Vascular Study Of Gastrointestinal Tract In Systemic Lupus Erythematosus Patients

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In Internal Medicine

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Abstract

Gastrointestinal symptoms are common in SLE patients, and more than half of them are caused by adverse reactions to medications and viral or bacterial infections. Patients and methods: 40 SLE patients were subjected to full clinical history and examination, kidney function, lipid profile, C 3, C4, ESR, CRP, urine analysis, 24 h urinary protein, stool analysis and culture, mesenteric , celiac , and carotid arteries doppler. In certain patients, when indicated, they underwent CT abdomen, upper and lower GIT endoscopies. 30 healthy controls were subjected to full clinical history and examination, mesenteric, celiac, carotid arteries doppler, stool analysis, ANA. Results; EDV of the mesenteric duplex is significant lower in SLE patients as compared to controls, prevalence of giardiasis in SLE patients was 30%. Biopsies of the bowel revealed 100% lymphocytic infiltration. Conclusion; increased vascular stiffness of mesenteric duplex, increased prevalence of giardiasis in SLE patients as compared to controls, all bowel biopsies revealed chronic lymphocytic infiltration.

Key wards:

GIT vasculitis, Systemic lupus erythematosus

IN THE NAME OF GOD

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Lists of tables

List of figures

List of pictures

Content

Abbreviations

Abd (In tables):	Abdomen
ACL:	Anticardiolipin
AECA:	Anti-endothelial cell antibodies
ANA:	Antinuclear antibody
ANCA:	Antineutrophilic cytoplasmic antibodies
Anti NCS:	Anti-nucleosome
AVN:	Avascular necrosis
AZA:	Azathioprine
C 3:	Complement 3
CD:	Cluster of differentiation
Ce:	Celiac
Cm/Sec:	Centimeter per second
CMI:	Chronic mesenteric ischemia
CRP:	C- reactive protein
CSF:	Cerebrospinal fluid
CT:	Computed tomography
CVD:	Cardiovascular disease
CYC:	Cyclophosphamide
DM:	Diabetes mellitus
DNA:	Deoxyribonucleic acid
E (In Tables):	Edema
EDV:	End diastolic velocity
ESR:	Erythrocyte sedimentation rate
F:	Female
Ga:	Gallium
GI:	Gastrointestinal

GIT:	Gastrointestinal tract
Gm:	Gram
H. pylori:	Helicobacter pylori
HB:	Hemoglobin
HM:	Hepatomegaly
HPF:	High power field
Ig:	Immunoglobulin
IL:	Interleukin
L%:	Lymphocyte percent
LA:	Lupus anticoagulant
Lc (In tables):	Left carotid
LMV:	Lupus mesenteric vasculitis
M:	Male
MD (In tables)	Mesenteric dopper
MHC:	Major histocompatibility
MMF:	Mycophenolate mofetil
N:	Neutrophil
No:	Number
Pic:	Picture
PLt:	Platelet
PSV:	Peak systolic velocity
R (in tables):	Raynaud`s
RBCs:	Red blood cells
Rc (In tables):	Right carotid
RI:	Resistivity index
RNA:	Ribonucleic acid
RNP:	Ribonucleic protein
RR:	Respiratory rate
RT:	Right

SLE:	Systemic lupus erythematosus
SLEDAI:	Systemic lupus disease activity index
SM:	Splenomegaly
SMA:	Superior mesenteric artery
TBM:	Tangible body macrophages
Tg:	Triglyceride
TNF α:	Tumor necrosis factor alpha
TTP:	Thrombotic thrombocytopenia purpura
U/S:	Ultrasound

Chapter I

Introduction

Systemic lupus erythematosus is an autoimmune disorder characterized by multisystem microvascular inflammation with the generation of autoantibodies **(Rahman et al., 2008)**, that affects virtually any organ in the body. The immunological dysregulation typical of SLE is characterized by polyclonal B-cell activation, production of pathogenic auto-antibodies, and an impaired cell-mediated immunity resulting from T-lymphocyte and antigen-presenting cell abnormalities **(Mok et al., 2003).** SLE is a chronic, recurrent, potentially fatal multisystem inflammatory disorder that can be difficult to diagnose **(Edworthy, 2001)**. SLE is a prototypic autoimmune disease characterized by the production of antibodies to components of the cell nucleus in association with a diverse array of clinical manifestations. The primary pathological findings in patients with SLE are those of inflammation, vasculitis, immune complex deposition, and vasculopathy **(Mok et al., 2003).**

Epidemiology:

The frequency of SLE varies by race and ethnicity with higher rates reported among black and hispanic people **(Balluz et al., 2001).** Although the prevalence of SLE is high in black persons in the United Kingdom, the disease is rarely reported among blacks who live in Africa **(symmons, 1995)**. SLE frequently starts in women of childbearing age, with a female to male ratio of 9:1 (**Nguyen et al., 2004)**, and the use of exogenous hormones has been associated with lupus onset and flares. The risk of SLE development in men is similar to that in prepubertal or postmenopausal women **(castenbader et al., 2007)**.

A familial predisposition to SLE has been identified **(Gray et al., 2000)**, the concordance rate for lupus is 25% among monozygotic twins and approximately 2% among dizygotic twins **(sullivan, 2000)**, these rates indicate that a genetic contribution is important, but it is not sufficient to cause the disease. Many genes that probably contribute to lupus have been identified by means of whole genome scans from families in which multiple members have lupus **(Namjou et al., 2007).**

Etiology:

Though certain susceptibility factors and triggering events like the hormonal milieu or exposure to ultraviolet radiation have been identified, the exact etiology of SLE remains elusive. A strong genetic basis has to be assumed, and several genetic polymorphisms have been suspected to contribute to SLE susceptibility **(Prokunina et al., 2004).**

There are mechanisms by which lupus is thought to develop: genetic, predisposition, environmental triggers, sex hormones, hypothalamo-pituitary-adrenal axis and drug reaction (drug induced lupus).

I)Genetic predisposition:

The concordance of SLE in identical twins, the increase in frequency of SLE among first degree relatives, and the increased risk of developing the disease in siblings of SLE patients reflects a polygenic inheritance of the disease. In a small proportion of patients < 5%, a single gene may be responsible **(Walport et al., 1998)**. However most of the SLE patients require multiple genes. It is estimated that at least four susceptibility genes are needed for the development of the disease **(Mok et al., 2003).**

Genes of the major histocompatibility complex (MHC), particularly HLA A1, B8 and DR3 have been linked to lupus. The response of a