بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــسـم ﴾

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لم ترد بالأصـــل

Study of the Role of Somatostatin and Insulin in Haemodialysis-induced Hypoglycaemia in Non-diabetic Patients with Chronic renal failure.

Thesis

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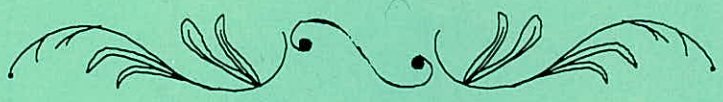
ABBREVIATIONS

CRF	:	Chronic renal failure	MCR	:	Mean catabolic rate
ESRD	:	End stage renal disease	PP	:	Pancreatic polypeptide
VLDL	:	Very low density lipoproteins	SRIF	:	Somatostatin release inhibiting factor
HDL	:	High density lipoproteins	VIP	:	Vasoctive intestinal polypeptide
HD	:	Haemodialysis	TRH	:	Thyrotropin releasing hormone
PAN	:	Polyacrylonitrile	TSH	:	Thyroid stimulating hormone
PS	:	Polysulphone	GRH	:	Growth release hormone
PMMA	:	Polymethylmethacrylate	BDA	:	British Diabetic Association
EEG	:	Electroencephalogram	OGTT	:	Oral glucose tolerance test
ECG	:	Eloctrocardiogram	NSB	:	Non specific binding
GFR	:	Glomerular filtration rate	IRMA	:	Immunorodiometric assay



Introduction

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Chronic Renal Failure (CRF)

CRF is a syndrome complex consisting of anaemia, osteodystrophy, neuropathy, acidosis and is frequently accompanied by hypertension, susceptibility to infection and generalized deterioration in organ function.[1] It may results from any progressive destructive condition affecting both kidneys.[2]

Renal functional deterioration may be described in successive stages as follows: [3]

1. Diminished renal reserve: kidney function as a whole is mildly or modestly reduced, but the excretory and regulatory functions are insufficiently intact to maintain a normal internal environment. The patient has no symptoms.

2. Renal insufficiency: at this stage some evidences of impaired capacity to maintain the internal environment appear. There tends to be mild azotemia, impaired concentrating ability, and some anaemia. However, these abnormalities are minimal untill the organism is stressed by dehydration, infection, heart failure, and so on.

3. Renal failure: kidney function has deteriorated to the point of chronic and persistent abnormalities in the internal environment, including azotemia, isothenuria and nocturea, metabolic acidosis, hypocalcaemia, and hyperphosphataemia. Hyponatraemia and hyperkalaemia are common.

4.The uremic syndrome: a constellation of clinical signs and symptoms appear in the patients with CRF, especially involving the gastrointestinal tract, cardiovascular system and nervous system.

Pathophysiology of CRF:

I. Water, electrolyte, and acid base metabolism in uremia:

1) Potassium:

In advanced CRF, the serum potassium concentration tend to be higher than normal, even though body stores of potassium may be reduced.[4]

2) Sodium:

The kidney has a remarkable ability to maintain total body sodium within normal limits until the end stages of CRF.[4]

3) Acid-base balance:

The kidney normally regulates blood pH within narrow limits by reabsorption (proximal tubule) and regeneration (distal tubule) of bicarbonate.[4] Metabolic acidosis develops when exogenous intake and endogenous production of acid exceeds and net acid secretion.[5]

4) Calcium:

The total serum calcium concentration in CRF is lower than normal. Patients with CRF tolerate hypocalcaemia quite well and tetany is uncommon.[4]

5) Phosphate:

Serum phosphate concentration is higher than normal. The retained phosphate is of major pathogenic importance in the development of secondary hyperparathyroidism in CRF.[6]

6) Magnesium:

CRF patients tend to have modest elevation in serum magnesium concentration.[4]

II. Hematopoietic system in uremia:

1) Anaemia: normochromic normocytic anaemia.[7]

2) Bleeding: usually from capillaries and is due to abnormal platelet function.[7]

3) Leucocyte dysfunction: although the granulocyte count is usually normal, some patients have a tendency toward granulocytopenia or lymphopenia.[4]

III. Cardiovascular system in uremia: [8]

1) Pericarditis.

2) Uremic cardiomyopathy.

3) Atherosclerosis.

4) Hypertension.

5) Arrhythmias.

IV. Uremic osteodystrophy:

Renal osteodystrophy is a complex disorder with several pathogenetic features. The most common component is osteitis fibrosa. Another important component is osteomalacia. Osteoporosis is also common in CRF.[9]

V. Nervous system in uremia: [7]

a) Higher mental functions: poor concentration, apathy, insomnia, irritability, tremors, astrexis, myoclonus and even seizures.

b) Peripheral nervous system: uremic neuropathy is a frequent complication of terminal uremia. End stage renal disease (ESRD) may result in dysfunction of the autonomic nervous system. Clinical manifestations may include abnormalities in such as gastrointestinal tract motility, gastro paresis or diarrhea and labile blood pressure.

VI. Myopathy.

Muscle weakness and wasting develop slowly but are common in CRF.[4]

VII.Endocrine and metabolic disorders:

a) Renal disease is associated with low or absent levels of erythropoietin and vitamin D.[8]

b) Pituitary, thyroid and adrenal function is relatively normal in CRF.[8]

c) Carbohydrates:

Several factors regulating carbohydrate metabolism are altered in association with renal failure. Gluconeogenesis and insulin metabolism