Renal failure from the Internist's perspective: clinical and laboratory management

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Abstract

A physician's approach to renal failure should always be multifactorial. It is important to establish a differential diagnosis as regards the various types of renal failure and to differentiate between an intrinsic renal lesion and multisystem disorder. The initial approach should be non invasive and consists of a careful clinical history and examination, the analysis of the urinary sediment and the renal ultrasound. The uncertainty about the etiological diagnosis and the need to correctly assess the degree of renal insufficiency determines the need to perform a renal biopsy. The most important clinical aspects are due to failure to excrete nitrogenous products, volume and salt overload.

Key words: systemic disease, acute renal failure, chronic renal failure, azotaemia.

Introduction

Multisystemic diseases with renal involvement, such as diabetes, high blood pressure and connectivitis, are relatively common in hospitals and external medical clinics. Progression to renal insufficiency (RI) is often inevitable, and the internist should be familiar with this nosological entity, in order to carry out the correct diagnostic approach, and apply therapeutic and preventative medications to control the condition.[1,2]

The renal patient often arrives at the Internal Medicine clinic with complaints directly related to the kidneys, such as low back pain, alterations in urinary debit, dysuria and hematuria, or due to extra-renal anomalies, such as edema, high blood pressure, and clinical signs of uremia. A considerable group of patients does not present any symptoms, and in these cases, the alterations in renal function are an accidental finding.

Anamnesis and objective examination provide valuable elements for detecting the manifestations of diseases with renal involvement. For example, simple palpation of the abdomen and pelvic area can indicate a low obstructive uropathy if there is vesicular distension, for the existence of polycystic kidneys, peripheral vascular disease, or inflammatory or neoplastic masses. The presence of abdominal murmurs may indicate a stenosis of the renal artery. Purpuric skin rash may suggest vasculitis.[1,4]

In the initial approach by the internist of a patient with renal insufficiency, it is important to make a differential diagnosis between acute renal insufficiency (ARI) and chronic renal insufficiency (CRI), which may be mild, moderate or severe, or acute chronic renal insufficiency (ACRI), which can be controlled after resolution of the acute problem.

Clinical and laboratory aspects

ARI is characterized by a rapid deterioration of renal function, with a decrease in glomerular filtration rate (GFR), resulting in an accumulation of nitrogenated products, such as urea and creatinine. There are seven main syndromes related to ARI, classified according to the site of the kidney lesion, namely: Pre-renal azotaemia, in which there is a decrease in GFR due to renal hypoperfusion, reversible with an increase in blood flow to the kidney, without structural lesion of the kidney; acute tubular necrosis (ATN), in which

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TABLE I

Elements indicative of ARI

Evidence of a recent decrease in GFR
Relationship between deterioration of GFR and possible etiological factors (anamnesis)
Adequate estimate of renal perfusion before and during deterioration of function (volume, cardiac function, renovascular, BP, weight, water/sodium balance)
Existence of exogenous or endogenous nephrotoxins
Existence of manifestations of extra-renal disease
Urine exam (volume, sediment, composition)
Imaging exams related to the kidney
Kidney biopsy

there is a decrease in GFR due to hypoperfusion or nephrotoxicity, not immediately reversible by interrupting the cause, and associated with tubular lesion; acute interstitial nephritis, in which there is a decrease in GFR due to glomerular inflammation; vasculitis, where there is a decrease in GFR caused by lesion of the small vessels; acute renovascular disease, which causes ARI by arterial or renal obstruction (thrombosis, embolism) in a single functioning kidney, or with bilateral renal disease; finally, obstructive uropathy, in which the decrease of GFR results in a blockage of the urinary collection system (postrenal azotaemia).[2,3]

ARI occurs in around 5% of all patients admitted to hospital, and of these, pre-renal cause (excessive salt and water restriction) and ATN account for 70% to 75% of all the etiologies.[2]

The most frequent causes of ARI in hospitalized patients are iatrogenic (aminoglycosides, radiological contrasts, post-surgical) and sepsis. Ampicillin and cephalosporins are also iatrogenic causes of ARI, but act by different mechanisms (acute interstitial nephritis). Another cause of ARI is angiotensin-converting enzyme inhibitors (ACEI) in patients with bilateral stenosis of the renal arteries or stenosis of the renal artery in a single functioning kidney, and non-steroid anti-inflammatories (NSA) in patients with renal hypoperfusion.[1,7] *Table I* shows some elements that can contribute to the diagnosis of ARI.

In CRI, it is important to give value to the elements of the clinical history, and complementary means of diagnosis that suggest chronicity. Thus, the presence of persistent nocturia may be associated with decreased capacity of urine concentration capacity or low obstructive uropathy, which occurs in patients with a history of neoplasm of the bladder or pelvic cavity, and prostate disease. Other signs of chronicity are persistent and progressive worsening of some Laboratory parameters, such as decreased hemoglobin, increased urea and creatinine, alterations in urinary sediment, and the existence of small-sized kidneys (renal echography). Amyloidosis, rapidly progressive glomerulonephritis and diabetic glomerulosclerosis do not follow this criterion. In these cases, the kidneys are of normal dimensions or enlarged.[1,4] *Table II* shows some useful elements in the diagnosis of CRI.

TABLE II

Elements indicative of CRI

Chronic history of nocturia, polyuria edema or hematuria, itching, other uraemic symptoms (e.g. neuropathy), predisposing disease (high blood pressure, diabetes mellitus)
Objective data: Small kidneys and renal osteodystrophy
Laboratory data: Hypocalcaemia, hyperphosphatemia, anemia

In situations of azotaemia, with retention of nitrogenous products, the most important clinical findings are gastrointestinal alterations such as anorexia, nausea, vomiting, abdominal pain, diarrhea, paralytic ileum, hiccups, uraemic halitosis, gastrointestinal hemorrhage, and neurological alterations.[1,3] The retention of salt and water leads to congestive cardiac insufficiency. Volume-dependant high blood pressure, pulmonary edema, peripheral edema, ascites and pleural effusion are also consequences of this condition. The relative frequency is highlighted, of asymptomatic patients with conditions of terminal uremia.[1,4,6]

In the laboratory investigation of the renal patient, urine analysis and renal echography are the best non-invasive methods for a preliminary approach. Normal urinary sediment occurs in situations of pre-renal azotaemia. Hematuria, pyuria, and crystalluria are observed in obstructive diseases. The presence of blood casts associated with proteinuria suggests glomerular disease. Leukocyturia, eosinophil urea and isothenuria are indications of tubulointerstitial nephritis. This study also includes 24-hour urine for study of proteinuria and determination of creatinine clearance which, if lower than 20%-25% of the no-

TABLE III

Urinary indices of diagnosis

Indices	Pre-renal azotaemia	Oliguric ARI
Urinary density	> 1020	≅ 1010
Urinary sodium Na (U) mEq/l	< 20	> 40
Urinary osmolality, mOsm/kg H20	> 500	< 350
Cr (U) / Cr (P)	> 40	< 20
Renal insufficiency:		
R.I.I. = Na (U) / Cr (U)/ Cr (P)	< 1	> 1
Excreted fraction of filtered Na: FENa = Na (U) x Cr (P) x 100 / Na(P) x Cr (U)	< 1	> 1

Key: Cr (U) = urinary creatinine; Cr (P) = plasma creatinine; Na (U) = urinary sodium; Na (P) = plasma sodium; R.I.I. = renal insufficiency index
Adapted from Urinary diagnostic indices, in Manual of Nephrology – 9.1994:143

minal value, indicates severe RI. The other routine exams should not be neglected.[1,5,8]

The urinary diagnostic indices most commonly used in clinical practice are summarized in *Table III*.

Radiological studies, besides renal echography, include simple X-ray of the abdomen and possibly, excretory urography, retrograde pyelogram, isotopic studies, angiography and computed axial tomography (CAT scan).

Renal biopsy should be carried out for etiological clarification, generally with a view to instituting therapy. Nephrotic proteinuria, alterations in urinary sediment, such as hematoproteinuria, erythrocyturia with blood casts or dysmorphic erythrocytes, particularly when associated with alterations in renal function, are situations with indication for renal biopsy.[1,5]

In the evaluation of the effects of diabetes on renal function, it is important to bear in mind not only the alterations in renal function itself, but also the duration of the disease and the involvement of the remaining organs. Thus, if the diabetes has an evolution of more than 10 years, with the existence of diabetic retinopathy, generally nephrotic proteinuria, and high urea and creatinine levels, renal biopsy does not have formal indication; on the contrary, if the RI is associated with diabetes of recent installation, with alterations in urinary sediment and absence of diabetic retinopathy, renal biopsy is necessary because in this situation, the cause of the renal dysfunction is probably not attributable to the diabetic nephropathy.[1,5]

The patient who has not yet started on dialysis should be accompanied by the internist, in collaboration with the nephrologist. Careful prescription of nephrotoxic drugs (antibiotics, ACEI), the control of blood pressure, dietary surveillance (restriction of salt and phosphorus) and prophylaxis of urinary infections are parameters to be carefully monitored, in order to prevent or delay the progression of the renal disease. The causes of morbidity and mortality in RI should be detected and treated early on, including, in this group, infections, complications of hydro electrolytic balance and gastrointestinal hemorrhage.

When the situation evolves to terminal chronic renal insufficiency, with indication for renal replacement therapy, the patient should be referred to a nephrologist, who should schedule the most appropriate therapy for this phase, and other procedures, such as vascular or peritoneal access. The psychological situation of the patient has been indicated as having a great impact on the success of the dialysis. Thus, patients with more flexible lifestyles, and good family support, have greater success and are more able to adapt to a life on dialysis, avoiding some long-term complications, such as cardiac, osteomuscular or atherosclerotic diseases.

Conclusions

It can be concluded that the internist plays an important role in the etiological, clinical and laboratory evaluation of systemic disease with renal repercussions. It is essential to have good articulation with the nephrologist for a complete planning in the dietary counseling, clinical, laboratory and therapeutic control. The prescription of renal replacement therapy is the responsibility of the nephrologist. However, there are fundamental rules that the internist should be aware of, in order to collaborate in its timely planning. ■

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