RENAL AFFECTION IN SYSTEMIC DISEASES

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By

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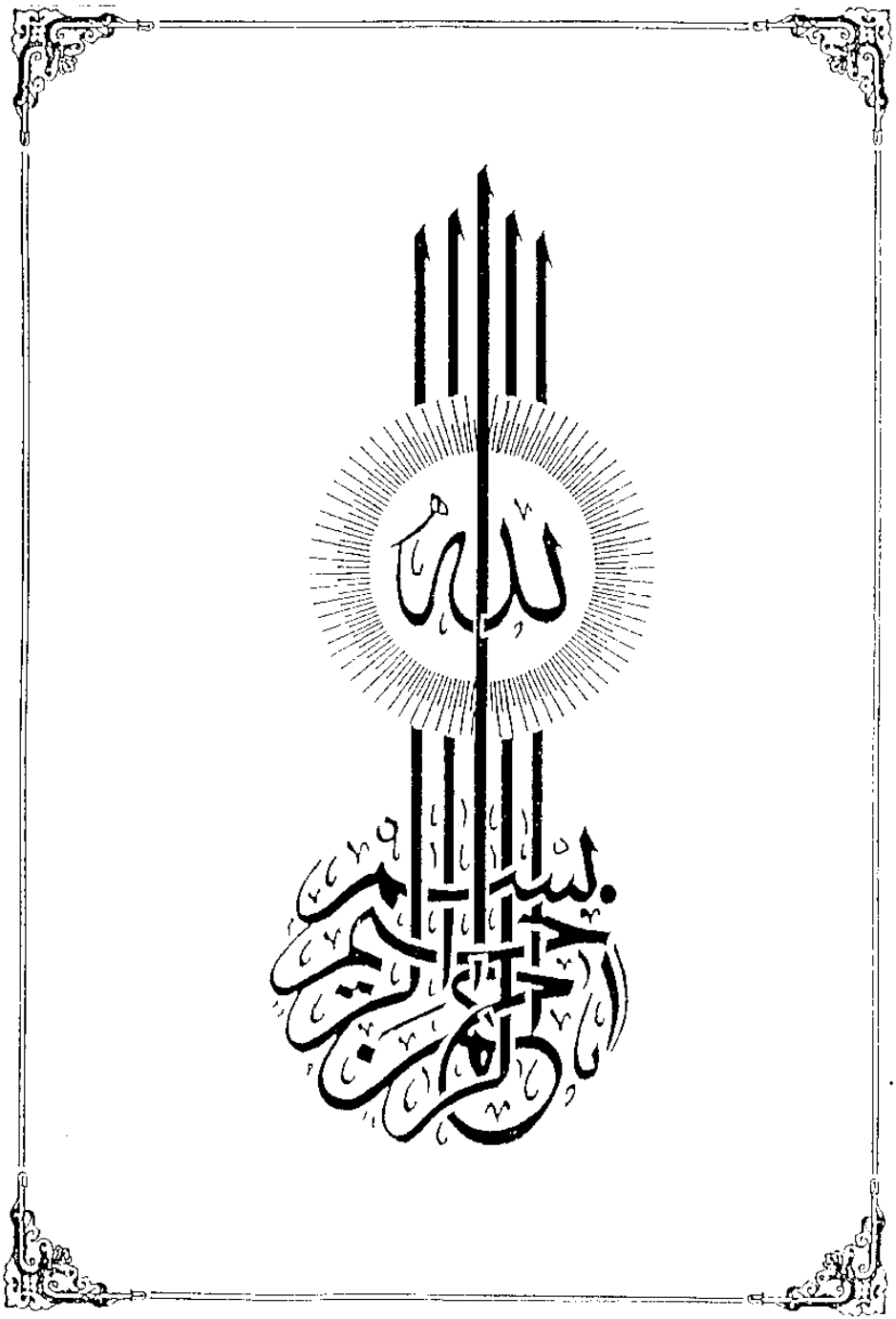

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INTRODUCTION

The kidney is the organ mainly concerned with keeping internal envirement of the body in a balanced and stable state. Disturbance of its function may result in a serious threat to patient's life.

The renal affection may be the cause of death in many systemic diseases with multiple organ involvement.In this review we are going to discuss the different various systemic diseases on the kidney. The review includes the mechanisms of renal involvement, the pathology and the clinical manifestations.

Systemic diseases can affect the kidney in different ways and by various mechanisms.In diabetes mellitus glomerulonephritis ,papillary necrosis,glycogen necrosis and bacteruria can occur .We referred in this review to " emphysematous pyelonephritis" which is a new term in regard to bacteruria. In this condition , which is encountered in diabetics and carries a high mortality rate , there is suppurative infection of the renal parenchyma with production of gas through fermentation.

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The kidney is also affected in many hepatic disorders such as Wilson's disease ,chronic active hepatitis and liver cell failure . In this review we emphasized the role of "endotoxins" in the pathogenesis of hepato-renal syndrome .About 50% of patients with ascites and liver cirrhosis have endotoxins. These are lipopolyaccharides derived from gram negative microbes and have been shown to produce several systemic effects including fever ,hypotension and haemorrhagic manifestations of the gastro-intestinal tract.

In normal persons the endotoxins are absorbed from the gut into the portal venous blood and are rapidly phagocytosed by Kupffer cells.This mechanism is impaired in liver diseases and endotoxins may reach the general circulation and produce their systemic effects.

Again ,the kidneyscan be affected in sarcoidosis by direct granulomatous involvement of the parenchyma,nephrocalcinosis ,renal stone formation and finally by causing glomerulonephritis.

One of the important systemic diseases affecting the kidney is gout.It affects the kidney through deposition of urate crystals either in renal parenchyma ,in the tubules or in renal pelvis. The end result is renal insufficiency and renal failure.

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Extra - renal malignancies can affect the kidneys through amyloidosis ,hyper-kalaemia,hyper-calcaemia ,hypercalcaemia hyperuricaemia,dysproteinaemia and direct involvement of the renal system.

Leprosy affects the kidneys [illegible] glomerulonephritis,amyloidosis and renal tubular dysfunction.

Collagen diseases can also [illegible] systemic lupus erythematosus affects the [illegible] causing glomerulonephritis , the tubules causing [illegible] atrophy and also causing oedema of the interstitium.
High incidence of renal [illegible] in the [illegible] rheumatoid arthritis.

Different [illegible] modalities [illegible] renal [illegible] may directly affect the kidneys [illegible] high [illegible].

[illegible] of the [illegible] polyuria [illegible] nephritis [illegible].

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The kidney is also affected in cardiac failure We are going to discuss the disturbance of kidney function in heart failure with particular emphasis on the disturbance in serum electrolytes ,renal haemodynamics ,and in the role of the kidney in regulation of effective blood volume.

Renal affection in hypertension ,Goodpasture syndrome,Schon - lein_Henoch purpura ,thrombotic -thrombocytopenic purpura,haemolytic uraemic syndrome and scleroderma are other examples that indicate the frequency with which the kidneys are involved in a way or another in nearly all systemic disorders.

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C H A P T E R I

THE KIDNEYS AND METABOLIC ABNORMALITIES.

I THE KIDNEYS IN DIABETES

II RENAL AFFECTION IN HYPERURICAEMIA

III THE KIDNEYS AND PARTIAL LIPODYSTROPHY

IV THE KIDNEY AND AMYLOIDOSIS

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THE KIDNEYS IN DIABETES

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THE KIDNEYS IN DIABETES

Renal affection in diabetes mellitus reveals itself as glycogen nephrosis, papillary necrosis and diabetic glomerulopathy.The most important lesion,we are going to discuss is diabetic glomerulopathy.

We can divide the changes that occur in the kidney according to the duration of diabetes into:

A) Early changes " short term diabetes ".

This corresponds to changes in the kidneys in diabetes of 1-6 years duration .This is reflected as an increase in the kidney size and weight by about 20%.

Osterby & Gunderson (1975) studied kidney biopsies from recent diabetics. They suggested that the mean glomerular volume and volume of capillary lumen per glomerulus were increased.The extra cellular matrial was also increased.

Rossa & Goldman (1971) have reported increased kidney weight few days after development of streptozotocin induced diabetes in rats. Therein also rise ofprotein synthesis and cellular hypertrophy. This is proved by the increase in D.N.A. as well as increase in R.N.A. / D.N.A. ratio (Sonn-Hansen 1976).With increase duration of diabetes mellitus the basement membrane of the glomeruli becomes thickened leading to total occlusion of many glomeruli.

As regards renal function in this early stage of diabetes,the glomerular filtration rate is high and persists high for many years in diabetic patients (Ditzel and Schwartz 1967).

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As regards to protein excretion in urine in early stage of diabetes, many studies have been donein this aspect,they showed that there is no proteinuria as indicated by the usual clinical tests as precipitate tests , but with increased duration of diabetes, there is slight proteinuria which increases by time. It is of interest to note that the amount of albumin loss in urine is concomitant with thickening of basement membrane . Mogensen(1976).

B) Late changes " Long term diabetes ".

Renal function in long term diabetes shows marked deterioration i.e low glomerular filteration rate and when this stage is reached a stage of constant proteinuria takes place.

We have to speak in brief about the proteinuria in diabetes,there are two stages,one of them is the intermittent proteinuria,the urine contains for example 2 gm/l at the first visit ,no protein is detected one month later and 4 gm/l in the following month and so on.

In the stage of permenant proteinuria,the protein excretion may remain moderate for many years,it increases year after year until in some patients ,high values are detected(10-15 g/l).

As regards to the blood pressure,it is generally normal ,however in long term diabetes in the stage of constant proteinuria,both systolic and diastolic blood pressure are slightly elevated.

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In the terminal stages of diabetes ,severe arterial hypertension may be seen in the patient with multiple signs and symptoms of diabetic angiopathy.

Renal biopsies from diabetics prove that the main lesion occur in the glomeruli and vascular structure in the form of nodular intracapillary glomerular sclerosis&sub-intimal hyalinization of both afferent and efferent arterioles.Capillary loops contain fibrin.

Bacteruria & pyelonephritis

Acute and chronic urinary tract infection and signs of pyelonephritis are more common in diabetics more than in non diabetics. Many studies have been done on "infection and diabetes ".

Osterly etal (1964) studied the prevalence of bacteruria in large groups of diabetics,the prevalence was about 5-18.5% in males and 3.7 - 7.9% in females.

Spagola (1978) introduced a new term as regard to the infection in diabetics,that is emphysematous pyelonephritis which is suppurative infection of renal parenchyma characterized by production of gas by bacteria through fermentation.The most common organism is escherichia coli.

This type of infection carries high mortality rate.

Papillary necrosis

Papillary necrosis is a pathological and clinical condition that occur with urinarytract infection and its incidence is more in diabetics.

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It consists of more or less complete necrosis of one or more papillary apices.

The clinical picture consists of colicky pain due to choking of the ureters with apices of the papillae,the disease is very often fatal.

Edmondson etal 1947 found glomerular sclerosis in over 50% of diabetic kidney with papillary necrosis.

The pathogensis of papillary necrosis is unknown but infection surely plays an important role.

Glycogen nephrosis.

This specific abnormality is found in diabetic patients ,who die from keto acidosis .

The tubular renal cells ,are filled with glycogen and the electron microscope reveals that glycogen accumulates in the cystoplasm pushing the granules to the periphery of the cells ,while the nucleus is normal.

Many studies are taking place now to see if the accumulation of glycogen in renal tubules with severe keto-acidosis is the cause of death in these patients.

Before we leave this chapter we have to mention an interesting point suggested by Vesely and Mintz 1978they found that diabetic patients with chronic renal failure show marked deterioration of their renal function after I.V.P.

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They suggested that this deterioration results from the fact that diabetes cause both diminution of renal blood flow and increase in R.B.C. aggregation, the addition of urographic dye Worsen this position by enhancing R.B.C. aggregation causing slowing of micro circulation and at the same time results in vaso constriction of minute arterial and venous vessels resulting in decreased renal blood flow.

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