شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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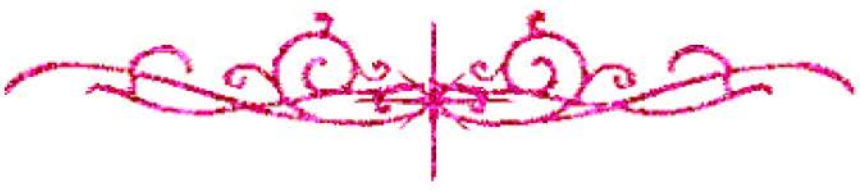

STUDY OF SOME MARKERS OF PROGRESSION IN SCHISTOSOMAL NEPHROPATHY.

THESIS

SUBMITTED TO THE FACULTY OF MEDICINE,

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LIST OF ABBREVIATIONS

3'amino-9'ethyl-carbimazole: *AEC*

3,3'diaminobenzidine terahydrochloride: *DAB*

Alanine amino transferase: *ALT*

Alpha smooth muscle actin: *α SMA*

Antibody diluting buffer: *ADB*

Aspartate amino transferase: *AST*

Avidin biotin peroxidase complex: *ABC*

Basic fibroblast growth factor: *b-FGF*

Bovine serum albumin: *BSA*

Circulating anodic antigen: *CAA*

Circulating cathodic antigen: *CCA*

Confocal: *con*

Connective tissue mast cells: *CTMC*

Diabetic nephropathy: *DN*

End stage renal disease: *ESRD*

Extracellular matrix: *ECM*

ε(γ-glutamyl-lysine) cross link: *C/L*

Fluorescine cadaverine: *FITC*

Focal and segmental glomerulosclerosis: *FSGS*

Frozen: *F*

Glomerular: *G*

Glomerulosclerosis: *GS*

Glomerular vimentin: *GV*

Granulocyte monocyte colony stimulating factor: *GM-CSF*

Hepatitis C virus: *HCV*

Interferon-γ: *IFN-γ*

Interstitial: *Int*

Interstitial cellular infiltration: *ICI*

Interstitial fibrosis: *IF*

Interstitial vimentin: *IV*

Mast cells: *MC*

Mast cells tryptase: *MCT*

Mesangiocapillary glomerulonephritis: *MCGN*

Membranoproliferative glomerulonephritis: *MPGN*

Membranous glomerulonephritis: *MGN*

Mesangial cellular proliferation: *MCP*

Mucosal mast cells: *MMC*

Mouse mast cell protease: *mMCP*

Paraffin: *P*

Phosphate buffered saline: *PBS*

Plasminogen activator inhibitors: *PAIs*

Platelet derived growth factor: *PDGF*

Reversed transcription polymerase chain reaction: *RT-PCR*

Schistosomal nephropathy: *SN*

Stem cell factor: *SCF*

Tissue inhibitors of matrix metalloproteinases: *TIMPs*

Tissue transglutaminase: *tTg*

Transforming growth factor-β: *TGF-β*

Tumor necrosis factor-α :*TNF-α*

Vimentin: *V*

INTRODUCTION

In Egypt, the incidence of schistosomiasis in one village in the Nile delta was 74 %, with renal affection in 12-15% of cases.[1]

Tissue fibrosis is a series of dynamic interactive processes to effect normal repair of injured tissue. These processes follow a specific time sequence and its phases are not mutually exclusive, but overlap in time. [2] In higher vertebrates, tissue loss that disrupts normal architecture precludes tissue regeneration; lost tissues are replaced by fibrous tissue.[3]

This process of fibrosis should usually end physiologically in an adaptive response and replacement of lost tissue. However, this process may result in a maladaptive response resulting in pathological states. These maladaptive responses seriously impair normal function of the affected tissues.[4]

Renal fibrosis is characterized by a progressive loss of specialized renal parenchymal cells with a disturbance of the delicate balance regulating ECM production towards the accumulation of excessive fibrous tissue. [5]

Investigation of the cellular and enzymatic factors responsible for this excessive fibrosis is in the spotlight.

A better understanding of the mechanisms involved in the process of renal fibrosis, the final common pathway of all advanced forms of renal pathology, is a valuable aid in the development of strategies and new methods of hampering this progressive deterioration of renal structure and function.

REVIEW OF LITERATURE

SCHISTOSOMIASIS:

I- Epidemiology

Schistosomiasis is considered as a leading cause of morbidity in endemic areas. It is currently estimated to affect in excess of 200 million individuals worldwide while 500 million are at risk of infection in over 74 countries in Africa, Asia and Latin America.[6]

Schistosomes are bisexual trematodes that parasitize the venous system. Seven species affect man as a definitive host. [7]

II- Life-cycle

Infection occurs by contact with contaminated fresh water containing the infective phase, the cercaria, which penetrates the skin and mucous membranes, after which it is transformed into "skin-stage schistosomulae'. They stay in the dermis for 1 to 3 days during which they change their surface structure and antigenicity and migrate by way of the lymphatics to the bloodstream where they become trapped in the pulmonary capillaries and are then termed 'lung-stage schistosomulae'. Afterwards they escape to the hepatic sinusoids where they differentiate into males and females. The worms migrate to their eventual habitat, the mesenteric veins for all human pathogenic schistosomes except S. haematobium that resides in the perivesical venous plexus. One or more couples live for some 3 to 8 years, but prolonged survival for up to 30 years has been reported. The worms live in almost continuous copulation. The female leaves the male's "gynecophoric canal" for a few hours every day, travels against the bloodstream to reach the mucosa of the colon, rectum, or lower urinary tract where it lays the eggs. The process of egg laying starts 8 to 10