HEMATOLOGIC CHANGES IN SEVERE RICKETS BEFORE & AFTER TREATMENT

THESIS

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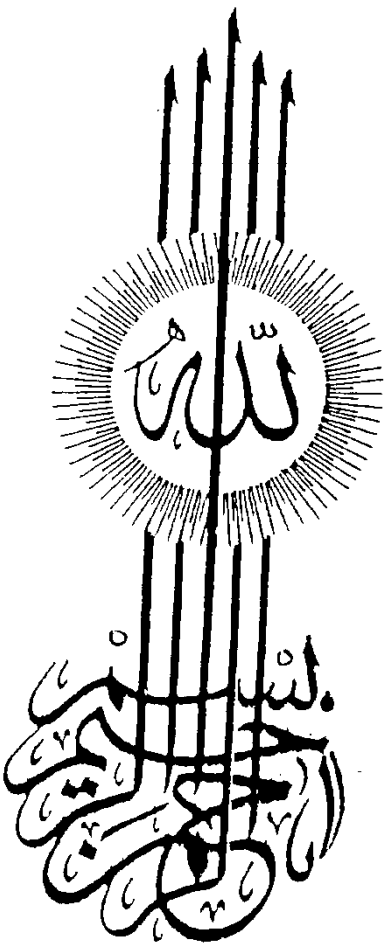

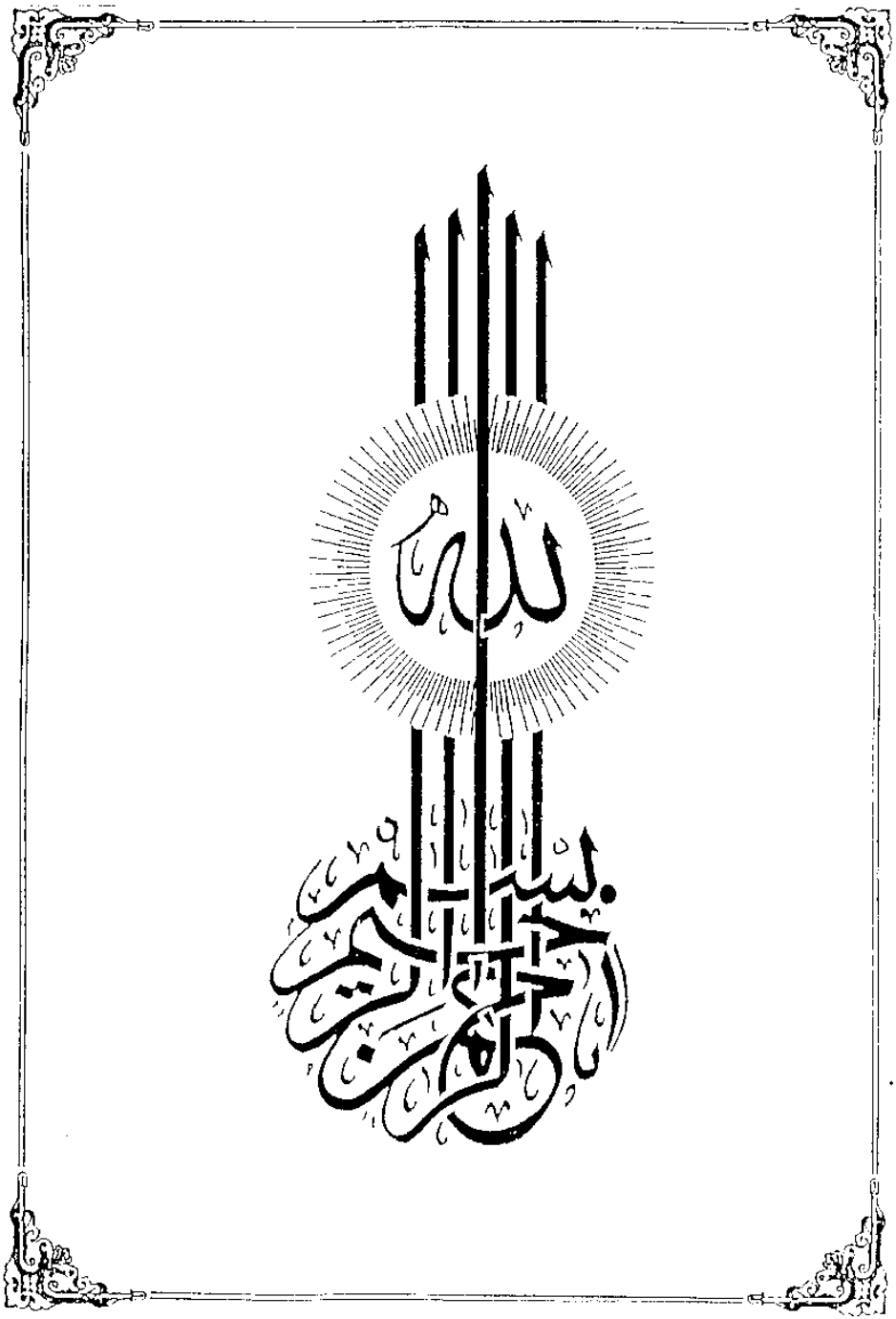

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Mohamed Saber Rezk

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and

AIM OF THE WORK

Introduction

In infantile rickets it is common to find variable degrees of anemia, sometimes associated withe leucocytosis. Several terms were given to describe this association such as Pseudoleukemia infantum or von Jacks anemia.

The exact pathogenesis of these hematologic changes was not conclusively settled.

yet, the depressed immunity observed in such cases may predispose to infection (Deyab,Z. 1981) and consequently to anemia and leucocytosis.

On the other hand, anemia may be secondary to associated nutritional deficiencies as iron and other vitamins (Deluca, 1980)

However, the clinical presentation of some rachitic patients with hepatosplenomegaly with anemia raised the question of extra medullary hematopoisis due to bone marrow depression(ozsoylu, 1980).

with this diversity of possible causes of hematologic changes,we decided to examine patients with infantile rickets before and after treatment to confirm if vitamin D deficiency might be an etiologic factor of these changes.

Aim of work:

- To confirm if vitamin D deficiency might be an etiologic factor of hematologic changes in severe rickets.

REVIEW OF LITERATURE

VITAMIN D

Metabolism of Vitamin D:

Vitamin D_3 (cholecalciferol) is normally provided to man in two ways: It is produced in the skin as a result of ultraviolet irradiation of 7-dehydrocholesterol, and is also derived from the intestinal absorption of the intact vitamin from the diet.

Vitamin D_2 (ergocalciferol) is derived from plant sterols and is used either as a therapeutic agent or for the fortification of foods. Both vitamin D_3 and vitamin D_2 are inactive in many biological systems and must undergo a series of metabolic transformations before exerting their effects at target tissues (Deluca, 1976).

The active metabolites of vitamin D can now be considered hormones in the sense that their production rates are controlled and they affect target tissues far from sites of production. In common with many endocrine systems, disorders of vitamin D metabolism can arise because of changes in the secretion of the active hormones or due to changes in the sensitivity of target tissues.

Production of 25-hydroxyvitamin D (25-OHD):

The first step in the metabolism of vitamin D_3 (or vitamin D_2) is its hydroxylation in the 25 position by the enzyme 25-hydroxylase. This step occurs predominantly in the liver (Olsan et al., 1976) and results in the formation of 25-hydroxyvitamin D (25-OHD).