PREVALENCE OF HELICOBACTER INFECTION IN PATIENTS WITH CHRONIC RENAL FAILAIRE

THESIS
Submitted for partial fulfilment of
Master Degree in

Internal Medicine

Presented by

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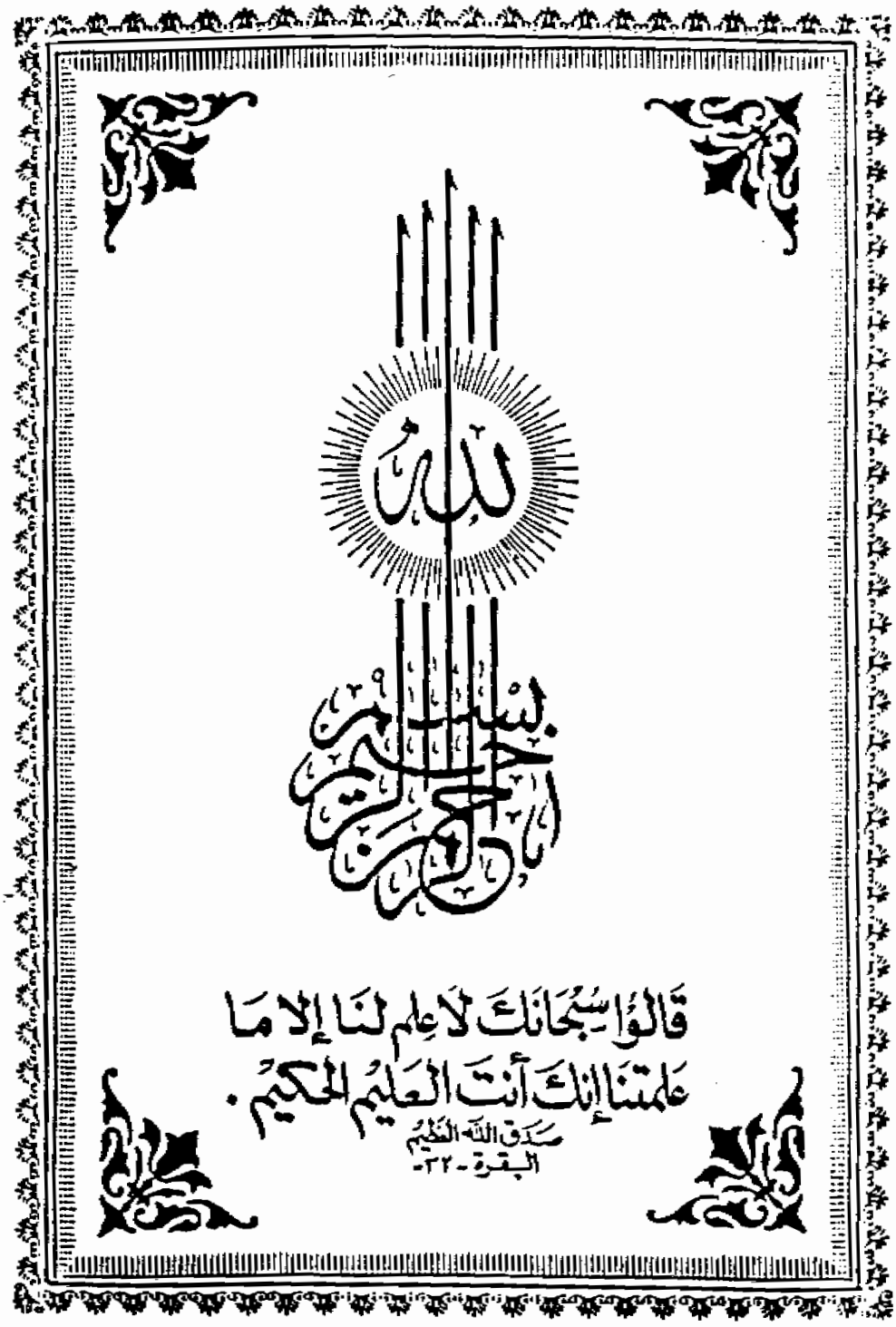
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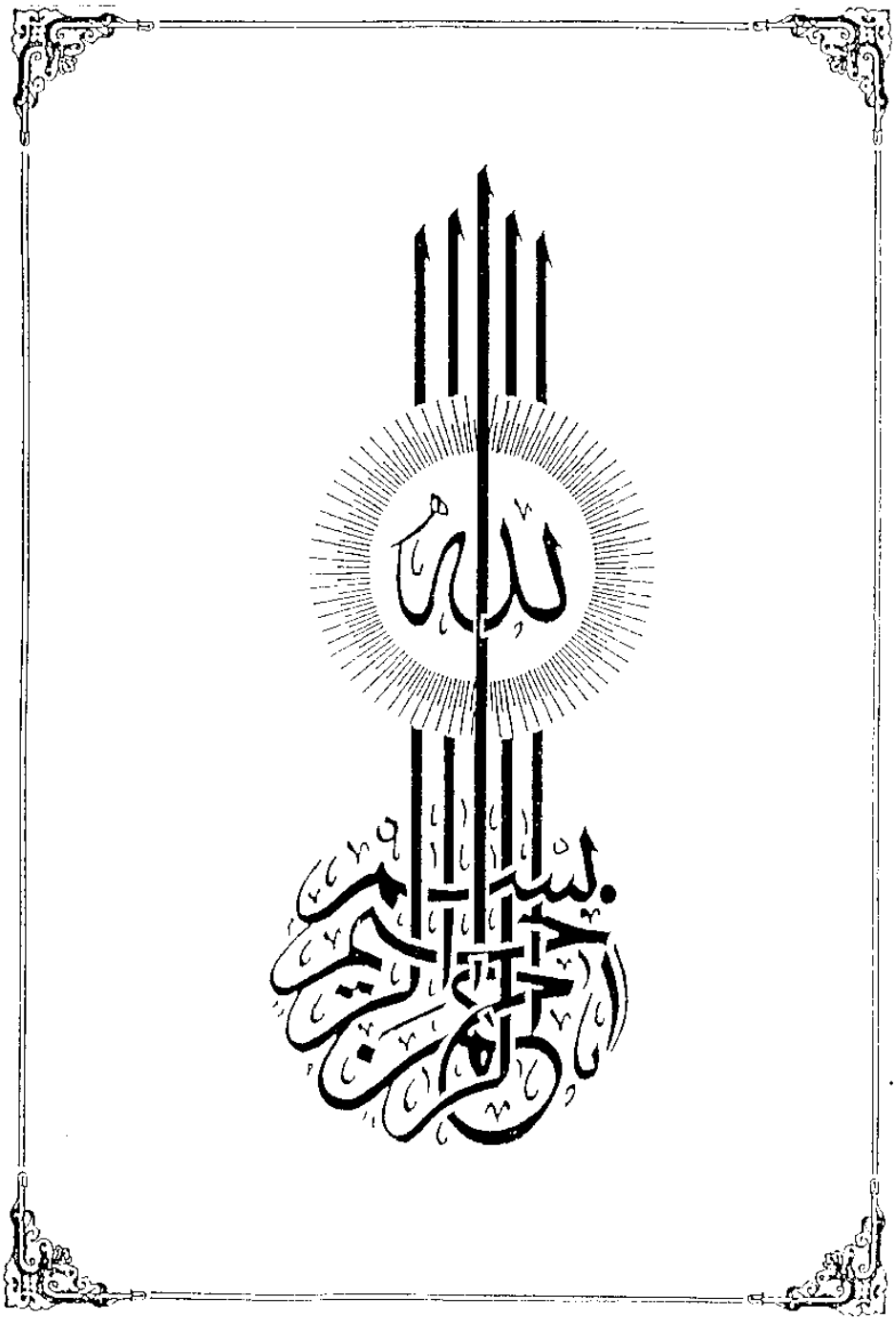

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CONTENTS

LIST OF ABBREVIATIONS

1	Ab	Antibodies
2	AIDS	Acquired immune deficiency syndrome
3	Anti-H.P	Antibodies against Helicobacter pylori
4	CBS	Colloid bismuth subcitrate
5	CLO	Campylobacter like organisms
6	CMV	Cytomegalovirus
7	CNS	Central nervous system
8	Conc	Concentration
9	CRF	Chronic renal failure
10	DU	Duodenal ulcer
11	ELISA	Enzyme linked immunosorbent assay
12	ESRF	End stage renal failure
13	FSH	Follicle stimulating hormone
14	GI	Gastrointestinal
15	GIT	Gastrointestinal tract
16	GU	Gastric ulcer
17	HCL	Hydrochloric acid
18	H&E	Haematoxylin & Eosine stain
19	H.J.	Helicobacter Jujeni
20	HLO	Helicobacter like-organisms
21	H.P.	Helicobacter pylori

22	K	Potassium
23	LH	Luteinizing hormone
24	Na	Sodium
25	NSAIDS	Non steroidal anti inflammatory drugs
26	NUD	Non-ulcer dyspepsia
27	PUD	Peptic ulcer disease
28	RF	Renal failure
29	UGT	Upper gastrointestinal tract
30	YS	Years

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INTRODUCTION

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Helicobacter pylori is a gram-negative microaerophilic bacillus which was isolated from antral mucosa. Colonization of gastric mucosa by H.P. is associated with antral gastritis, peptic ulcer disease and non ulcer dyspepsia. But the role of H.P. in the pathogenesis of peptic ulcer disease remains unclear.

Uremic patients whether on maintenance dialysis or not are prone to develop GIT complications. The gastric mucosa of patients with CRF by its high urea concentration was postulated to be an attractive nich for H.P. colonization, and this was supposed to be partially responsible for the GIT symptoms in these patients.

AIM OF THE WORK

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This study aims to determine the prevalence of endoscopic and histopathological gastroduodenitis and its relation to the presence of H.P. organism in patients with chronic renal failure whether under regular dialysis or not, and compare these results to the prevalence of H.P. in patients with non ulcer dyspepsia and normal kidney function.

CHRONIC RENAL FAILURE

CHRONIC RENAL FAILURE

Systemic Consequences of CRF

Natural history

*** Staging**

CRF is a continuous process that begins when some nephrons are lost and ends when the remaining nephron population can no longer sustain life. Clinicians generally recognize three or four stages of CRF that merge into one another. The first stage may be called renal insufficiency. GFR is reduced and azotemia is present, as well as nocturia & mild anaemia. Some authorities divide this stage into two, labelling the early period, when GFR is 50% or more of normal as the phase of diminished renal reserve. The rationale for this distinction is that the modest serum creatinine elevation may escape recognition. We believe it is better to realize that if creatinine concentration is 1.5 mg/dl, whereas normal is 1 mg/dl GRF has been reduced to 1/1.5 or 67% of normal (Maher, 1975).

The second stage is that of renal failure, where the kidneys no longer regulate the volume and the composition of extracellular fluid adequately, and hypocalcaemia, hyperphosphatemia and acidemia appear. Hyperkalemia can occur, although it is usually prevented by extra-renal adaptation.