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In Shortly about Renal Disease

Review Article

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Abstract

Successful care for patients with kidney disease depends primarily on accurate diagnosis. It is essential to collect detailed data on the duration and severity of the symptoms of the disease, to collect information on family history and social status. No less significant data are related to drug intake, possible exposure to harmful agents. It is very important to collect general information in first examining patients. Patients are often anxious and interpreting the symptoms of the disease with difficulties.

Keywords: Renal, Kidney, Disease, Biopsy

Introduction

A patient with renal disease can present either as an initial outpatient or inpatient consultation [1]. Some patients may be referred because of abnormal urinary findings, such as hematuria or proteinuria, which may have been incidentally discovered during routine clinical evaluation or as part of initial employment requirements. Depending on the stage of renal disease, they can present with mild edema or generalized pruritus, as well as more advanced signs and symptoms of uremia, such as decreased appetite, weight loss, and even alterations in mental status. In general, the symptoms and signs of patients with renal disease tend to be nonspecific. Still others would present only with elevation in serum creatinine.

A freshly passed specimen of urine may be placed directly on a glass slide, covered with a fine glass cover slip and examined using a microscope [2]. Frequently, the urine is first centrifuged so that heavier material in the liquid urine sample is concentrated towards the bottom of the centrifuge tube and then a small aliquot of the deposit so obtained is placed upon the slide and examined. Normal urine contains less than two white blood cells and two red blood cells per field of vision when examined under the high power of a standard microscope. An excess of white cells (pyuria) or red cells (microscopic haematuria) constitutes an abnormality. Normal urine contains a protein (Tamm-Horsfall protein) which forms models of the renal tubules in its passage through the renal tubular system. Such 'casts' of the renal tubular system are seen in normal urine and are termed 'hyaline casts'. Models of the renal tubules composed of white cells or broken down white cells (white cell casts), or tubular cellular material and other debris (granular casts) are indicative of an abnormality. In the first case, renal sepsis is the likely explanation. Granular casts are a non-specific indicator of disease of the renalsubstance. Casts composed of red cells typically indicate disease of the renal glomeruli (glomerulonephritis). Red cells derived from lower down the urinary tract (such as those emanating from a bladder cancer) have not passed through the tubules and do not form casts.

Kidney

The kidney is an anatomically complex organ with exceptional cellular heterogeneity [3]. Our understanding of renal physiology has been advanced by studies of hand-dissected individual tubules (microdissection). Glomeruli or renal tubules are dissected away from surrounding structures, and subjected to microassays for enzymatic activity and receptor function, in vitro microperfusion for transport rates, and single-tubule reverse transcriptase-polymerase chain reaction (RT-PCR). Thus, microdissection methods have allowed assay of activity and mRNA and protein expression in single-nephron segments in normal or uninjured kidneys. Renal disease may be global, but more typically involves selective injury to the glomerulus, portions of the nephron, interstitium, or blood vessels. Less is known about renal pathophysiology and the nephron-specific response to injury. For example, how do different portions of the nephron interact during renal injury? Why are some renal diseases focal? How do individual glomeruli react in focal semental glomerulosclerosis (FSGS)? Does the interaction tend to propagate or to defend against further injury? Microdissection techniques have not been widely used to study renal injury because microdissection is often limited by tissue necrosis or fibrosis. In addition, the time required for microdissection often exceeds the time-course of rapid and transient changes in a cellular response such as certain metabolic intermediates or early-response genes. Instead, these questions are usually approached using immunohistochemistry or in situ hybridization. However, this depends upon the availability of specific antibodies that function in tissue sections, or optimization of complicated hybridization conditions. Also, genes must be studied one at a time, and, there is little specific information about human injury because of limited tissue availability.

Renal Function and Disfunction

Renal dysfunction is a common comorbidity complicating the natural course of heart failure (HF), and similarly patients with kidney disease often present accompanying heart disease [4]. This has led to the concept of cardiorenal syndrome (CRS), which is defined as 'pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ induces acute or chronic dysfunction in the other'. Although, guidelines for the management of patients with HF and kidney disease are well developed and regularly updated by the cardiology and nephrology societies, there are no agreed guidelines/recommendations for the management of patients with cardiorenal and/or renocardiac syndromes, as these patients have typically been excluded from clinical trials. In practice, however, such patients are commonly seen by either cardiologists or nephrologists and often become a real clinical challenge. The question which often arises is whether management of patients with HF requires modification for those with impaired renal function and vice versa.

Coronary artery disease and hypertension are the commonest causes of chronic HF, followed by valvular heart disease and cardiomyopathies. Renal dysfunction is a common finding in patients with HF, strongly associated with increased morbidity and mortality. It is estimated that more than 50% of HF patients may have at least moderately impaired renal function and its prevalence increases with HF severity, age, a history of hypertension or diabetes mellitus. Conversely, even mild renal dysfunction constitutes a risk factor for HF development and this association becomes stronger with deterioration in renal function.

The underlying cause of renal dysfunction should always be sought in order to detect potentially reversible causes (such as hypotension and/or dehydration due to drug overdosing, deterioration in renal function due to ACE inhibitors, angiotensin receptor blockers [ARBs] or other concomitant medications, e.g. non-steroidal anti-inflammatory drugs). When coincidental renal disease is suspected, further renal investigations are indicated. Renal dysfunction is not only a consequence of HF, but itself may play a key role in the pathophysiology of the HF syndrome. Thus, in all HF patients renal function should be regularly monitored by measurements of blood urea nitrogen, serum creatinine levels and/or estimation of glomerular filtration rate. Therapy in HF patients with concomitant renal dysfunction is not evidence-based, as these patients are not satisfactorily represented in randomized clinical trials of HF.

HF patients with renal dysfunction often have excessive salt and water retention, which requires more intensive diuretic treatment. In patients with a creatinine clearance of <30 ml/min, thiazide diuretics are less effective and loop diuretics are preferred. Patients may be at a higher risk of further deterioration in renal function when high doses of diuretics are used. More potent options may be

diuretic infusions instead of intravenous boluses and a combination of loop diuretic and thiazide/metolazone.

Many glomerular and a small number of tubulo-interstitial and vascular disorders are immunologically mediated [5]. These may be the result either of antibody-mediated or cell-mediated processes. In most instances in humans, the immediate cause or antigenic stimulus for the immune reaction is not known. The detection of antibody-mediated damage in renal tissue depends on the use of immunofluorescence microscopy.

Most glomerulopathies are immunologically mediated and are the result of antibody-induced injury. This can occur as a consequence of antibody combining with an intrinsic antigen in the glomerulus or antibody combining either in situ or in the circulation with an extrinsic glomerular antigen, with immune complexes localizing or depositing in glomeruli. With circulating immune complexes, the antigens may be of endogenous or exogenous origin. Endogenous antigens occur in diseases such as systemic lupus erythematosus and include components of nuclei such as DNA, histones, etc. Exogenous antigensare usually of microorganism origin and include bacterial products, hepatitis B and C viral antigens, malarial antigen, etc. Circulating immune complexes are trapped or lodge in glomeruli in the mesangium and subendothelial aspects of capillary walls. Less commonly, they may be found in subepithelial locations. It is the electron microscope that precisely localizes the deposits. Certain diseases are characterized by deposits in predominately one site, whereas other diseases may be characterized by deposits in more than one location. Once immune complexes are deposited, complement is fixed and often leukocyte infiltration follows. The white blood cells accumulate incapillary lumina and infiltrate into the mesangium; in addition, intrinsic mesangial cells may divide and may also extend into peripheral capillary walls. The leukocytes, in part, may be responsible for removal of deposited immune complexes. The names of the many glomerular disorders, diagnostic criteria, and prognostic and therapeutic implications depend on the correct localization and identification of the immune complexes in the glomeruli.

Urography

Intravenous urography (IVU), sometimes referred to as intravenous pyelography (IVP) is one of the most valuable and frequently carried out diagnostic tools for the investigation of renal disease [2]. A plain abdominal X-ray is first taken, mainly in order to identify opaque stones overlying the urinary tract, and then an organic iodine-containing contrast medium is injected intravenously. The contrast is filtered at the glomeruli and concentrated in the renal tubular system and thus opacifies the kidneys, collecting systems, ureters and bladder. Numerous renal diseases can be diagnosed with confidence on IVU. Reactions to the contrast media employed are uncommon. Severe reactions are very uncommon, but when they occur may be lifethreatening, although the use of modern contrast media (non-ionic contrast) has reduced the incidence of such reactions. Facilities for resuscitation must be available and patients at special risk (those with a history of allergy to iodine or a previous contrast reaction, those with multiple allergies and asthmatics) should receive corticosteroid cover for the procedure. A typical regimen would involve the patient receiving prednisolone 40–60 mg 24 hours before and on the day of the examination. Contrast media may also be nephrotoxic in the absence of an allergic response, particularly in dehydrated patients, those with diabetes mellitus, those with pre-existing renal impairment and patients with the condition multiple myelomatosis.

Biopsy

Since its introduction in the 1950s, kidney biopsy carried out in life has played a major role in the investigation, diagnosis and successful treatment of kidney disease [2]. The technique is carried out under local anaesthetic with the patient prone, ie lying on his or her face with a pillow supporting the abdomen. The kidney to be biopsied is localised by ultrasound as to its position and depth, and the operator then prepares the skin, infiltrates local anaesthetic and advances a biopsy needle to the capsule of the kidney. With the patient holding his or her breath in inspiration, the biopsy needle is advanced so as to cut a small portion of kidney tissue and retain it within the equipment which is then removed. The procedure can be carried out under continuing ultrasound guidance or without such guidance, but bearing in mind the depth and position of the kidney previously ascertained. The biopsy equipment may be operated directly by the operator or may be automatic and activated by compression of a trigger which fires the biopsy needle. Biopsy can be carried out without ultrasound guidance but this technique is less reliable and probably carries greater risk. It would be difficult to defend adopting this approach in the event of complications occurring.

Hypertension

Hypertension (high blood pressure) is extremely common and is a major risk factor for coronary artery disease, heart failure and brain haemorrhage [2]. Most patients with serious renal disease are hypertensive. Conversely, most patients with high blood pressure do not have renal disease, leaving aside for the moment the argument that unexplained hypertension (so-called essential hypertension) which is overwhelmingly the most common variety, may have as its underlying cause a renal abnormality. It is unrealistic to expect all patients with hypertension to be referred to a specialist physician, still less to a specialist nephrologist. Referral is indicated if symptoms or signs of renal disease are present, or if a surgically correctable endocrine or renal cause for high blood pressure (such as overactivity of the adrenal glands in the former case and narrowing of a renal artery causing hypertension in the latter case) are suspected.

Patients with apparent essential hypertension may be found to have impaired renal function. The question then arises as to whether parenchymal renal disease exists which has caused the hypertension, or whether hypertensive kidney damage has occurred. The question can be settled by transcutaneous renal biopsy, but the yield of treatable disease is not high, especially in Afro-Caribbeans. Unless the history or examination findings suggest a primary renal disorder, or urinalysis and urine microscopy are suggestive of this (for example, by demonstrating heavy proteinuria or microscopic haematuria), renal biopsy in Afro-Caribbeans almost always shows primary hypertension. If complications of biopsy occur in this group of individuals, the indication for the investigation could be challenged and might form the subject of a claim for compensation.

Pregnancy

Renal physiologic changes are characterized by marked vasodilation, which leads to increases in glomerular filtration rate (GFR) and renal plasma flow (RPF) [6]. These changes occur early in the first trimester and peak increases in GFR and RPF to 50% above baseline are seen by the end of the first trimester. The filtration fraction (GFR/RPF) falls significantly, indicating a greater rise in effective RPF. Creatinine production is unchanged in pregnancy but creatinine clearance is increased, resulting in lower levels of serum creatinine; the normal creatinine value during pregnancy is <0.8 mg/dL.

Pregnancy in women with pre-existing renal disease can be harrowing for both the mother and the doctors, but increasingly results in healthy infants [7]. Pregnancy is typically complicated by hypertension, worsening proteinuria, and prematurity with or without worsening renal function.

There is a decrease in systolic blood pressure of about 9 mmHg and in diastolic blood pressure of 17 mmHg during pregnancy. The lowest blood pressure is seen between 16 and 20 weeks of gestation, and the blood pressure gradually increases toward term. Hypertension is the most common medical problem occurring during pregnancy.

Obesity Paradox

The 'obesity paradox' is not limited to patients with renal disease [8]. In a large population with hypertension and coronary heart disease, overweight and obese patients had decreased risk of major cardiovascular events, particularly mortality, compared with 'normal'weight patients. These data are in agreement with a recent meta-analysis that demonstrated better cardiovascular outcomes in overweight and mildly obese coronary heart disease patients compared with those with ideal weight and especially compared with underweight patients. In this meta-analysis the better outcomes for cardiovascular and total mortality seen in the overweight and mildly obese groups could not be explained by adjustment for confounding factors.

Urinary Tract Infections

Urinary tract infections (UTI), one of the most common bacterial infections, affl ict 50 % of all women at least once in their lifetime with a 20–30 % chance of a recurrent infection [9]. Further, the risk of UTI is elevated in people with altered metabolism, including diabetes, obesity, and pregnancy. The major etiological agent is uropathogenic Escherichia coli (UPEC) accounting for ~80 % of noncomplicated community- acquired UTI.

While lower UTI represents the majority of UTIs, upper UTI can result in severe effects on host metabolism. This is thought to be due to abrogation of kidney function via renal damage and scarring. In response to bacterial kidney colonization, the immune system, predominantly the innate component, mounts an aggressive response designed to eliminate the pathogen. This can result in signifi cant kidney damage via reactive oxygen species and ischemia generated by a robust immune response and

infl ammation. This damage can lead to severe metabolic complications including hypertension, uremia, and kidney failure.

Diagnosis

There are three main ways in which kidney damage can occur: pre-renal, post-renal and renal CRF (Chronic Renal Failure) [10]. With pre-renal causes, conditions like hypovolaemia, as in major bleeds, or poor cardiac function or stenosis of the renal arteries can cause continuous hypoperfusion which may ultimately lead to kidney ischaemia and necrosis, resulting in CRF. In post-renal CRF, a disruption of urine flow from the kidneys by bladder obstruction, ureteric stones, retroperitoneal fibrosis, etc. increases pressure within the kidneys, eventually damaging nephrons and resulting in CRF. The most common causes of irreversible damage occur primarily within the kidney (renal CRF) and include diabetic nephropathy, hypertensive nephrosclerosis, vasculitis (including lupus and Wegener's granulomatosis), interstitial nephritis, and polycystic kidney disease.

Diagnosis is founded on a detailed medical history, physical examination and a considerable array of immunological and other serological tests. Precise diagnosis is important for identifying and treating potentially reversible causes. It can also assist nephrologists with assessing prognosis and planning for replacement therapy. Kidney biopsies are sometime inconclusive as late in the disease diffuse scarring may obscure the primary cause. In these cases or where a biopsy was not performed the diagnosis may be given as unknown.

The first step in the development of renal failure is an insult, which results in loss of functioning nephrons [11]. This may be acute, for example, hemodynamic compromise leading to renal ischemia, or chronic such as in the case of a persistent glomerulonephritis. Not all insults lead to progressive kidney disease; for example, patients with renal impairment secondary to urinary tract obstruction can maintain stable renal function in the long-term once the obstruction has been relieved.

However, once a threshold has been passed— usually when 60% to 70% of renal function has been lost, CKD tends to become progressive. This is the consequence of pathogenic processes, which lead to glomerulosclerosis and tubulointerstitial fibrosis.

Critically Ill Patients

Critically ill patients with renal failure often have multiple ongoing acute processes and require a multidisciplinary approach to management [12]. This is ideally performed in a multidisciplinary ICU (Intensive Care Unit) with patients cared for by trained intensivists available 24h/day, 7days/week. This does not mean there is no place for the nephrologist in the care of such patients, but overall responsibility must lie with the intensivist. Any consultant from any specialty should be welcome on the ICU at any time; the patient must be at the center of attention, and visits from other specialists must be welcomed if they can improve patient care. In collaborating with other specialties it is important that each party acknowledges the respective competences of the other, and that there be open and honest discussion of all issues associated with patient management. Critical care nephrology is a multidisciplinary field, and only a multidisciplinary team approach to patient management, under the ultimate coordination and control of a trained intensivist, will provide the best possible care for ICU patients with renal disease.

Conclusion

Renal disease causes significant changes in patients' lives. Many of them have good quality of life. They have a fulfilling life, they are employed, they study, they play sports, they take care of their family. Many patients begin dialysis treatment with the hope of a quick transplant, which is not always successful. Patients must be allowed to choose treatment.

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