

International Journal of Advanced Biotechnology and Research (IJBR)
ISSN 0976-2612, Online ISSN 2278–599X,
Vol-8, Issue-4, 2017, pp499-505
http://www.bipublication.com

Research Article

Serum Calcium and Magnesium Level in Alcoholic Population: The Clinical Experience at Tertiary Care Hospital

Muhammad Iqbal¹, Suhail Ahmed Almani¹, Atif Ahmed², Hamid Nawaz Ali Memon³, Syed Jahanghir⁴, Abdul Subhan Talpur⁵, Zubair Suhail Almani⁵ and Zulfiqar Ali Qutrio Baloch⁶

¹Department of Medicine, Liaquat University of Medical and Health Sciences (LUMHS) Jamshoro

²Bilawal Medical College (LUMHS), Jamshoro

³Zulekha Hospital Dubai United Arab Emirates

⁴Liaquat University of Medical and Health Sciences, Jamshoro

⁵Liaquat University Hospital Hyderabad / Jamshoro

⁶Brandon Regional Hospital Brandon, Florida, U.S.A

<u>CORRESPONDENCE:</u> *Dr. Muhammad Iqbal, Department of Medicine (unit-II), Liaquat University of Medical and Health Sciences (LUMHS) Jamshoro

Email: zulfikar229@hotmail.com & muhammad.iqbal@lumhs.edu.pk Cell # 92-3003057155 and 92-3003034963

ABSTRACT

OBJECTIVE: To evaluate the serum calcium and magnesium level in alcoholic population.

PATIENTS AND METHODS: This one year cross sectional study was conducted at tertiary care hospital and the inclusion criteria of the study were the patient ≥ 20 years of age, either gender visited in ward or general outpatient department had history of alcohol intake for ≥ 3 years duration were recruited and enrolled in the study. All the specific patients were evaluate for hypocalcemia and hypomagnesemia by taking 2 CC venous blood sample and sent to laboratory for analysis. The frequency and percentages was calculated while the numerical statistics were used to compute mean $\pm SD$.

RESULTS: During one year study period total fifty patients with alcohol intake were recruited, enrolled and evaluate for the glycemic status. The mean \pm SD of age, serum calcium and magnesium for whole population was 39.93 ± 6.75 years, 6.53 ± 2.21 and 1.42 ± 2.72 . Out of fifty individuals 45 (90%) were males and 05 (10%) were females while majority of the population belonged to urban areas 32 (64%). The hypocalcemia and hypomagnesemia was detected in 37 (74%) and 38 (76%) patients with statistical significance.

CONCLUSION: In alcoholic population, the raised BMI and GGT and contradictory decrease in serum calcium (hypocalcemia) and magnesium level (hypomagnesemia). Thus, the estimation of serum calcium and serum magnesium along with GGT and BMI may be useful indicator for clinical prognosis in alcoholic population.

KEYWORDS: Alcohol, Calcium and Magnesium and alcohol dependence syndrome.

INTRODUCTION:

Alcoholism is the major dependences for people and responsible for morbidity and mortality and its intake causes metabolic alterations & impairs homeostasis of micro & macro elements in body [1, 2]. The alcohol dependence syndrome [ADS] is a group of physiological behavioral & cognitive

phenomenon in which the high priority is given to alcohol intake than the other behaviors had great value [3]. The reported prevalence of ADS is 21% and 8.5% for males and females total population while the rates reported to be higher in psychiatrically ill individuals [4]. Bengal V, et al, [5] and Das SK, et al [6] reported alcohol dependence syndrome to be approximately 30-35% & 6.6% in males and females population while the other studies had shown to be around 15-50% [6-7]. ADS is the reversible causes for psychiatry & medical morbidity with the well known already mentioned harmful effects in the publish literature [8]. ADS is a heterogeneous disorder and observed the various drinking patterns in different cultures and also considered as polygenic, multifactorial disorder [9]. The measures have been taken regarding the pattern of alcohol drinking, family burden, personality profiles and psychopathology, socialism and vulnerability markers [10]. In India the substance dependence for alcohol and opiates shown to be 22%, opium 0.5%, cannabis 3.2% and heroin 0.3% [5]. Chronic alcoholism is the most psychoactive substance and is a major public health issue and leads to toxicity and various complications and also associated with several degenerative & inflammatory processes in the nervous system [11]. The disturbance in serum calcium & magnesium i.e. decreases (hypocalcaemia & hypomagnesemia) has an inverse association to alcohol concentration in alcoholic individuals and responsible for clinical conditions like musculoskeletal disorders and malnutrition [12, 13]. No previous association was explored in our population despite of alcoholic patients flow in our hospital, thus this study is to evaluate the prevalence of hypocalcemia & hypomagnesemia in alcoholic individuals population visited at our hospital so early nutritional measures that can accomplished to save the patients from life threatening complications with associated deficiency of these minerals.

PATIENTS AND METHODS: This one year cross sectional study was conducted at tertiary care hospital and the inclusion criteria of the study were the patient ≥20 years of age, either gender visited in ward or general outpatient department had history of alcohol intake for ≥ 3 years duration were recruited and enrolled in the study while the exclusion criteria of the study were the patients with history of hypo / hyper parathyroidism, chronic renal failure, malabsorption syndrome, osteoporosis, the patients already on diuretics, vitamin D, calcium and magnesium supplements, malignancy and pregnant & lactating ladies. After informed consent all the patients had detail clinical history, relevant clinical examination and along with baseline investigations advised the specific investigations accordingly. All the specific patients were evaluate for hypocalcemia and hypomagnesemia by taking 2 CC venous blood sample and sent to laboratory for analysis. The hypomagnesemia and hypocalcemia was labeled when serum magnesium and serum calcium level was < 1.8 mg/dL and <8 mg/dL. During the study period the state of confidence of all subjects were maintained and the financial compensation of the study were paid by the collaboration of whole research team. The data saved on predesigned proforma & kept secure while analyzed in SPSS version 16 to calculate the mean ± SD (quantitative variables) frequencies and percentages (qualitative variables).

RESULTS: During one year study period total fifty patients with alcohol intake were recruited, enrolled and evaluate for the glycemic status. The mean \pm SD of age, serum calcium and magnesium for whole population was 39.93 ± 6.75 years, 6.53 ± 2.21 and 1.42 ± 2.72 . Out of fifty individuals 45 (90%) were males and 05 (10%) were females while majority of the population belonged to urban areas 32 (64%). The cross tabulation for age, gender, duration of alcoholism, hypocalcemia and hypomagnesemia are presented in Table 1-7

TABLE 1: THE DISTRIBUTION FOR AGE AND GENDER

	=	GEN		
		Male	Female	Total
AGE (yrs)	20-29	6	0	6
	20-29	13.3%	.0%	12.0%
	30-39	16	2	18
	30-39	35.6%	40.0%	36.0%
	20.20	7	1	8
	30-39	15.6%	20.0%	16.0%
	40-49	8	1	9
	40-49	17.8%	20.0%	6 12.0% 18 36.0% 8 16.0%
	50+	8	1	9
		17.8%	20.0%	18.0%
Total		45	5	50
		100.0%	100.0%	100.0%

TABLE 2: THE DISTRIBUTION FOR GENDER AND DURATION OF ALCOHOLISM

		GENDER		
		Male	Female	Total
Duration (yrs)	3-4	25	0	25
	J -4	55.6%	.0%	50.0%
	4-5	15	5	20
	4-3	33.3%	100.0%	40.0%
	> 5	5	0	5
	> 3	11.1%	.0%	10.0%
Total		45	5	50
		100.0%	100.0%	100.0%

*P-value: 0.01

TABLE 3: THE DISTRIBUTION FOR GENDER AND HYPOCALCEMIA

		HYPOCALCEMIA		
		Yes	No	Total
GENDER Male		35	10	45
	1/1412	94.6%	76.9%	90.0%
	Female	2	3	5
	remaie	5.4%	23.1%	10.0%
Total		37	13	50
		100.0%	100.0%	100.0%

P-value: 0.05

TABLE 4: THE DISTRIBUTION FOR GENDER AND HYPOMAGNESEMIA

	-	HYPOMAGNESEMIA		
		Yes	No	Total
GENDER Male		36	9	45
	With	94.7%	75.0%	90.0%
	Famala	2	3	5
Female		5.3%	25.0%	10.0%
Total		38	12	50
		100.0%	100.0%	100.0%

P-value: 0.04

TABLE 5: THE DISTRIBUTION FOR DURATION OF ALCOHOLISM AND HYPOCALCEMIA

		HYPOCALCEMIA		
		Yes	No	Total
DURATION (yrs)	3-4	20	5	25
	3-4	54.1%	38.5%	50.0%
	4.5	13	7	20
	4-5	35.1%	53.8%	40.0%
	> 5	4	1	5
	> 3	10.8%	7.7%	10.0%
Total		37	13	50
		100.0%	100.0%	100.0%

P-value: 0.49

TABLE 6: THE DISTRIBUTION FOR DURATION OF ALCOHOLISM AND HYPOMAGNESEMIA

		HYPOMAGNESEMIA		
		Yes	No	Total
DURATION (yrs)	3-4	22	3	25
	3-4	57.9%	25.0%	50.0%
	4-5	13	7	20
	4-3	34.2%	58.3%	40.0%
	> 5	3	2	5
	> 3	7.9%	16.7%	10.0%
Total		38	12	50
		100.0%	100.0%	100.0%

P-value: 0.13

TABLE 7: THE HYPOMAGNESEMIA AND HYPOCALCEMIA IN ALCOHOLIC PATIENTS

	-	HYPOMAGNESEMIA		
		Yes	No	Total
HYPOCALCEMIA	Yes	32	5	37
		84.2%	41.7%	74.0%
	No	6	7	13
		15.8%	58.3%	26.0%
Total		38	12	50
		100.0%	100.0%	100.0%

P-value: 0.003

DISCUSSION: Alcohol consumption leads to alterations in cell functions and antioxidant system, in current series it was evident that the serum calcium and magnesium was reduced in alcoholic patients while the gamma glutamyl transferase (GGT) and BMI were increased than normal in alcoholic patients [14-16]. Patients with alcoholic liver disease may have low levels of calcium and has been found to increase calcium excretion; moreover the patients with liver cirrhosis have decrease calcium absorption of vitamin D while if alcoholic patients has malabsorption of fat, the calcium in the intestinal lumen may leads to formation of insoluble soaps and preventing intestinal absorption [17]. The long-term alcohol consumption can impair bone growth and bone tissue replacement (called as remodeling), causes decreased bone density and increased fracture risk through many hormones, cell types and growth factors that regulate bone and mineral metabolism. The alcohol consumption during adult life decreases bulk bone mass and causes weak adult bones which are more vulnerable to fracture. The alcohol consumption can also disrupt the balance between the remodeling of the bone tissue and erosions contributing in the pathogenesis of alcoholic bone disease. The imbalance is due to alcohol related osteoblasts inhibition, specialized cells which stored new bone while the former evidence found

that reduce alcohol drinking may reduce the risk of postmenopausal woman bone fractures [18-20]. The alcohol associated increase in BMI observed in present study and this might be due to increased fat or fat free mass & composition of body being affected by growth patterns, ethnicity, cultural behavioral patterns & Socio-economical status [21]. The reduction in adipose tissue in chronic alcoholic population who continue to drink is might be due to inadequate nutritional intake [22]. The GGT is an enzyme induced by alcohol and its serum levels also raise in response to hepatocellular injury and characterizes as marker for chronic, long-term misuse of drug (alcohol). Former evidence found that serum GGT activity is useful in evaluation of alcohol liver disease. However, Krastev Z, et. Al [23] did not observed any association between severity of liver injury and the extent of gamma GT rises during follow up period while some other studies reported as moderate specificity and sensitive & most common marker of chronic alcohol consumption and seem to describe more intense vulnerability to chronic alcohol liver disease [24].

The gamma GT rises rapidly due to heavy alcohol usage and with abstinence from alcohol it returns back to normal most rapidly [25]. Hypomagnesemia was the most common electrolyte disturbance observed in 29% alcoholic population and also most common electrolyte

abnormality detected in alcoholic individuals based on different pathophysiologic processes [26]. Hypomagnesemic subjects frequently had different electrolytes and acid base abnormalities such as, hypokalemia, hypocalcemia, hypophosphatemia & respiratory alkalosis as compared to normomagnesemic individuals and serves as an important risk factor for progression of fatty liver to steato-hepatitis and fibrosis [27, 28].

CONCLUSION:

In the present study, a total of fifty subjects were studied for serum calcium and magnesium status, it has been concluded that increase in BMI and GGT in alcoholics and contradictory there is significant decrease in serum calcium and magnesium level. Therefore, the estimation of serum calcium and serum magnesium along with GGT and BMI may be useful indicator for clinical prognosis in alcoholic population.

REFERENCES:

- Gilpin NW, Koob GF. Neurobiology of alcohol dependence: Focus on motivational mechanisms. Alcohol Res Health. 2008; 31(3): 185–195.
- 2. Mayfield RD, Harris RA, Schuckit MA. Genetic factors influencing alcohol dependence. Br J Pharmacol. 2008 May; 154(2):275–287.
- 3. Becker HC. Alcohol Dependence, Withdrawal, and Relapse. Alcohol Res Health. 2008; 31(4): 348–361.
- Woodman R Ferrucci, Guraiink. J. Anemia in older adults. Curr Opin Hematol. 2005; 12:123-28.
- Bengal V, Velayudhan A, Jain S. Social costs of alcoholism: a Karnatka pertspective. NIMHANS Journal.2000;18(12) 67.
- Das SK, KR. Hiran, S. Mukherjee and D. M Vasudevan. Oxidative stress in the primary event: effects of ethanol consumption in brain. Ind. J. Clin. Biochem. 2007; 22(1): 99-104

- 7. Cunningham JA, McCambridge J. Is alcohol dependence best viewed as a chronic relapsing disorder?. Addiction. 2012 Jan; 107(1): 6–12
- 8. White A, Hingson R. The Burden of alcohol use excessive alcohol consumption and related consequences among college students. Alcohol Res. 2014; 35(2): 201–218
- 9. Smith JP, Randall CL. Anxiety and alcohol use disorders comorbidity and treatment considerations. Alcohol Res. 2012; 34(4): 414-31.
- Sartor SE, Lynskey MT, Bucholz KK, Madden PAF, Martin NG, Heath AC. Timing of First Alcohol Use and Alcohol Dependence: Evidence of Common Genetic Influences. Addiction. 2009 Sep; 104(9): 1512–1518.
- 11. Rehm J. The risks associated with alcohol use and alcoholism. Alcohol Res Health. 2011; 34(2): 135–143
- 12. Abbott L, Nadler J, Rude RK. Magnesium deficiency in alcoholism: possible contribution to osteoporosis and cardiovascular disease in alcoholics. Alcohol Clin Exp Res. 1994 Oct;18(5):1076-82.
- 13. Kim MJ, Shim MS, Kim MK, Lee Y, Shin YG, Chung CH, et al. Effect of chronic alcohol ingestion on bone mineral density in males without liver cirrhosis. Korean J Intern Med.2003;18(3):174-80.
- 14. Gearhardt AN, Corbin WR. Body mass index and alcohol consumption: family history of alcoholism as a moderator. Psychol Addict Behav.2009;23(2):216-25.
- 15. Bruha R, Dvorak K, Petrtyl J. Alcoholic liver disease. World J Hepatol. 2012 Mar 27; 4(3): 81–90.
- Bellini M, Tumino E, Giordani R, Fabrini G, Costa F, Galli R, et al. Serum gammaglutamyl-transpeptidase isoforms in alcoholic liver disease. Alcohol Alcohol.1997;32(3):259-66.
- 17. Kalbfleisch JM, Lindeman RD, Ginn HE, et al: Effect of ethanol administration on urinary

- excretion of magnesium and other electrolytes in alcoholic and normal subjects. J Clin Invest .1963;42:1471-75.
- Gonzalez-Reimers E, Quintero-Platt G, Rodríguez-Rodriguez E, Martinez-Riera A, Alvisa-Negrin J, Santolaria-Fernandez F. Bone changes in alcoholic liver disease. World J Hepatol. 2015 May 28; 7(9): 1258-64.
- Lopez-Larramona G, Lucendo AJ, Gonzalez-Delgado L. Alcoholic liver disease and changes in bone mineral density. Rev Esp Enferm Dig.2013;105(10):609-21
- Sampson HW. Alcohol's harmful effects on bone. Alcohol Health Res World. 1998;22(3):190-4.
- 21. Rico H. Alcohol and bone disease. Alcohol Alcohol. 1990;25(4):345-52.
- 22. Ryle PR, Thomson AD. Nutrition and vitamins in alcoholism. Contemp Issues Clin Biochem. 1984;1:188-224.
- 23. Krastev Z, Mateva L, Danev S, Nikolov R. Clinical meaning of GGT activity in follow-up of patients with alcohol-related liver injury and cholestasis. Ital J Gastroenterol. 1992;24(4), 185-187.
- 24. Daeppen JB, Schoenfeld-Smith K, Smith TL, Schuckit MA. Characteristics of alcohol dependent subjects with very elevated levels of GGT. J Study Alcohol.1999; 60(5): 589-594.
- 25. Wu A, Slavin G, Levi AJ. Elevated serum gamma-glutamyl-transferase (transpeptidase) and histological liver damage in alcoholism. Am J Gastroenterol. 1976;65(4):318-23
- Elisaf M, Merkouropoulos M, Tsianos EV, Siamopoulos KC. Pathogenetic mechanisms of hypomagnesemia in alcoholic patients. J Trace Elem Med Biol.1995;9(4):210-4.
- 27. Assadi F. Hypomagnesemia: an evidence-based approach to clinical cases. Iran J Kidney Dis. 2010;4(1):13-9.
- 28. Swaminathan R. Magnesium Metabolism and its Disorders. Clin Biochem Rev.2003;24(2):47-66.