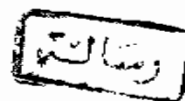


**STUDY OF IMMUNOGLOBULINS IN CHILDREN WITH  
CHRONIC RENAL INSUFFICIENCY**

**THESIS**



Submitted for partial fulfillment of the  
**Master Degree in Pediatrics**

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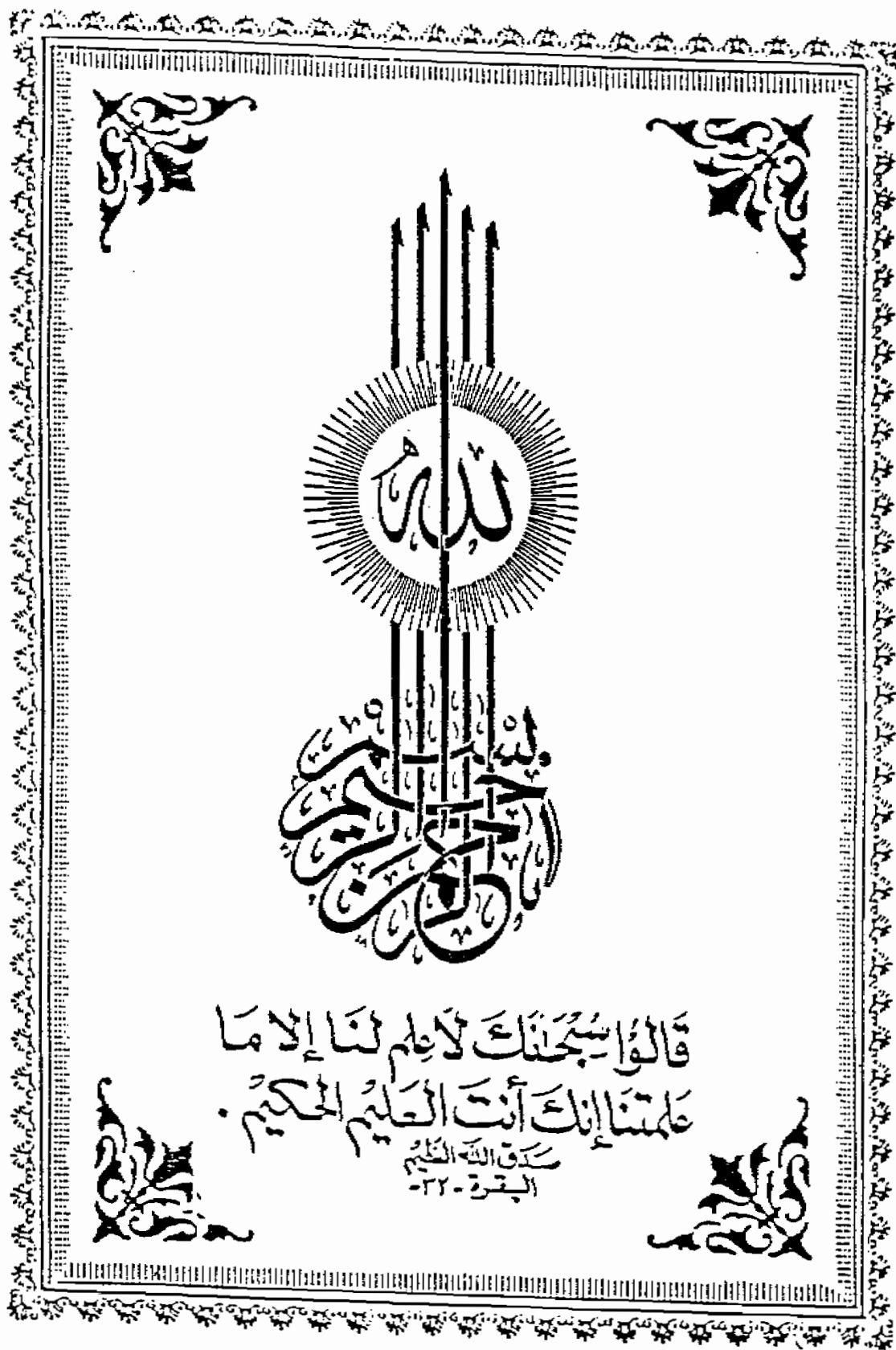
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# ***Introduction***

## INTRODUCTION

The susceptibility of uremic patients to infectious diseases has been widely reported, but the host immune factors associated with the increased incidence of infection have not been clearly defined (*Goldblum and Reed, 1980*).

Uremia is claimed to cause impaired immune responsiveness (*Wilson et al., 1965* and *Goldblum and Reed, 1980*). The mechanism of this impairment is not fully understood. Unknown uremic toxin (*Touraine et al., 1975* and *Raska et al., 1980*) and reduction in cellular reactivity have been implicated (*Huber et al., 1969* and *Alvey et al., 1981*).

There have been only a few detailed investigations concerned with the antibody response in uremic patients, and available data are controversial: some authors reported normal responses (*Friedman et al., 1980*). While others found reduced antibody responses (*Macy et al., 1991*).

Some investigators found a diminished responses to tetanus toxoid in chronic uremic patients. On the other hand, others found a normal

secondary response to diphtheria toxoid in patients with uremia (*Goldblum and Reed*, 1980).

The influence of dialysis membrane on immunoglobulin production was studied in vitro and it was concluded that the long term exposure of mononuclear cells to artificial surfaces during dialysis may contribute to the impaired humoral response observed in dialysis patients. This effect may be due to a decline in B-cell stimulation by monocytes (*Paczek et al.*, 1990).

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# *Aim of the Work*

## **AIM OF THE WORK**

This work will be carried out with the aim of studying the serum immunoglobulins of uremic children and the effect of hemodialysis on their levels hoping at identification and early treatment of patients at risk of infection.

# ***Review of Literature***

## CHRONIC RENAL FAILURE

### Definition

Chronic renal failure can be defined as chronic impairment of kidney functions of gradual onset leading eventually to a complex of clinical, chemical and metabolic disturbances with failure of the kidneys to maintain a normal internal environment (*Brenner and Lazarus*, 1987).

Uremia means "Urine in the blood" and implies that certain substances such as urea or other products normally excreted into the urine are retained, thereby causing systemic toxicity. The term "Uremia" was originally applied by *Priorry and Heritier* (1840). It seems preferable to use the expression "renal failure" which is most often due to extensive and irreversible disease of the kidneys. In certain cases it may be partially or wholly reversible.

In most reversible forms of renal failure, glomerular filtration is reduced by factors which probably affect every glomerulus. This includes reduction in filtration pressure or increase in the pressure opposing filtration. The increase in the pressure opposing filtration may