Trace Elements in Polyneuropathy - Complicated Chronic Renal Failure.

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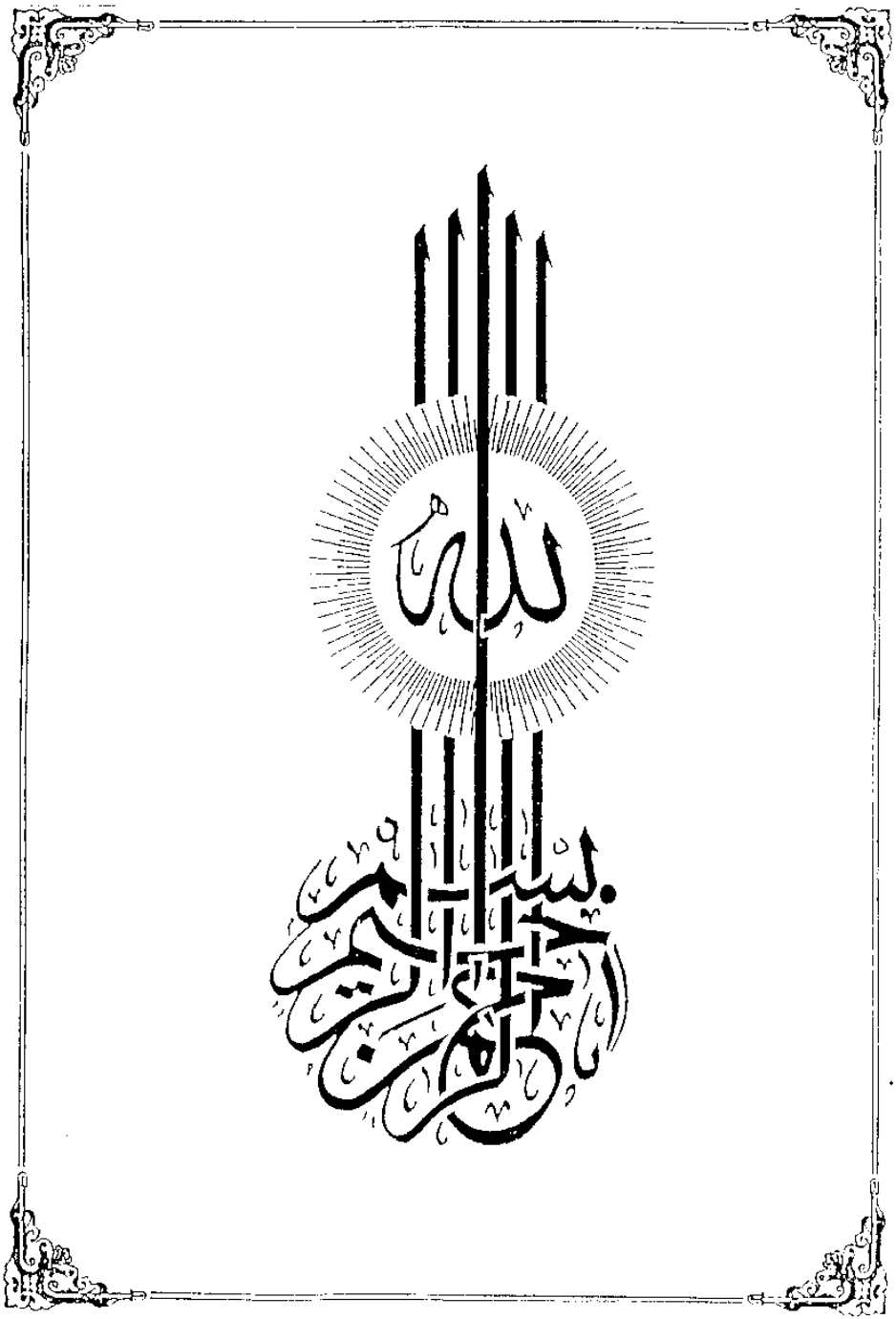

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Trace elements performs functions indispensable to maintenance life, growth and reproduction, changes in the level of trace elements may impair cellular and physiological functions and often causes illness.

The term of neuropathy callectively describes the vacid syndromes of impairment of function of the peripheral nervous system. These syndromes vary from a mere loss of reflexes, loss of vibration sense and position sense in the legs to severe limb or trunk pain with few objectives finding.

Polyneuropathy can occures with variety of conditions of which renal failure is one of the important causes. Pathogenesis of polyneuropathy in renal failure seems to be multifactorial. Changes of trace elements specially Zinc, Magnesium and Copper in chronic renal failure may participate in such pathogenesis.

The aim of this work is to study the serum concentrations of Zinc, Magnesium and Copper in renal

failure complicated with polyneuropathy and to correlate changes in their levels with the conduction velocity in the cases that will be studied.

REVIEW OF LITERATURE

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TRACE ELEMENTS

Certain elements although present in minute amounts in the tissues, are essential nutrients. Their presence was long overlooked and even after their identification, shortcomings of the methods available for analysis together with failure to recognize their importance led to their being reported as present in traces and therefore to their deprecatory designation as trace elements. They perform functions indispensable to maintenance of life, growth and reproduction.

Inadequate intake of trace elements may impair cellular and physiological functions and often causes illness.

ZINC

Raulin., (1869), first showed that Zinc was essential for growth of Aspergillus niger. This was confirmed 40 years later by (Bertrand & Javillier., 1911) and in (1926), its essentiality for higher forms of plant life was established, (Sommer and Lipman., 1926).

Tadel et al., (1934), proved that Zinc was essential for the growth of the rats but recognition that lack of Zinc could be a limiting factor in nutrition under non-experimental conditions was delayed for another 20 years untill (Trucke & Salmon., 1955), found that an increased Zinc intake corrected parakeratosis in swine. Subsequent studies proved that Zinc deficiency can be responsible for a syndrome of infantilism in adolescence, in (Prasad et al., 1961) and (Prasad et al., 1963).

Availability and Absorption:

Human requirements have been estimated as between 10 to 20 mg/day for an adult, but since the metal is widespread in nature, cases of gross deficiency are

rarely due to nutritional causes only. Prasad et al., (1963), demonstrated that many causative agents interplay to influence the deficiency of Zinc and other trace elements among the Egyptian Villagers.

Zinc is widely distributed in foods such as meat, eggs, fish, coca, tea, nuts, grains, legumes and cow's milk.

All cereals and most vegetables contain phytate and fibres, which can bind Zinc, particularly in the presence of calcium and reduce its biological avialability, (Cartwright,et al., 1960), also a high content of Copper in diet causes decrease in Zinc absorption, (Carter et al., 1969).

On the other hand, protein content of the diet appeared to influence the absorption and retention of Zinc, (Osis et al., 1973).

Zinc is absorbed from the small intestine. It does not take place by simple diffusion.

Davis et al., (1968), demonstrated that there are at least two types of proteins in the intestinal nucosa that bind Zinc and play a role in its absorption,

(Evan et al., 1973), showed that Zinc absorption is inversely related to intestinal mucosal Zinc content and they suggested that the latter may be regulated by Zinc content of plasma, but the mechanism of the regulation of Zinc absorption is not known completely.

Distribution of Zinc in Body:

An adult 70 Kgm. normal human body contains 1.4 to 2.3 gm of Zinc with the largest amount being localized in muscles, bone, and skin, (Prasad et al., 1970). They also demonstrated that Zinc is present within all body cells, existing in highest concentration in the liver, pancreas, kidney, heart, pituitary, adrenals, prostate and leucocytes.

The concentration of Zinc in plasma when assayed by modern techniques is approximately 100 ug/100 ml. with a standard deviation of approximately 10 ug/100 ml. (Pakarek et al., 1970). Parisi and Vallee.,(1970), stated that plasma Zinc is distributed between two main fractions, 30-40% is firmly bound to alpha macroglobulin and the remaining Zinc is apparently loosely bound to albumin. Concentration of Zinc in serum is higher by 6 to 15 ug/100 ml. than those in plasma,

mainly because Zinc is released from platelets during clot formation, (Faley et al., 1968).

Erythrocytes contain 12-14 ug/ml. of R.B.C_s. So erythrocytes contain 75-85% of blood Zinc, primarily in the Zinc metaloenzyme carbonic anhydrase. Plasma contains 12-22% and leucocytes contains 3% of blood Zinc, (Vallee., 1959).

There is no specific plasma protein has been definitely identified as a transport protein for Zinc, like transferrin for iron and ceruloplasmin for Copper, (Prasad., 1966).

So, the exchange of Zinc from one tissue to another is limited. Carter et al., (1969), used radioactive $Zinc^{(65)}$ to demonstrate that liver, kidney, spleen, intestinal mucosa, lung, pancreas, thyroid, pituitary, testis and adrenals have a rapid uptake as well turnover of $Zinc^{(65)}$, on the other hand, the turnover rate of $Zinc^{(65)}$ is relatively slow in brain, muscle and erythrocytes, and very slow in hair and bone.

Excretion:

Spencer et al., (1965), demonstrated that the gastrointestinal tract is the main excretory pathway for Zinc, through which a maximum of 1.5 mg can be excreted per day. This amount is through pancreatic juice and bile intestinal secretion, 300-700 ug/ of Zinc may be lost in urine daily independent on the urine volume. This amount may increase markedly under conditions of stress, e.g. prolonged starvation, (Spencer., 1970), or traumatic injury, (Fell et al., 1973).

Prasad et al., (1963), demonstrated that 1 mg/liter may be lost in sweat. Zinc also may be partly excreted through desquamation of skin and via colostrum and about 308-616 ug/menses, (Shraeder., 1967). As early as 1942, it was noted that uraemic patients excreted excess Zinc in their urine, (Lindeman, Baxter and S. Kraikitpanitch., 1978).

Metabolic Importance of Zinc:

Numerous Zinc metalloenzymes as well as Zinc dependent enzymes have been described since the intial discovery of carbonic anhydrase in (1939), (Keilin and Mon., 1940). Also Zinc is known to be as essential