hossam maghraby

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

hossam maghraby

شبكة المعلومات الجامعية

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

hossam maghraby

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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

hossam maghraby

شبكة المعلومات الجامعية

بعض الوثائق الأصلية تالفة

hossam maghraby

شبكة المعلومات الجامعية

بالرسالة صفحات لم ترد بالأصل

Study of Plasma Amino Acids Changes in Elderly Patients with Chronic Renal Failure

Thesis

Submitted to the Faculty of Medicine
University of Alexandria
In partial fulfillment of the requirement for

Master Degree
Of
Internal Medicine

By
Tharwat Khamis Mohamed Salamona
MBBCh, Alex.
Faculty of Medicine
Alexandria University
2004

SUPERVISORS

Prof.Dr.Mohamed Ahmed Mehanna

Professor of Internal Medicine

Faculty of Medicine

University of Alexandria

Dr.Ahmed Gaber Adam

Assistant Professor of Internal Medicine

Faculty of medicine

University of Alexandria

Dr.Taisser Mohamed Mostafa

Lecturer of Clinical Pathology

Faculty of Medicine

University of Alexandria

ACKNOWLEGEMENT

Thanks to ***Allah***, for every thing done in this work.

It is great honor to express my deepest gratitude and cordial appreciation to prof. ***Dr: Mohamed Ahmed Mehanna,*** *Professor of Internal medicine, Faculty of Medicine, University of Alexandria for his meticulous supervision, constant guidance and encouragement.*

I also would like to express my great appreciation and thanks to ***Dr: Ahmed Gaber Adam,*** *Assistant Professor of Internal Medicine, Faculty of Medicine, University of Alexandria for his valuable suggestion, encouragement and supervision all over the course of this work.*

special thanks to ***Dr: Taisser Mohamed Mostafa,*** *lecturer of Clinical Pathology, Faculty of medicine, University of Alexandria for her constant supervision and encouragement.*

My great appreciation is given to all those who shared either practically or morally in the creation of this work.

CONTENTS

INTRODUCTION

INTRODUCTION

CHRONIC RENAL FAILURE (CRF)

The syndrome of chronic renal failure is difficult to be defined as it has many different aspects, which should be included in the definition. CRF can be defined as a chronic reduction of glomerular filtration rate; i.e. a diminution in creatinine clearance and corresponding increase in serum creatinine. This definition considers only the excretory function of the kidney and its glomerular component in particular, while it underestimates the effect in tubular and endocrine function of the kidney, whose course usually parallels the decrease in glomerular filtration rate (GFR).[1]

Another definition is persistent impairment of both glomerular and tubular function; of gradual onset, and of such severity that the kidneys are no longer able to keep the internal environment normal [2]. End stage chronic renal insufficiency is usually defined as a reduction in glomerular filtration rate to under 5-15 ml/minute. This corresponds to less than 5% of normal nephron function. In end stage failure, glomerular filtration can be estimated from the mean of urea and creatinine clearance in the 24-hour-urine collection [3].

Chronic renal failure is a complex syndrome consisting of anemia, oesteodystrophy, neuropathy, and acidosis and is frequently accompanied by hypertension, susceptibility to infection and generalized deterioration in organ function [4].

PATHOGENESIS OF UREMIC SYNDROME:

It is mainly attributed to retention of nitrogenous waste products, to excessive accumulation of several peptide hormones as consequence of the loss of renal function or as a compensatory mechanism and to deficiency of essential compound not produced in uremia e.g. erythropoietin and 1,25 dihydrocholecalciferol. So it is very likely that the pathogenesis of uremia is multifactorial in nature [5].

CAUSES:

In United States as reported by the united states Renal Data System Annual Report 1997, the most common disease causing end stage renal disease (ESRD) are diabetes mellitus (33.2%), hypertension (24%) and glomerulonephritis (17.2%); however reflux pyelonephritis, renal hypoplasia, dysplasia, congenital cystic disease and renal tumors may also lead to ESRD [6]. Similar data were seen in Egypt (NKF-Data, personal communications). However, glomerulonephritis are seen more in the developing countries. These data should be used either to

establish or to exclude the most common causes of CRF in any evaluation of patient with chronic renal failure [6].

APPROACH TO PATIENT:

In the evaluation of patients with an elevated serum urea and creatinine levels, it is important to establish the following:

1. The acute or chronic nature of the renal function impairment.
2. The causes of renal dysfunction.
3. The presence of superimposed reversible factor.

First, establishing the presence of CRF will influence the long-term survival of the residual renal function

Second; identification of a specific pathologic process will influence short and long term therapeutic intervention. Aggressive treatment of the primary pathologic process as well as the metabolic derangement of CRF will postpone to some extent the progression to ESRD.

Lastly, the identification and correction of any secondary reversible factor contributing to the renal dysfunction may quickly restore a level of renal function compatible with conservative management and will prevent or modify renal damage from a secondary pathologic process [6].

After the presence of CRF is established, the degree of renal dysfunction must be defined in order to guide further intervention. The degree of intervention may range from aggressive treatment of the primary disease and coexisting hypertension in mild renal insufficiency to conservative follow up and preparation of patients for dialysis in those with severe renal insufficiency[7]. The severity of renal dysfunction is assessed through the recognition of advanced clinical features of CRF (the uremic syndrome) and through the quantitation of the remaining glomerular filtration rate [7, 8].

MONITORING THE PAROGRESSION OF CHRONIC RENAL FAILURE:

As the renal function declines, the deteriorated metabolic disorders associated with CRF will result in the following clinical and laboratory manifestations:

Symptoms:[8]

-General:	fatigue, weakness, lethargy.
-Skin:	itching, easy bruising, skin discoloration, pallor or frost
-Cardiovascular:	dyspnea, orthopnea, edema or chest pain.
-Gastrointestinal:	anorexia, nausea, vomiting, early satiety or hiccups.

-Neuromuscular:	decreased ability to concentrate, restlessness, parathesia, muscle cramps and/or twitching.

Signs: [8, 9]

-Skin:	pallor, hyperpigmentation, hyperkeratosis and ecchymosis
-Oral:	oral ulcer and uremic breath.
-Cardiovascular:	hypertension with its different grades, ejection systolic murmur, edema, pericardial friction rub.
-Neuromuscular:	sensory/motor peripheral neuropathy, drowsiness, mental confusion seizures and may be coma.

Laboratory findings:[6]

-Elevated blood urea and serum creatinine.

-Metabolic acidosis.

-Anemia most commonly normochromic normocytic.

-Proteinuria.

-Granular casts in urine analysis.