISCUSSION

The present study was conducted using a total of hundred participants. Means (SD) of serum zinc and magnesium levels in patient with metabolic syndrome are 0.20±0.13 mg/L and14.9±5.2 mg/L respectively. Means (SD) of serum zinc and magnesium levels in patient without metabolic syndrome are 0.67±0.34 mg/L and 18.9±2.5 mg/L respectively (Table 1). In this study we observed a significant differences in serum zinc levels, magnesium levels, triglyceride, BMI, HDL cholesterol and systolic blood pressure between case and control with p value 0.003, 0.006, 0.000, 0.003, 0.014 and 0.000 respectively (Figure 1) but no significant difference in diastolic blood pressure was observed (p = 0.228). In Table 2 significant correlations were observed between serum zinc levels, Triglyceride (r = - 0.521, p =0.000) and DBP (r = - 0.505, p = 0.000); but insignificant correlations with HDL cholesterol (r = - 0.126, p = 0.220), Systolic BP (r = - 0.175, p = 0.088) and BMI (r = - 0.179, p = 0.081). Significant correlations were also observed between serum magnesium levels, Triglyceride (r = - 0.333, p = 0.001), and DBS (r = - 0.346, p = 0.000); but insignificant correlations with HDL cholesterol (r = - 0.057, p = 0.570), Systolic BP (r = 0.113, p = 0.263) and BMI (r = - 0.029, p = 0.777) (Figure 2).

This study investigated the levels of serum zinc and magnesium among patient with Met S. This study revealed significant differences in serum zinc levels between case and control (p = 0.003). Other factors not included in the clinical definition of Met S, such as chronic inflammation or oxidative stress, may lead to the development of Met S. On the other hand, oxidative stress, which occurs when reactive oxygen species (ROS) exceed the antioxidant capacity, may play an important role in Met S. Zinc reduces inflammatory cytokine production via up-regulation of a zinc-finger protein, which inhibits nuclear factor kB (NF-kB)

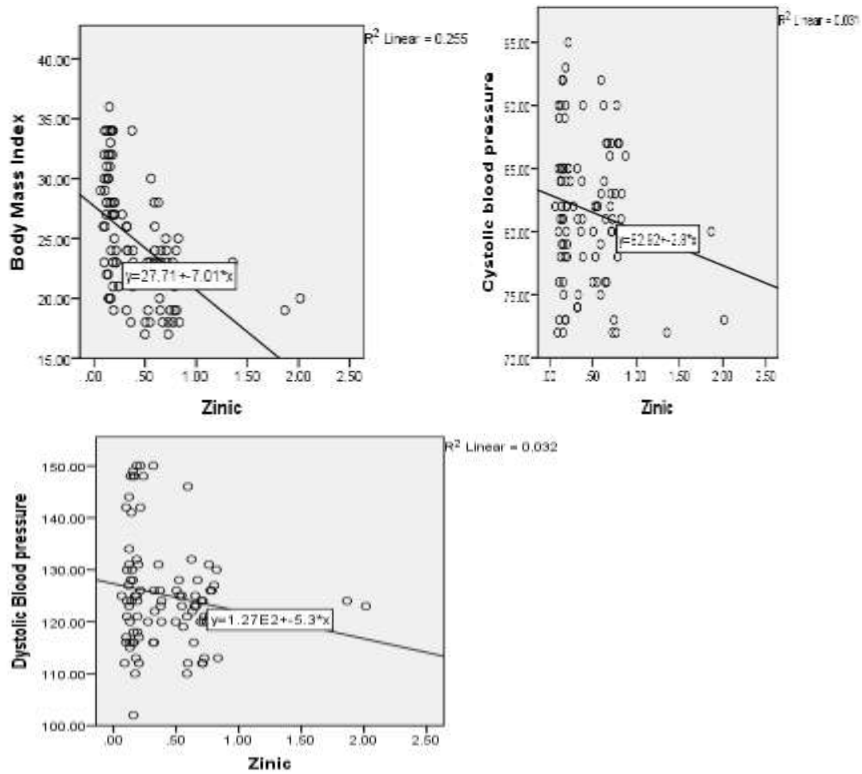

Figure 1. Correlation between zinc and the study parameters.

Table 2. Correlation between zinc, magnesium with studied parameters.

Parameters	Zinc		Magnesium	
	R-value	p-value	R-value	p-value
Triglyceride	- 0.521	0.000	- 0.338	0.001
HDL cholesterol	- 0.126	0.220	- 0.057	0.570
Systolic blood pressure	- 0.505	0.000	- 0.346	0.000
Dystonic Blood pressure	- 0.175	0.088	- 0.113	0.263
Body Mass Index	- 0.179	0.081	- 0.029	0.777

activation. Furthermore, zinc a cofactor for antioxidant enzymes, such as superoxide dismutase and glutathione peroxidase, decreases ROS generation and induces metallothionein, which decreases the OH burden, suggesting that a decrease in body zinc status may contribute to the development or aggravation of Met S. In addition, chronic inflammation or oxidative stress may contribute to the decreased serum zinc levels. This study agrees with study done by Al- Daghri et al., 2013; Seo et al., 2010, who reported significant different in zinc between case and control, however it disagree with study done by Rotter et al. (2015).This study revealed there was significant differences in serum magnesium levels, between case and control p value (p= 0.006); a possible explanation for the low concentration of serum Mg in Met S in subjects with diabetes could be the increased renal Mg excretion which was caused by insulin deficiency or resistance. This study also agree with study done by Saari et al. (2005), Rotter et al. (2015) and Al-Daghri et al. (2013).

The study also show that there was a significant difference in triglyceride levels, between case and control; which agrees with study done by Bonnet et al. (2013) but disagree with study done by Seo et al. (2010). There was significant differences in HDL cholesterol levels, between case and control (p= 0.001); which agrees with study done by Bonnet et al. (2013) but disagree with study of Seo et al. (2010). There was insignificant differences in DBP levels, between case and control (p = 0.228); which agrees with the study done by Seo et al. (2010) but disagree with study done by Bonnet et al. (2013).

There was observed significant differences in SBP levels, between case and control (p = 0.000); which disagree with study done by Zehra et al. (2016). There was significant differences in BMI levels, between case and control (p = 0.003); and agrees with study done by Van der Poorten et al. (2008), but disagree with study done by Seo et al. (2010).

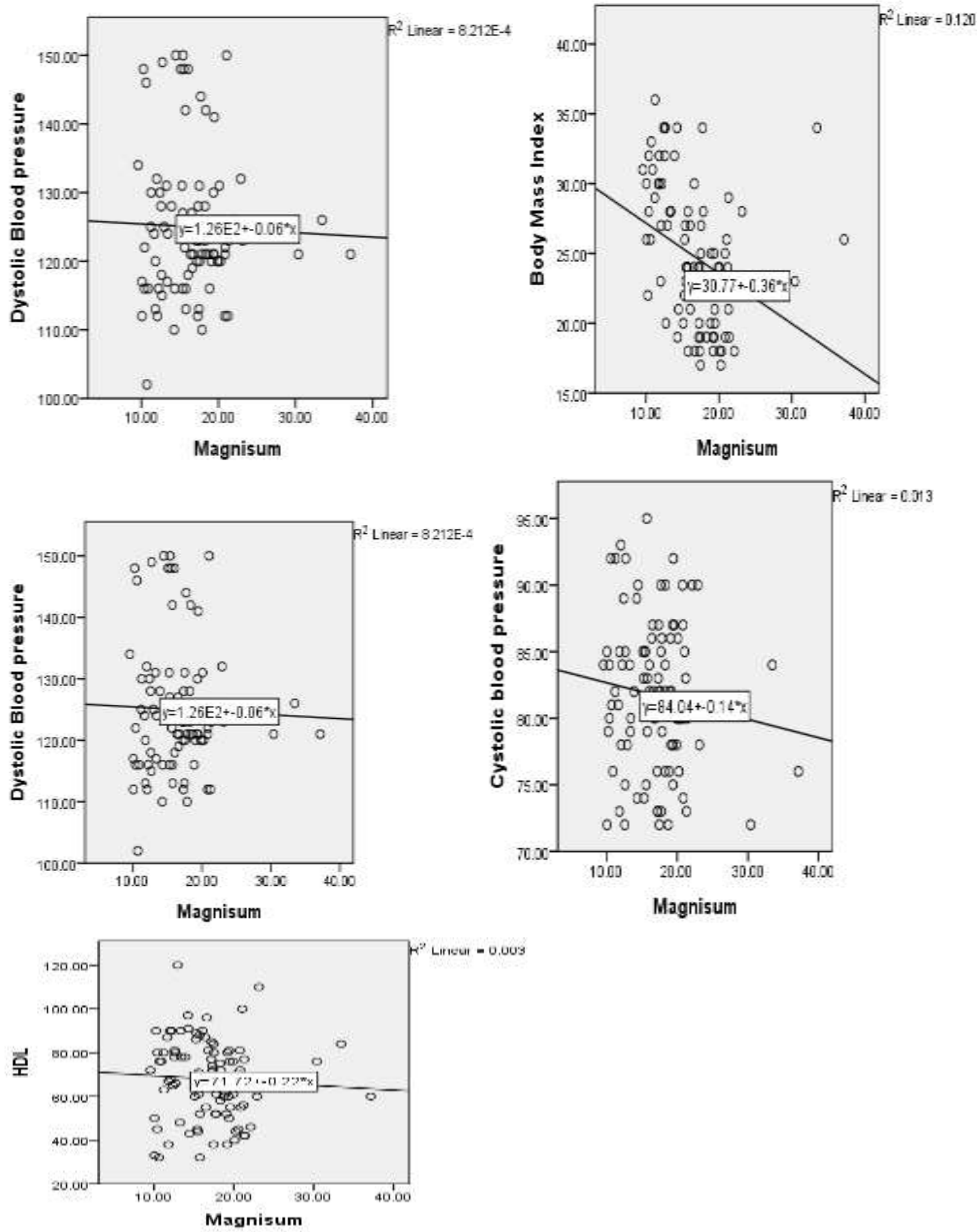

Figure 2. Correlation between Magnesium and the study parameters.

CONCLUSION

The study concludes that low serum magnesium and zinc levels may associates with metabolic syndrome in Sudanese populations. Further studies is needed to evaluate the effect of dietary intake of zinc and magnesium on metabolic syndrome in Sudanese.

REFERENCES

Al-Daghri NM, Alkharfy KM, Al-Saleh Y, Al-Attas OS, Alokail MS, Al-Othman A, Moharram O, El-Kholie E, Sabico S, Kumar S, Chrousos GP (2012). Modest reversal of metabolic syndrome manifestations with vitamin D status correction: a 12-month prospective study. Metabolism, 61(5): 661-666.

Al-Daghri NM, Nasiruddin K, Khalid MA, Omar SA, Majed SA,Hanan AA, Abdulaziz A, Paul MV (2013). Selected dietary nutrients and the prevalence of metabolic syndrome in adult males and females in Saudi Arabia: a pilot study. Nutrients, 5(11): 4587-4604.

Anjani KP, Vijay KS, Anil C, Kopal G (2013). Association between diabetes mellitus and periodontal status in north Indian adults. Eur J. General Dent. 2(1): 58-61.

Bonnet F, Fritz-Line VC, Alain G, Séverine D, Catherine M, Alioune C, Laurent L, Beverley B, Pierre-Henri D (2013). Role of sex steroids, intrahepatic fat and liver enzymes in the association between SHBG and metabolic features. Clin. Endocrinol. 79(4): 517-522.

Chambers JC, Zhang W, Sehmi J, Li X, Wass MN, Van der Harst P, Holm H, Sanna S, Kavousi M, Baumeister SE, Coin LJ, Deng G, Gieger C, Heard-Costa NL, Hottenga JJ, Kühnel B, Kumar V, Lagou V, Liang L, Luan J, Vidal PM, Mateo Leach I, O'Reilly PF, Peden JF, Rahmioglu

N, Soininen P, Speliotes EK, Yuan X, Thorleifsson G, Alizadeh BZ, Atwood LD, Borecki IB, Brown MJ, Charoen P, Cucca F, Das D, de Geus EJ, Dixon AL, Döring A, Ehret G, Eyjolfsson GI, Farrall M, Forouhi NG, Friedrich N, Goessling W, Gudbjartsson DF, Harris TB, Hartikainen AL, Heath S, Hirschfield GM, Hofman A, Homuth G, Hyppönen E, Janssen HL, Johnson T, Kangas AJ, Kema IP, Kühn JP, Lai S, Lathrop M, Lerch MM, Li Y, Liang TJ, Lin JP, Loos RJ, Martin NG, Moffatt MF, Montgomery GW, Munroe PB, Musunuru K, Nakamura Y, O'Donnell CJ, Olafsson I, Penninx BW, Pouta A, Prins BP, Prokopenko I, Puls R, Ruokonen A, Savolainen MJ, Schlessinger D, Schouten JN, Seedorf U, Sen-Chowdhry S, Siminovitch KA, Smit JH, Spector TD, Tan W, Teslovich TM, Tukiainen T, Uitterlinden AG, Van der Klauw MM, Vasan RS, Wallace C, Wallaschofski H, Wichmann HE, Willemsen G, Würtz P, Xu C, Yerges-Armstrong LM (2011). Genome-wide association study identifies loci influencing concentrations of liver enzymes in plasma. Nat. Genet. 43(11): 1131-1138.

Dubinsky MC, Lin YC, Dutridge D, Picornell Y, Landers CJ, Farrior S, Wrobel I, Quiros A, Vasiliauskas EA, Grill B, Israel D, Bahar R, Christie D, Wahbeh G, Silber G, Dallazadeh S, Shah P, Thomas D, Kelts D, Hershberg RM, Elson CO, Targan SR, Taylor KD, Rotter JI, Yang H, Western Regional Pediatric IBD Research Alliance (2006). Serum immune responses predict rapid disease progression among children with Crohn's disease: immune responses predict disease progression. Am. J. Gastroenterol. 101(2): 360-367.

Ebel H, Gunther T (1980). Biochemical functions of magnesium. J. Clin. Chem. Biochem.18: 256-69.

Fawcett WJ, Haxby EJ, Male DA (1999). Magnesium: physiology and pharmacology. Br. J. Anaest. 83(2): 302-320.

Ha Y, Seo N, Shim JH, Kim SY, Park JA, Han S, Kim KW, Yu E, Kim KM, Lim YS, Lee HC, Chung YH, Lee YS (2015). Intimate association of visceral obesity with non-alcoholic fatty liver disease in healthy Asians: A case-control study. J. Gastroenterol. Hepatol. 30(11): 1666-1672.

He K, Liu K, Daviglus ML, Morris SJ, Loria CM, Van Horn L, Jacobs DR Jr, Savage PJ (2006). Magnesium intake and incidence of metabolic syndrome among young adults. Circulation, 113(13): 1675-1682.

Jahnen-Dechent W, Ketteler M (2012). Magnesium basics. Clin. Kidney J. 5(Suppl 1):i3-i14.

Jansen J, Karges W, Rink L (2009). Zinc and diabetes clinical links and molecular mechanisms. J. Nutr. Biochem. 20(6): 399-417.

Javed F, Tenenbaum HC, Nogueira-Filho G, Nooh N, O'Bello Correa F, Warnakulasuriya S, Dasanayake AP, Al-Hezaimi K (2013). Periodontal inflammatory conditions among gutka chewers and non-chewers with and without prediabetes. J. Periodontol. 84(8):1158-1164.

Jayawardena R, Ranasinghe P, Galappatthy P, Malkanthi R, Constantine G, Katulanda P (2012). Effects of zinc supplementation on diabetes mellitus: a systematic review and meta-analysis. Diabetol. Metab. Syndr. 4(1):13.

Kiilerich S, KeldHvid-Jacobsen, Allan V, Sven SS (1990). 65 zinc absorption in patients with insulin-dependent diabetes mellitus assessed by whole-body counting technique. Clinica Chimica Acta, 189(1): 13-18.

Morimoto-Yamashita Y, Ito T, Kawahara K, Kikuchi K, Tatsuyama-Nagayama S, Kawakami-Morizono Y, Fijisawa M, Miyashita K, Emoto M, Torii M, Tokuda M (2012). Periodontal disease and type 2 diabetes mellitus: Is the HMGB1 RAGE axis the missing link? Medical hypotheses. 79(4): 452-455.

Rajhans NS, Ramesh MK, Viren GC, Nilkanth HM (2011). A clinical study of the relationship between diabetes mellitus and periodontal disease. J. Indian Soc. Periodontol. 15(4): 388-392.

Rotter I, Kosik-Bogacka D, Dołęgowska B, Safranow K, Lubkowska A, Laszczyńska M (2015). Relationship between the concentrations of heavy metals and bioelements in aging men with metabolic syndrome. Int. J. Environ. Res. Public Health, 12(4): 3944-3961.

Saari KM, Lindeman SM, Viilo KM, Isohanni MK, Järvelin MR, Laurén LH, Savolainen MJ, Koponen HJ (2005). A 4-fold risk of metabolic syndrome in patients with schizophrenia: the Northern Finland 1966 Birth Cohort study. J. Clin. Psychiatry. 66(5): 559-563.

Sales CH, Pedrosa Lde (2006). Magnesium and diabetes mellitus: their relation. Clin. Nutr. 25(4): 554-562.

Seo JA, Song SW, Han K, Lee KJ, Kim HN (2014). The associations between serum zinc levels and metabolic syndrome in the Korean population: findings from the 2010 Korean National Health and Nutrition Examination Survey. PloS one. 9(8): e105990.

Thomas B, Kumari S, Ramitha K, Ashwini KMB (2010). Comparative evaluation of micronutrient status in the serum of diabetes mellitus patients and healthy individuals with periodontitis. J. Indian Soc. Periodontol. 14(1): 46-9.

Van der Poorten D, Milner KL, Hui J, Hodge A, Trenell MI, Kench JG, London R, Peduto T, Chisholm DJ, George J (2008). Visceral fat: a key mediator of steatohepatitis in metabolic liver disease. Hepatology, 48(2): 449-457.

Viktorínová A, Toserová E, Krizko M, Duracková Z (2009). Altered metabolism of copper, zinc, and magnesium is associated with increased levels of glycated hemoglobin in patients with diabetes mellitus. Metabolism 58(10): 1477-1482.

Zehra S, Mozaffer RH, Erum I, Masood AQ (2016). Circulating adipokine levels and insulin resistance in type 2 diabetes mellitus and metabolic syndrome. Endocrine Abstracts,41 EP452.