MAGNESIUM METABOLISM IN LEPROSY

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Although there is considerable evidence of the importance of magnesium in disease conditions, studies of magnesium metabolism in patients with leprosy have not been reported. Observations made in the clinical laboratory of this hospital are reported here.

The literature is sparse regarding the metabolism of magnesium in man. Studies by Martin et al. (5) indicate that magnesium-free urine does not occur, but that there may be deficits due to continued urinary loss in the absence of a minimal magnesium intake. Accelerated loss of magnesium in the urine may occur under certain circumstances, such as diuretic therapy. A low serum magnesium may also be due to a decreased gastrointestinal absorption, or an increased intracellular shift of magnesium ions.

Magnesium is a component of soft tissue, and is one of the cations of intracellular fluid and of extracellular fluids including serum. It is also a component of certain organic complexes, notably those containing members of the B group of vitamins. It appears to catalyze certain enzyme reactions, as phosphatase (7). Magnesium also, apparently, plays an important role in protein and carbohydrate metabolism, bone formation, and neuromuscular conduction (6). In chronic renal disease, low, normal or high magnesium levels have been reported, and in the acute type the serum magnesium level has risen above normal (4,5).

Magnesium is in an unusual position in the body, since there is a large amount present in the extracellular phase of bone, and in the cells of the body, but only a very small quantity in the extracellular fluids. The magnesium present in the extracellular compartment, as measured by the serum level, is available for study.

The normal range of concentration of magnesium in the serum, as determined by titrimetric and other methods, has been reported as from 1.4 to 3.6 mgm. per 100 cc. in normal adults by Cantarow, Dine and Meyers (1, 2, 6). In the present study, magnesium levels were determined by a spectrophotometric method in a group of 27 nonpatient controls and ranged from 0.65 to 1.20 mgm. per 100 cc. Our normal values are significantly lower than those reported by other workers, due perhaps to variations in methods used.

PRESENT STUDY

Venous blood was drawn in the fasting state, and the separated sera were used for the chemical analysis. The serum magnesium was determined by a spectrophotometric adaptation based on the methods and modification of Kolthoff (1927), Hirschfelder, Serles and Haury (1934), Haury (1938) and Kunkel, Pearson and Schweigert (1947) and described fully by Fister (3). A Coleman Junior spectrophotometer was used for the readings.

Multiple magnesium determinations were made in a total of 177 cases of leprosy, comprising 156 of the lepromatous and 21 of the tuberculoid types. The gross results are shown in Table 1.

Table 1.—Serum magnesium in 177 leprosy cases, lepromatous and tuberculoid, and 27 normal controls, minimum, maximum and average.

Type and group	Number of cases	Serum magnesium (mgm./100 cc.)				
		Minimum	Maximum	Average		
Lepromatous	With the part of the					
Active	113	0.00	2.00	0.86		
Quiescent	43	0.10	1.55	0.78		
Total	156	0.00	0.20	0.83		
Tuberculoid		-				
Active	8	0.45	1.05	0.70		
Quiescent	13	0.65	1.70	0.93		
Total	21	0.45	1.70	0.84		
Controls	27	0.65	1.20	0.83		

a "Active" signifies bacteriological positivity, not clinical activity of the disease; "quiescent" signifies bacteriological negativity (see Table 2).

The lepromatous and tuberculoid cases as a group showed arithmetical averages within the normal range of our controls. The distribution curve varies somewhat. Of the total 177 cases, 132 (75.2%) had levels within the normal range. Slightly elevated levels were seen in 17 cases, 16 lepromatous and 1 tuberculoid. Low levels were seen in 27 of the lepromatous cases and 1 of the tuberculoid type. The figures are shown in Table 2, which correlates the findings with the bacteriologic status.

In 14 of the 156 lepromatous cases in the series, a clinical diagnosis of amyloid nephrosis had been made. Amyloidosis is a rather common complication of long-standing lepromatous leprosy, at least in this hospital, and the uremia resulting from amyloid nephrosis is a major contributing cause in 40 per cent of deaths that occur here. Abnormally low magnesium levels were found in 8 of these 14 cases of this form of kidney disease, a significantly greater incidence of magnesium depletion than was seen

Table 2.—Evaluation of the serum magnesium levels and the bacteriologic findings.

	1-1-1	Number of cases	Serum magnesium (mgm./100 cc.)		
Type and group	Bacteriology		0.0 - 0.64	0.65 - 1.20	1.21 - 2.0
Lepromatous	D		00	70	
Active	Positive	113	23	79	11
Quiescent	Negative	43	4	34	5
Tuberculoid					
Active	Positive	8	1	7	0
Quiescent	Negative	13	0	12	1
Totals		177	28	132	17

a Concerning the groups, see footnote of Table 1.

in any of the tuberculoid cases or in the uncomplicated lepromatous ones. Slightly elevated concentrations were found in 6 of the 14 cases. These results are shown in Table 3.

Table 3.—Serum magnesium in 14 cases of lepromatous leprosy complicated with amyloid nephrosis, 106 determinations.

		Serum magnesium (mgm./100		
Case No.	Number of analyses	Range of values	Average	
1050	3	0.15 - 0.35	0.25	
1635	3	0.45 - 1.75	0.98	
1401	3	0.35 - 1.75	1.11	
1332	3	0.85 - 1.05	0.95	
1954	4	0.35 - 0.95	0.65	
2203	5	1.00 - 1.50	1.18	
2074	5	1.00 - 1.80	1.07	
2108	6	0.00 - 1.60	0.79	
1382	6	0.75 - 1.80	1.18	
836	7	0.75 - 1.90	1.28	
796	9	0.75 - 1.90	1.15	
2163	15	0.00 - 0.75	0.52	
1339	16	0.00 - 0.55	0.16	
1740	21	0.45 - 2.00	1.35	

SUMMARY AND CONCLUSIONS

Serum magnesium determinations were performed in 177 cases of leprosy, of which 156 were of the lepromatous and 21 of the tuberculoid type. As controls, 27 nonpatients were similarly examined.

Of the 177 cases, 132 (75.2%) showed magnesium levels within the normal range. Significantly low concentrations were found in 27 lepro-

matous cases and 1 tuberculoid case which were bacteriologically positive. Of the 28 cases with low levels, 8 had a clinical diagnosis of amyloid nephrosis and one had diabetes. Slightly elevated levels were seen in 16 lepromatous cases and 1 tuberculoid case. Of this latter group, 6 had a clinical diagnosis of amyloid nephrosis.

Leprosy *per se* does not appear to affect magnesium metabolism. In the series studied, bacteriologically-positive lepromatous cases complicated with amyloid nephrosis tended to show abnormal elevation or depletion of serum magnesium; in 3 of the 14 cases with the tuberculoid condition zero levels were recorded.

RESUMEN

Ejecutáronse determinaciones del magnesio sérico en 177 casos de lepra, siendo 156 de éstos de la forma lepromatosa y 21 de la tuberculoidea. Como testigos, se examinó en forma semejante a 27 sujetos que no eran leprosos.

De los 177 casos, 132 (75.2%) revelaron concentraciones de magnesio dentro de los límites normales. Concentraciones significativamente bajas fueron observadas en 27 casos lepromatosos y 1 caso tuberculoideo, que eran positivos bacteriológicamente. De los 28 casos de índices bajos, en 8 había un diagnóstico de nefrosis amiloidea y uno tenía diabetes. Descubriéronse cifras ligeramente elevadas en 16 casos lepromatosos y 1 caso tuberculoideo. En este último grupo, en 6 había un diagnóstico clínico de nefrosis amiloidea.

La lepra no parece afectar per se el metabolismo del magnesio. En la serie estudiada, los casos lepromatosos positivos bacteriológicamente y complicados con nefrosis amiloidea tendieron a revelar elevación o empobrecimiento anormales del magnesio sérico; en 3 de los 14 casos de la forma tuberculoidea se registraron índices de cero.

REFERENCES

- CANTAROW, A. and TRUMPER, M. Clinical Biochemistry. Philadelphia: W. B. Saunders Company, 1950.
- DINE, R. F. and LAVITES, P. H. Serum magnesium in thyroid disease. J. Clin. Invest. 21 (1942) 781-785.
- FISTER, H. J. Manual of Standardized Procedures for Spectrophotometric Chemistry. New York: Standard Scientific Supply Corp., 1950.
- HIRSHFELDER, A. D. Effect of renal insufficiency upon plasma magnesium and magnesium excretion after ingestion of magnesium sulphate. J. Biol. Chem. 104 (1934) 647-653.
- MARTIN, E. H., MEHL, J. and WERTMAN, M. Clinical studies of magnesium metabolism. Med. Clin. North America 36 (1952) 1157-1169.
- MEYERS, G. B. and ISERI, L. T. Abnormalities of body water, sodium, potassium and magnesium. A. M. A. Arch. Int. Med. 95 (1955) 503-537.
- STEARNS, G. Human requirements of calcium, phosphorus and magnesium. J. American Med. Assoc. 142 (1950) 478-480.