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RESEARCH ARTICLE

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A COMPARATIVE STUDY OF SERUM MAGNESIUM IN PULMONARY TUBERCULOSIS AND BRONCHIAL ASTHMA

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ABSTRACT

In the first half of the last century tuberculosis was the main focus of attention amongst all respiratory diseases. Bronchial asthma is a serious global health problem. The present study was carried out to estimate magnesium levels in serum in pulmonary tuberculosis and bronchial asthma cases and to compare them with the serum magnesium levels of controls.

This study was undertaken in 120 subjects, 40 diagnostic cases of pulmonary tuberculosis (group I), 40 diagnostic cases of bronchial asthma (group II), and 40 normal control groups (group III). Magnesium levels were analyzed from serum by spectrophotometer, using calmagite method. Our study shows a significant decrease (<.0001) serum magnesium levels in group I (pulmonary tuberculosis) and group II (bronchial asthma) cases as compared to group III (Control). Our study also reveals that the mean levels of serum magnesium are markedly lower in cases having far advanced disease (1.25±.080 meq/L) than in those with moderately advanced disease (1.37±.084 meq/L) or with minimal disease (1.50 meq/L).

Pulmonary tuberculosis and bronchial asthmatic patients have lower serum magnesium concentrations compared to healthy controls. This may be of use in future therapeutic management of pulmonary tuberculosis and bronchial asthma.

KEY WORDS

Magnesium, Pulmonary Tuberculosis, Bronchial asthma, Hypomagnesemia

INTRODUCTION

Tuberculosis (TB) is as old as mankind. It is considered as one of the most important infectious diseases, caused by a bacterium species called Mycobacterium tuberculosis. This bacterium pathogen (*M. tuberculosis*) was discovered and identified by Robert Koch in 1882 [1]. The 1990 World Health Organization (WHO) report on the Global Burden of Disease ranked TB as the seventh most morbidity-causing disease in the world, and expected it to continue in the same position up to 2020 [2].

Bronchial asthma is a serious global health problem and it is widely believed that the prevalence of asthma has increased over the last 25 years. It is characterized by increased responsiveness of the tracheobronchial tree to a multiplicity of stimuli[3]. Tuberculosis and bronchial asthma result in changes in the serum level of many electrolytes. Magnesium and zinc are two important electrolytes in the immune system and metabolism [4].

Malnutrition is frequently observed in patients with pulmonary tuberculosis, and also in bronchial asthma but their nutritional status, especially of magnesium is still poorly documented [5]. Magnesium (Mg) is the fourth most abundant mineral in the body and the most abundant intracellular divalent cation, with essential roles in many physiological functions[6]. Magnesium regulates hundreds of enzyme systems [7]. It is also involved in immune function, both in innate and acquired in immune response according to researchers [8.] According to Leo Galland, magnesium acts as a cofactor for immunoglobulin synthesis, immune cell adherence, antibody- dependent cytolysis, IgM lymphocyte binding, T helper B-cell adherence and additional responses [9]. Epidemiological studies have linked higher intakes of magnesium with lower incidences of respiratory problem[10]. Magnesium has been used to treat and improve lung function in some individuals of asthma[10]. Recently there has been considerable interest generated in the possible importance of magnesium ions in the regulation of bronchial smooth muscle tone directly and indirectly[11]. Britton et al. [12] demonstrated that a lower dietary magnesium intake was associated with impaired lung function, bronchial hyperreactivity and an increased risk of wheezing. Medications used in the treatment of asthma cause decreased magnesium value in total body stores [13] especially in acute asthma management [14].

In spite of all these knowledge regarding importance of magnesium in human body, very little is known about the levels of serum magnesium in pulmonary tuberculosis and bronchial asthma. The data that are available from such studies are not conclusive to draw definite relation between serum magnesium with pulmonary tuberculosis and bronchial asthma. Hence, the purpose of this study is to detect serum magnesium values in pulmonary tuberculosis and bronchial asthma and compare it with healthy subjects, and assessing the difference between them.

MATERIAL AND METHODS

The study was carried out in the department of Biochemistry, Rohilkhand Medical College and Hospital, Bareilly. The study included 120 subjects and divided into three different groups. Group I included forty diagnosed patients with pulmonary tuberculosis. There were thirty (30) males and ten (10) females. The ages of patients range from 20 – 56 years. They were recruited for the study after taking their due written consent. The criterion for selection of these cases was based on detailed history of all such patients, positive findings on clinical

examination, radiological, pathological and other supportive laboratory investigations. The classification of the extent of disease in cases of pulmonary tuberculosis as minimal, moderately advanced and far advanced was done according to the one described by National Tuberculosis Association, America, as cited by Pagel et al (1964) [15].

Group II included forty diagnosed patients with bronchial asthma. Thirty three (33) of them were males and seven (7) were females. The age range was 20—56 years for the patients. Diagnosis of bronchial asthma was based on clinical history, physical examination, spirometric pulmonary testing, reversibility of FEV1, or peak expiratory flow >15%, diurnal variation of peak expiratory flow rate >20%, and chest x-ray. Patients were under observation and regular examination. Information was obtained by a questionnaire including the patient's age, sex, and duration of asthma, nocturnal abuser, and symptoms of primary magnesium deficiency.

Group III included forty control subjects which were randomly picked among medical students, nurses and employees of the college as well as hospital, who were in the age group of 20 to 56 years. The control subject includes thirty (30) males and ten (10) females. Alcoholism, malnutrition, history of smoking, hypertension, history of chronic diarrhea, use of diuretics, reduced renal function and past history of severe lung disorders were excluded from the present study.

The fasting blood was collected from the cubital vein with all aseptic precautions in glass tubes free from electrolytes. Serum was separated and serum magnesium was measured by spectrophotometer at 530 nm, using Calmagite method [16].

The principle of the Calmagite method depends on the formation of coloured complex between magnesium ions and calmagite in alkaline medium. EGTA (ethylene glycol tetraacetic acid) reduces calcium interference, KCN (Potassium Cyanide) reduces interference of heavy metals, and surfactant reduces interference of proteins and lipaemic blood samples.

Statistical Analysis

The data were analyzed by using paired student's *t* test (95 percent confidence limits). P value of less than 0.05 was considered significant.

RESULTS

The present study comprises 120 participants, 40 diagnosed patients with pulmonary tuberculosis (Group I), 40 diagnosed patients with bronchial asthma (Group II) and 40 Healthy control group (Group III). The age and sex distributions of Group I, Group II, and Group III are shown in table 1(a) and table-1(b).

Table -1(a)

Age and sex distributions of Group I (pulmonary tuberculosis) and Group II (bronchial asthma)

Group I (pulmonary tuberculosis) n=40					Group II (bronchial asthma) n=40			
Age in years	**No of cases**	**%**	**Male**	**Female**	**No of cases**	**%**	**Male**	**Female**
20-29	10	25	09	03	10	25	10	02
30-39	14	35	12	05	14	35	11	03
40-49	09	22.5	05	02	09	22.5	07	01
50-56	07	17.5	04	00	07	17.5	05	01

Table-1(b)
Age and sex distributions of Group III (Control subjects)

Group-III (Control subjects) n=40				
Age in years	**No of cases**	**%**	**Male**	**Female**
20-29	10	25	10	03
30-39	14	35	12	04
40-49	09	22.5	05	02
50-56	07	17.5	03	01

As seen from the table-2, there is a significant decrease in serum magnesium levels with the advancement in age.

Table-2
Serum magnesium levels in Group I (pulmonary tuberculosis), Group II (Bronchial asthma) and Group III (controls) as per the age distribution

Age in years	**Serum Magnesium mean ± SD,(meq/L)**		**pvalue**	**Serum Magnesium mean ± SD,(meq/L)**		**pvalue**
	Pulmonary tuberculosis	**Control group**		**Bronchial asthma**	**Control group**	
20-29	1.36 ± .102	1.72±0.102	.0001	1.39±0.146	1.72±0.102	.001
30-39	1.31±.073	1.59±0.140	<.0001	1.27±0.080	1.59± .140	<.0001
40-49	1.29±.092	1.49± .067	<.001	1.25±0.116	1.49± .067	<.001
50-56	1.25±0.146	1.43±.050	.039	1.20±0.120	1.43±.050	.004

In the present series of pulmonary tuberculosis cases, serum magnesium values were found to be markedly low in those cases who had far advanced disease (stage-3) [mean serum magnesium value1.25±0.080 meq/L] than in those with moderately advanced disease (stage-2) [mean serum magnesium value1.37± 0.084 meq/L] or with minimal disease (stage-1) [mean serum magnesium value 1.50 meq/L] as shown in table-3.

Table 3
Serum magnesium levels in cases of pulmonary tuberculosis depending on the extent of the disease

Sl. No	Extent of disease	No. of cases	Serum Magnesium (meq/L),	
			mean	SD (±)
1.	Minimal	1	1.50	
2.	Moderately advanced	18	1.37	.084
3.	Far advanced	21	1.25	.080

Moreover, when the groups were compared to each other, the pulmonary tuberculosis group (Group-I) revealed a significant fall in serum magnesium levels in contrast to the control

group (Group-III). Also there was a significant decrease serum magnesium levels (p < .0001) in Group-II (bronchial asthma) cases as compared to control group (Group-III). Overall, Group-I (pulmonary tuberculosis) and Group-II (bronchial asthma) showed a significant fall (p value less than 0.0001) in average serum magnesium levels in contrast to the Group-III (control group) as shown in table-4.

Table-4

Showing serum magnesium levels (*meq/L*) in Group I (pulmonary tuberculosis), Group II (bronchial asthma), and Group III (control subjects)

Groups	Range	Mean	SD (±)	pvalue
Pulmonary tuberculosis (Group-I)	1.10—1.50	1.31	0.10	<.0001
Bronchial asthma (Group-II)	1.10—1.70	1.29	0.12	<.0001
Control (Group-III)	1.40—1.90	1.56	0.14	

Group I versus group III p<0.0001
Group II versus group III p<0.0001.

DISCUSSION

According to Durlach (2002), evaluation of total magnesium in plasma or serum appears as a better marker than ionized magnesium in magnesium imbalance, because it is easily available and inexpensive [17]. In the present study we used serum magnesium levels to asses the magnesium status in pulmonary tuberculosis and bronchial asthma.

In our study, group-I, group-II and group-III 14 (35%) cases were in the age group of 30—39 years, 10 (25%) cases were in the age group of 20—29 years, 9 (22.5%) cases were in the age group of 40—49 years and 7 (17.5%) in the age group of 50—56 years. There were 30 (75%) males and 10 (25%) females in group-I cases and 33 (82.5%) males and 7 (17.5%) females in group-II cases. In group-III cases, there were 30 (75%) males and 10 (25%) females (Table-1a and 1b), however such age and sex distribution resembled with the study of other author[18-20].

Our results depicted in table-2, indicates that the levels of serum magnesium were lower with advancement in age as in cases of pulmonary tuberculosis and bronchial asthma. Similar results have been documented in previous studies conducted by Jain et al[19]. and O.O.Oladipo et al[21] .

This study also observed that serum magnesium concentration were found to be markedly low in those cases who had far advanced disease (stage-3) [mean serum magnesium value1.25±0.080 meq/L] than in those with moderately advanced disease (stage-2) [mean serum magnesium value1.37± 0.084 meq/L] or with minimal disease (stage-1) [mean serum magnesium value 1.50 meq/L]. Similar findings were also reported by Jain et al[19].

In table-4, it has been observed that hypomagnesemia in pulmonary tuberculosis patients is higher as compared to general population. The results are in agreement with the results of other authors [19, 22, 23]. But the results of this study are contrary to Podlesna (1972) research [24], who did not find any significant difference in the concentrations of serum magnesium between pulmonary tuberculosis patients and controls. However, he found hypermagnesemia in cases of pneumonia and advocated this investigation to differentiate

pneumonia from pulmonary tuberculosis. Elo et al (1970) [25] also did not find any significant change. The exact cause of hypomagnesemia in pulmonary tuberculosis patients cannot be clearly explained. Hypomagnesaemia in malnutrition has been observed by several workers [26, 27], while Wacker and Vallee,(1958) [28] have also stressed that hypomagnesaemia can occur due to poor dietary intake. One of the possible factors of hypomagnesemia in our study is that it is possible due to poor dietary intake of magnesium due to malnutrition.

In Group-II study (bronchial asthma) shows that the decreased serum magnesium concentration is a constant finding during the active phase of bronchial asthma. Our results agree with previous findings of some authors[20, 21, 29]. Causes of hypomagnesaemia in asthma may be multifactorial. It may be genetically determined[30]. It is also attributed to either low magnesium intake in asthmatics or increased urinary loss of magnesium as a side effect of therapy with β2 agonist, theophylline and corticosteroids [31]. The possible factors participating in the development of hypomagnesemia in our patients might be low dietary intake of magnesium or increased urinary loss of magnesium as a side effect of therapy.

Improvement of magnesium deficiency through magnesium supplement may be effective in pulmonary tuberculosis and bronchial asthma patients, and achieving better therapeutic results, but further investigation is needed to complete this hypothesis.

CONCLUSION

From the above study we conclude that decreased serum magnesium is a constant finding during pulmonary tuberculosis and active phase of bronchial asthma. Therefore, a routine biochemical assessment of magnesium status in patients with pulmonary tuberculosis and bronchial asthma is an important step in the management of protocol to reduce progression of the disease.

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