An Update on Glomerular Disease in the Elderly

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KEYWORDS

• Glomerulonephritis • Nephrotic syndrome • Immunosuppressive therapy
• Primary glomerular disease • Secondary glomerular disease

KEY POINTS

- Primary and secondary glomerular disease
- Older adult
- Nephrotic syndrome

INTRODUCTION

Both acute and chronic glomerular disease are common causes of disability, hospitalization, and mortality in the elderly population.[1,2] The underlying causes of the glomerular disease in this population are diverse but can be apportioned into those that affect the kidneys primarily (primary glomerular disease) and those in which the kidney damage is a part of a system-wide process (secondary glomerular disease). Regardless of the underlying cause, the clinical features can be condensed into a few clinical syndromes. The comorbid features often associated with advancing age (such as diabetes, atherosclerosis, and congestive heart failure) have important modifying influences on the clinical presentation and course of glomerular disease. This article discusses the common presentations of glomerular disease in the elderly and the clinical features, prognosis, and management of the primary and secondary glomerular diseases in this population of patients.

CLINICAL PRESENTATION

The common clinical syndromes observed at presentation for glomerular disease are shown in **Box 1**.

Acute nephritic syndrome (ANS) is characterized by the abrupt onset of hematuria and proteinuria, often accompanied by edema, hypertension, and reduced renal function (impaired glomerular filtration rate [GFR]). In the elderly, because of concomitant

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Box 1
The common clinical syndromes of glomerular disease

Acute nephritic syndrome

Rapidly progressive glomerulonephritis

Nephrotic syndrome

Asymptomatic hematuria and/or proteinuria

Chronic glomerular disease

chronic ischemic heart disease or preexisting hypertensive cardiomyopathy, marked fluid retention with acute congestive heart failure (pulmonary edema) may be a presenting feature in the ANS, even when the urinary findings are easily overlooked. The disorders evoking the ANS are often accompanied by a tendency for spontaneous recovery, although this is less evident in the elderly compared with younger persons. An antecedent streptococcal (throat or skin) or an active staphylococcal (skin or soft tissue) infection may be documented (poststreptococcal glomerulonephritis or staphylococcal-related glomerulonephritis).

Rapidly progressive glomerulonephritis presents in a more insidious fashion and also manifests hematuria and proteinuria, but is characterized by a progressive and relentless (if untreated) loss of renal function and less evidence of edema and hypertension. Progression to end-stage renal disease (ESRD) may occur in a matter of days or weeks. Although an infection may also be present, such features are usually absent, especially in the elderly. If caused by a systemic disease (such as vasculitis) extrarenal symptoms and signs are commonly present (eg, palpable purpura, lung hemorrhage).

Nephrotic syndrome is characterized by the abrupt or insidious onset of marked proteinuria (usually more than 3.5 g/d up to more than 20 g/d), with or without hematuria, but with a prominent tendency for edema (face and legs), hypoalbuminemia, and hyperlipidemia. Renal function is variable and may be normal or reduced. In the elderly, the peripheral edema may be severe if there is concomitant congestive heart failure or venous insufficiency.

Asymptomatic hematuria and/or proteinuria is characterized by symptom-free (eg, no lower urinary tract symptoms such as dysuria or frequency) excretion of abnormal numbers of intact erythrocytes and/or increased amounts of protein (albumin), or both, in association with normal renal function (adjusted for age), normal blood pressure, and absence of edema. This presentation is often discovered serendipitously during a routine urinalysis, although at times the hematuria may be episodic and gross or macroscopic, thus bringing the patient to the physician's attention. Most often the hematuria is microscopic and persistent or episodic.

Chronic glomerular disease can be said to be present when any of the conditions causing glomerular injury have progressed slowly to definitely impaired renal function (usually a GFR<45 mL/min/1.73 m^2 in the elderly), nearly always with an increased serum creatinine (Scr) concentration. Some degree of proteinuria, often nonnephrotic, and hypertension is also nearly always present.

One feature of the hematuria observed in the glomerular disease syndromes is the excretion of increased numbers of abnormally shaped, smaller than normal (microcytic), poorly hemoglobinized (hypochromic) erythrocytes in the urine.[3] This is also known as dysmorphic or glomerular hematuria. It is a valuable diagnostic tool that requires careful microscopic examination of the urinary sediment (using specials stains or a phase contrast microscope) for accurate detection. When present in larger than