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# Introductory Chapter: Renal Diseases

*Edward T. Zawada*

## 1. Introduction

Renal diseases are notoriously silent. Renal diseases are notoriously expensive once they have led to end-stage renal failure requiring dialysis or transplantation. Most acute and chronic renal diseases present with rising serum creatinine and blood urea nitrogen, electrolyte abnormalities, frequent proteinuria, occasional red blood cells in the urine, occasional white blood cells in the urine, or renal cells in the urine often mistaken for white blood cells. It is not easy to make a specific diagnosis from these similar presentations.

I will now present the rationale for renal biopsy to establish renal diagnoses [1]. The classification which follows is the opinion of the author and editor based on over 45 years of experience and exposure to the pioneers in use of renal biopsy for diagnosis [2–4]. My goal is to simplify this argument for performing this invasive procedure to clarify the confusing array of renal diseases with indistinct or asymptomatic presentations.

## 2. Renal disease diagnosis

Acute renal diseases are described as prerenal, renal, and postrenal, but most patients totally recover normal or near-normal renal function. It is the chronic, often symptomless chronic diseases which lead surreptitiously to end-stage renal disease. Chronic diseases include vascular, glomerular, tubular, and interstitial diseases. The main structures of the kidney are arteries and veins, glomeruli, tubules, and interstitium. Renal biopsy is often required to determine the site of injury. As described below, even when the site of injury is known, such as in glomerular diseases, the renal biopsy is needed to distinguish between the many similar diseases in order to develop possible remission-inducing therapy strategies.

I have been teaching about renal diseases since for 40 years since 1979. I have been struck by three main perspectives. First, diseases can occur in very small areas of the structures of the kidney while the rest of the kidney tissues work well and try to compensate. Second, glomerular diseases are confusing to understand because one needs a framework to separate the multiple very similar diseases into separate clinically relevant entities allowing individual management. Finally, the tubular diseases are the most silent of renal diseases, needing more effort to teach, understand, identify, and manage. Renal biopsy results will be briefly described as the tool to make a definitive diagnosis necessary for proper patient management.

### 3. Renal biopsy

Normal renal histology must be known to the clinician before biopsy can be interpreted. The glomeruli will be numerous and have wide-open, thin-walled capillary loops; no inflammatory cells seen; and no increase in numbers of mesangial cells. There can be less than 30% of any glomerulus or all glomeruli which contain amorphous loss of architecture called sclerosis. The blood vessels need to be open without cellular or muscular thickening. Finally, the cross section of renal tubules normally abuts directly against each other without evidence of inflammatory cells or scarring by fibrosis between them.

### 4. Renal diseases identified by biopsy

Glomerular diseases require the visualization of light microscopy to best understand the numerous entities, many of which have similar immune-mediated pathophysiology. There are nine main light microscopic patterns. Each has a primary and secondary form. This classification allows one to understand the differences of 18 different confusing entities. These patterns are nil lesion, diffuse membranous, diffuse proliferative, diffuse membranoproliferative, focal proliferative, focal sclerosis, nodular glomerulosclerosis, fibrillary, and amyloidosis. For almost everyone, there is a primary and secondary form. The primary forms are idiopathic. The secondary forms are due to a bacteria or virus, a drug, or a systemic disease such as lupus. Based on the light microscopy, the clinician determines if there is a definite cause or whether it is primary. From either category a specific management has reached consensus by nephrologists through international symposia and guidelines.

Tubular diseases do not often show azotemia early. Rather subtle electrolyte findings such as hyperkalemia, hyperuricemia, or non-anion gap metabolic acidosis suggest this category of disease. However, since those disturbances can occur with nonrenal diseases such as gastrointestinal problems, they are not often recognized as early signs of renal damage. Renal biopsy helps distinguish the following most common causes: obstructive or reflux nephropathy, hypertensive injury called acute or chronic nephrosclerosis, drug-induced diseases such as due to analgesics, heavy metal injury such as due to lead, crystal diseases such as urate or oxalate, and unknown causes such as Balkan or other environmental nephritides.

Vascular diseases may occur with or without glomerulonephritis. Fibrinoid necrosis, chronic hypertensive changes of the arteries (nephrosclerosis), and vasculitis are the most common patterns. Fibrinoid necrosis is seen with malignant hypertension, thrombotic thrombocytopenic purpura, scleroderma, eclampsia, and disseminated intravascular coagulation.

Interstitial diseases are identified by acute and chronic inflammatory cells or fibroblasts and collagenous scarring between the tubules causing secondary tubular injury and renal failure. Chronic allograft nephropathy and chronic pyelonephritis are two examples.

### 5. Conclusion

The above framework to be used for the management of patients with chronic unexplained or understood renal insufficiency is frequently changing. New entities are appearing to add more patterns, more secondary causes of glomerular diseases, and new algorithms for management [5, 6]. Renal disease due to human

immunodeficiency virus, new drugs including new biologicals, and new immunologic diseases accounts for some of these newer entities.

In conclusion, a word should be said about repeat renal biopsies. Repeat biopsies are encouraged to assess response to treatment such as in allograft rejection. Repeat biopsies are used to reassess those diseases such as lupus nephritis which can change course leading to new pathophysiology and demanding a change in therapeutic strategy.

Finally, the group at New York Presbyterian/Columbia University Medical Center should be commended as they have continued to provide the most comprehensive annual tutorial and update of the interpretation of renal biopsies based on the very large number of biopsies referred to them [7]. Many of the chapters in this book concentrate on the importance of renal biopsy for diagnosis. Other chapters deal with methods of performing renal biopsy. Finally, there are chapters dealing with complications of renal biopsy.

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