Renal Manifestations Of Rheumatic Diseases

Essay
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أعراض إعتلال الكلى فى الأمراض الروماتيزمية

مقـــــــــالة

توطئة للحصول على درجة الماجستير

في الباطنة العامة

مقدمة من

أبو النجا عبد الراضي عبد الرحيم

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الأستاذ الدكتور

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AA	Aquired systemic amyloidosis
AASV	ANCA associated systemic vasculitides
ACE	Angiotensin converting enzyme
ACL	Anticardiolipin antibodies
ACR	American college of rheumatology
ANA	Antinuclear antibodies
ANA	Anti-nuclear antibody
ANCA	Anti neutrophil cytoplasmic antibodies
APSGN	Acute post streptococcal glomerulonephritis
ARB	Angiotensin receptor blockers
AS	Ankylosing spondylitis
ASCT	Autologous stem cell transplantation
ATG	Anti-thymocyte globulin
AZA	Azathioprine
BD	Behcet's disease

C	Complement
CSA	Cyclosporine a
CSS	Churg–strauss syndrome
CYC	Cyclophosphamide
DNA	Deoxy ribo neuclic acid
DRTA	Distal renal tubular acidosis
DS DNA	Double stranded dna
DSC	Doubling of serum creatinine)
ELISA	Enzyme linked immunosorbent assay
EM	Electron microscopy
ESRD	End stage renal disease
GBM	Glomerular basement membrane
GC	Glucocorticoids
GIN	Granulomatous interstitial nephritis
GN	Glomerulonephritis
HBV	Hepatitis b virus
HCV	Hepatitis c virus
HLA	Human leucocytic antigen

HSC	Hematopoietic stem cells
HSP	Henoch-schonlein purpura
IF	Immunofluorescence microscopy
IFN	Interferon
IG	Immunoglobulins
IGA	Immunoglobulin a
IgAN	IgA nephropathy
IgG	Immunoglobulin g
IgM	Immunoglobulin m
IL	Interleukine
INCGN	Idiopathic necrotizing and crescentic glomerulonephritis
IVIG	Intravenous immunoglobulin
LA	Lupus anticoagulant
LM	Light microscopy
LN	Lupus nephritis
MHC	Major histocompatibility complex
MMF	Mycophenolate mofetil
MP	Methylprednisolone

MPA	Microscopic polyangiitis
MPGN	Membranoproliferative glomerulonephritis
MPO	Myeloperoxidase
NDI	Nephrogenic diabetes insipidus
NIH	National institutes of health
NSAID	Non-steroidal anti-inflammatory drugs
PAN	Polyarteritis nodosa
PE	Plasma exchange
PMN	Polymorphonuclear neutrophl
PR3	Particular proteinase 3
PSS	Primary sjogren's syndrome
RA	Rheumatoid arthritis
RF	Rheumatoid factor
RPGN	Rapidly progressive glomerulonephritis
RTA	Renal tubular acidosis
RTX	Rituximab
SAA	Serum amyloid a protein
SAP	Serum amyloid p component

SLE	Systemic lupus erythematosus
SLEDAI	Systemic lupus erythematosus disease activity index
SRC	Scleroderma renal crisis
SS	Sjogren's syndrome
SSC	Systemic sclerosis
TNF	Tumor necrosis factor
TTP	Thrombotic thrombocytopenic purpura
UK	United kingdom
UV	Ultraviolet
WG	Wegener's granulomatosis

Introduction

Rheumatic diseases are a group of multisystem chronic inflammatory disorders that involve multiple body systems; not only the musculoskeletal, and therefore exhibit a wide spectrum of clinical manifestations. In the absence of reliable serological markers, accurate clinical tools are required to assess disease activity and damage for treatment decisions**.(Cush et al., 1999) .**

The synovial membrane is the principal site of inflammation in inflammatory arthritides. Synovitis is characterized pathologically by neovascularization; infiltration of the synovium with lymphocytes, plasma cells, and macrophages; and synovial lining cell hyperplasia. These cause synovial proliferation, recognized clinically by warmth, tenderness, and a boggy consistency of the soft tissues overlying the involved joint. The inflamed synovium may infiltrate and erode intra-articular bone and cartilage. There are many extra-articular manifestations of most rheumatic diseases; that may accompany, precede or follow the onset of these disorders, the most common and serious is renal affection. **(Krishnan et al.,2006)** .

A variety of renal disorders can be seen in patients with rheumatoid arthritis, due both to the underlying disease and to drugs used to treat the inflammatory process. The most common are membranous nephropathy, secondary amyloidosis, a focal, mesangial proliferative glomerulonephritis, rheumatoid vasculitis, and analgesic nephropathy. Other disorders, such as IgA nephropathy and minimal change disease

have also been reported in patients with rheumatoid arthritis, although this may represent only superimposed disease. **(Stokes.,2005).**

Scleroderma (SSc) is a multisystem connective tissue disease of unknown etiology that occurs more commonly in women, follows a chronic course, and is associated with substantial morbidity and mortality. The first descriptions of acute renal failure associated with systemic sclerosis led to an appreciation of the important link between this prototypic fibrotic disease and vascular injury . **(Christopher et al., 2008)**

Renal involvement remains a major complication of scleroderma. Long-term outcome after renal crisis remains poor despite the use of angiotensin-converting enzyme inhibitors (ACEI). There is no evidence at present to support the use of ACEI prophylactically. The mechanisms and significance of chronic renal impairment in scleroderma need to be better defined**.(Penn et al., 2008).**

There is now a much better understanding of the risk factors and outcome for Scleroderma Renal Crisis (SRC) based on recently reported cohorts of patients. These confirm that although early outcome has improved markedly following the introduction of ACEIs, there is still a high morbidity and mortality in scleroderma patients who have a renal crisis **.(Penn et al., 2008).**

Systemic lupus erythematosus (SLE) is a systemic multiorgan autoimmune disease of unknown cause. The unique nature of the disease and the diversity of the clinical presentations together with the unpredictable course, have lead to the fact that every SLE

patient should be treated and followed up individually and should have his own treatment plan **. (Tarr et al.,2007).**

After a multivariable analysis, only high systolic blood pressure, cutaneous vasculitis, hemoglobin < 10 mg/dl and serum creatinine > 1.3 mg/dl remained as statistically significant risk factors for developing lupus nephritis. **(Satirapoj et al.,2008).**

The vasculitis syndromes comprise a group of inflammatory disorders of presumed autoimmune origin characterized by inflammation and necrosis of blood vessels frequently in combination with granuloma formation **.(Badakere et al.,2002).**

Since the kidney is highly vascularized, vasculitis frequently occurs in the kidney. The histopathological findings of ANCA-related vasculitis in the kidney are show a variety of lesions, of which crescentic and/or focal necrotizing glomerulonephritis as well as small vessel arteritis are the most prominent.**(Arnnette et al.,1994).**

Early recognition of the disease facilitates prompt treatment and results in better prognosis; however, the use of basic laboratory indicators, such as hematuria, proteinuria, or the serum creatinine level, is considerably limited in facilitating the prediction of the affected site of vasculitis **.(Arnnette et al.,1994).**

Gouty arthritis and renal problems appear to be the most frequent complication of hyperuricemia. 20% to 40% of patients with gout have albuminuria, which is