شبكة المعلومات الجامعية

التوثيق الإلكتروني والميكروفيلم

بسم الله الرحمن الرحيم

HANAA ALY

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جامعة عين شمس

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قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

HANAA ALY

Role of Antificolin II Antibody in Lupus Nephritis

Thesis

Submitted for Partial Fulfillment of Master Degree in **Internal Medicine**

By

Ibrahim Sami Ibrahim
M.B.B.Ch, M.SC
Cairo University

Supervised by

Prof. Dr. Mohamed Nazmy Farres
Professor of Internal Medicine and Clinical Immunology
Faculty of Medicine, Ain Shams University

Dr. Sylvia Talaat Kamal
Lecturer of Internal Medicine and Clinical Immunology, Faculty of Medicine, Ain Shams University

Dr. Fatma Abdelrahman Ahmed
Lecturer of Internal Medicine and Nephrology
Faculty of Medicine, Ain Shams University

Faculty of Medicine
Ain Shams University
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Ibrahim Sami Ibrahim Abdulaziz Sehsah

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List of Abbreviations

Abb.	Full term
ACEi	*Angiotensin-converting enzyme inhibitor*
ACR	*According to American College of Rheumatology*
AI	*Activity index*
ANA	*Antinuclear antibodies*
Anti- NCS	*Anti-nucleosome*
APO	*Apolipoprotein*
APOL1	*Apolipoprotein risk variants*
ARB	*Angiotensin receptor blocker*
BAFF	*B-cell activating factor*
BLC	*Blymphocyte chemoattractant*
BLyS	*B-lymphocyte stimulator protein*
CCL2	*Urinary MCP-1*
CI	*Chronicity index*
CKD	*Chronic kidney disease*
CL-K1	*Collectin-11*
CRHD	*Chronic rheumatic heart disease*
CXCL16	*Chemokine ligand 16*
ESI	*Electrospray ionization*
ESRD	*End-stage renal disease*
FcgR	*Fc receptors for IgG*
FCN2	*L-ficolin gene*
FDs	*Fibrinogen-like domains*
GBM	*Glomerular basement membrane*
GFR	*Glomerular filtration rate*
ICAM	*Intracellular adhesion molecule*
ICAM-1	*Intracellular adhesion molecule-1*
IFN- α	*Interferon- α*
ISN	*International Society of Nephrology*

List of Abbreviations (cont...)

Abb.	Full term
Kim-1	*Kidney injury molecule-1*
L-ficolin	*Ficolin-2*
LN	*Lupus nephritis*
MAGE-B2	*Melanoma associated antigen gene B2*
MASP's	*MBL/Ficolin associated serine proteases*
MBL	*Mannose–binding lectin*
MCP-1	*Monocyte chemoattractant protein-1*
MDRD	*Modification of Diet in Renal Disease*
M-ficolin	*Ficolin-1*
MIG	*Monokine induced by IFN-c*
miRNAs	*MicroRNAs*
MS	*Mass spectrometry*
NETS	*Neutrophil extracellular traps*
OPG	*Osteoprotegerin*
pDCs	*Plasmacytoid dendritic cells*
pSLE	*Pediatric SLE*
PTX3	*Pentraxin 3*
Q	*Quadrupole*
RAAS	*Renin-angiotensin-aldosterone system*
ROC	*Receiver operating characteristic curve*
RPS	*Renal Pathology Society*
SLE	*Systemic lupus erythematosus*
TOF	*Time of flight tandem*

INTRODUCTION

Systemic Lupus Erythematosus

SLE is a chronic autoimmune disease that can affect virtually any organ of the body in which different immunological occasions can prompt a comparative clinical picture, described by a wide scope of clinical manifestations and target organs with erratic flares and abatements that in the long run lead to permanent injury.

SLE can affect several organs and systems, including the joints, skin, brain, heart, lungs, blood vessels, and kidneys. ***(Colliard et al., 2018).***

It can be diagnosed based on a combination of clinical findings and laboratory work. According to American College of Rheumatology (ACR) presence of 4 out of the 11 clinical criteria for diagnosis of SLE yields a sensitivity and specificity of 85% and 95% respectively.

Auto antibodies circulating the body directed against self-antigen as ds DNA, nuclear antigen and several cytoplasmic components feature main histopathology of SLE ***(Ortega et al., 2010).***

Lupus nephritis

It is one of the most serious complications of SLE

affecting almost 66-90% of lupus patients with variable incidence and prevalence depending on studied population. It is higher in Asian and Africans than Caucasians 55%, 51% and 14% respectively.

LN develops early in the course of lupus activity leading to development of end-stage renal disease (ESRD) after 10 years of renal affection in up to 25% of patients; however, it may appear after several years in 5% of patients known as delayed lupus nephritis.

Auto antibodies and immune complexes with subsequent infiltration by inflammatory cells in renal tissue is the most likely pathogenesis of LN ***(Nisihara et al., 2013).***

Complement system

As part of innate immunity, complement system plays a dual role in pathogenesis of SLE. Genetic deficiency of complement factors is associated with the occurrence of SLE, however, massive complement activation during lupus activity is a major contributor to lupus inflammatory reactions.

There are three distinct pathways for complement system activation.

Binding of the pattern recognition molecule C1q to antibody-antigen complexes initiate classical pathway.

Slow, spontaneous hydrolysis of the central complement