DIAGNOSIS OF RENAL AFFECTIONS IN SYSTEMIC LUPUS ERYTHEMATOSUS

A Thesis Submitted in Partial Fulfillment of the Requirement
For the Doctor Degree in Physical Medicine

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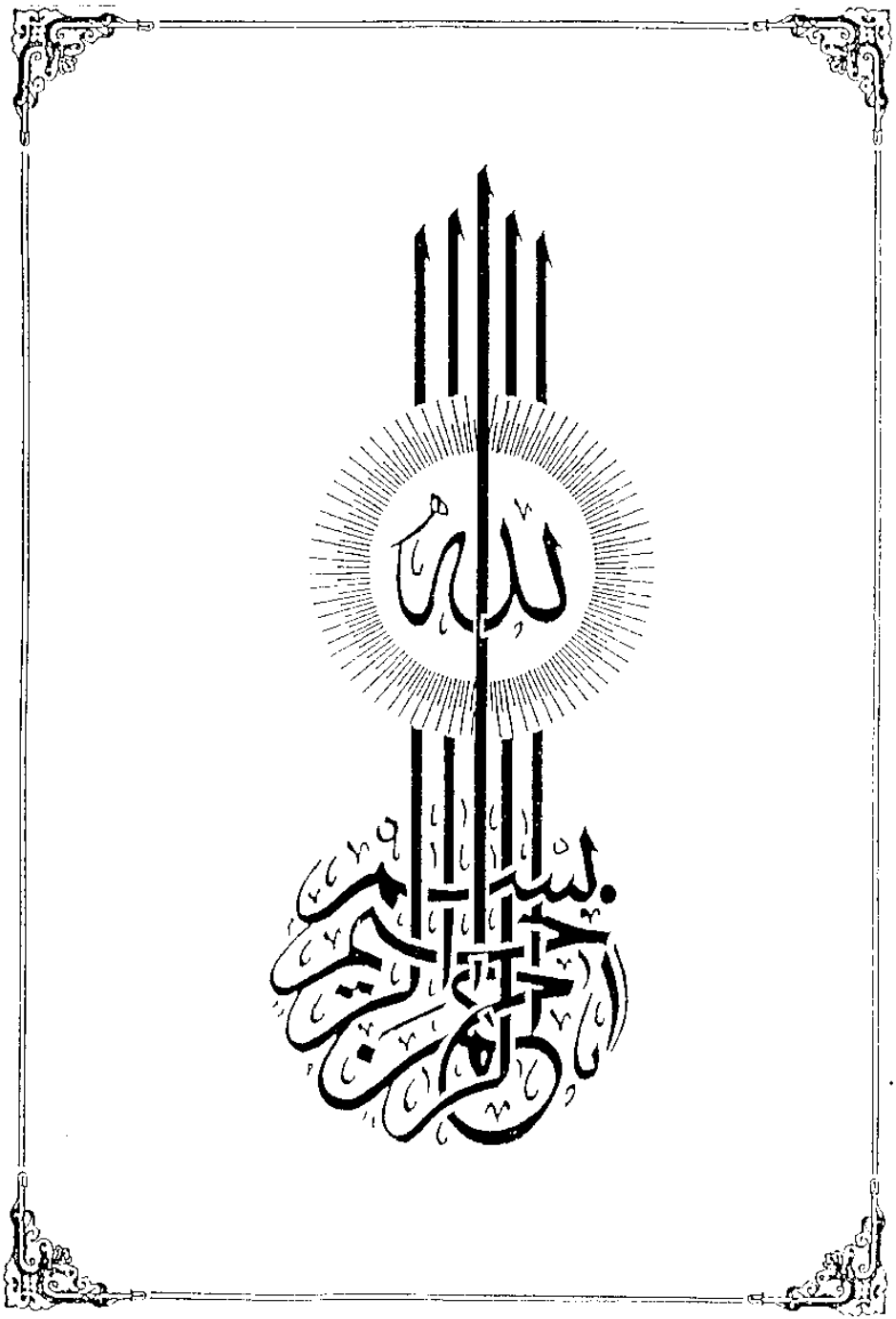

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CONTENTS

LIST OF ABBREVIATION

abs	: Antibodies
ACR	: American College of Rheumotology
ADCC	: Antibody dependent cellular cytotoxicity
Ag	: Antigen
AI	: Activity index
ANA	: Antinuclear antibody
Anti-sm antibody	: Anti Smith antibody
Anti-UHRNP	: Anti-uridine-1 ribonucleoprotein
APC	: Antigen presenting cell
APO-1	: Apoptosis-1
ARA	: American Rheumatism Association
Bcl	: B cell lymphoma
BUN	: Blood urea nitrogen
C	: Complement
CBC	: Complete blood count
CI	: Chronicity index
CIC	: Circulating immune compex
CR	: Complement receptor
DBP	: Diastolic blood pressure
DNA	: Deoxyribonuclic acid
DPGN	: Diffuse proliferative glomerulonephritis
ds	: double strand
EBV	: Epstein:Barr Virus
ELISA	: Enzyme -linked immunosorbent assay

EM	: Electron microscopy
ESR	: Erythrocyte sedimentation rate
ESRD	: End stage renal disease
FPGN	: Focal proliferative lupus Nephritis
g	: Gram
GBM	: Glomerular basement membrane
GFR	: Glomerular filtration rale
H	: Histone
h	: hour
HLA	: Human leucocytic antigen
HPF	: High power field
HS	: heparan sulphate
HSPG	: Heparan sulphate proteoglycan
IV	: Intravenous
ICs	: Immune complexes
Id	: Idiotypes
Ig	: Immunoglobulin
IL	: Interleukine
ICAM	:Intracellular adhesion molecule
Kg	: Kilogram
LE cell	: Lupus erythematosus cell
LM	: Light microscopy
LN	: Lupus nephritis
Mc	: Mesangial
mg	: Milligram
MHC	: Major histocompatibilty complex

MLN	: Membranous lupus nephritis
MPS	: Mononuclear phagocyte system
NHI	: National Institute of Health
NK	: Natural killer
NSAIDs	: Non steroidal anti-inflammatory drugs
ON	: Oligo nucleosome
P.T.T.	: Partial thromboplastin time
PAS	: Periodic acid schiff
PMNS	: Polymorphonuclear leucocytes.
PT	: Prothrombin time
RBCs	: Red blood cells
RNP	: Ribonucleoprotein
S.	: serum
SD	: Standard deviation
SLE	: Systemic lupus erythematosus
snRNPs	: Small nuclear ribonucleoproteins
ss	: Single strand
TBM	: Tubular basement membrane
Th	: T-helper
TIDs	: tubulointerstitial immune deposits
TNF	: Tumour necrosis factor
U/S	: Ultrasonography
USC	: University of Southern California
VCAM	: Vascular cell adhesion molecule
VH	: Heavy chains
VL	: Light chains

WBCs	: White blood cells
WHO	: World Health Organization
X	: Mean

INTRODUCTION AND AIM OF THE WORK

Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by immune dysregulation that results in the production of auto antibodies, generation of circulating immune complexes, and activation of complement system. The origin of auto antibody production is an area of intense research interest; the contributions of an antigen - driven process, primary B cell hyperresponsiveness, or impaired tolerance have been debated. A role in SLE for impaired processing of immune complexes due to decreased CR1 expression, impaired Fc receptor function, and inherited deficiencies of early complement components has also been indicated by experimental and human SLE data **(Belmont et al., 1996)**.

Specific symptoms referable to the kidney are not volunteered by the patients until there is advanced nephrotic syndrome or renal failure. The revised criteria for the classification of SLE recognizes proteinuria and the presence of casts as evidence of renal disease. In addition, the presence of hematuria, and/or pyuria in the absence of infection, and the detection of an elevated serum creatinine, have been recognized as evidence for clinical renal disease. It is important to appreciate that in order to identify the presence of renal disease, a urinalysis, as well as a serum creatinine have to be performed regularly. A renal biopsy may provide a more accurate documentation of renal disease. Most lupus patients manifest some abnormality on renal biopsy, although in some cases it is only possible to document it with special techniques, such as immunofluorescence or electron microscopy **(Gladman and Urowitz, 1994)**.

Lupus nephritis (LN) is characterized by multiple intermittent clinical flares of disease activity which often cause irreversible damage to the kidneys. Periods when there are no clinical signs of active glomerulonephritis may nonetheless be associated with progressive immunologic damage to the remaining nephrons. Therefore it would be advantageous to anticipate disease activity by serological tests to better adjust immunosuppressive therapy (**Mills, 1994**).