Effect of Aging on Renal Function Plus Monitoring and Support

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KEYWORDS

• Elderly patients • Aging kidney • Acute kidney injury • Renal replacement therapy

KEY POINTS

- There are several histologic changes in the aging kidney that occur as a patient grows older, including renovascular changes, tubulointerstitial fibrosis, glomerulosclerosis, and loss of nephrons.
- The structural changes of the aging kidney are accompanied by physiologic alterations in renal function, with a decreased ability of autoregulation of renal blood flow, diminished capacity to maintain water and electrolyte homeostasis, and heightened sensitivity to changes in renal blood flow.
- The net effects of the anatomic changes and alterations in renal physiology in the elderly patient lead to an increased susceptibility toward developing hypovolemia.
- Acute kidney injury (AKI) is common in hospitalized elderly patients, and leads to substantial morbidity and mortality; although the causes of AKI in the elderly are the same as in the general population, the distribution of the causes is slightly shifted.
- No specific therapy exists for treatment of the elderly patient with AKI; treatment is mainly supportive and should include the full spectrum of therapies offered to younger patients, including renal replacement therapy.

INTRODUCTION

Because of advances in medical therapy, the elderly represent the fastest growing segment of the general population. It is projected that, in the United States and Western Europe alone, there will be an increase in individuals older than 60 years from 231 million in 2000 to approximately 400 million by 2050.[1] To care for this rapidly increasing portion of the population, it is essential for clinicians to comprehend the effects that aging has on normal physiology, and the accompanying clinical consequences that these physiologic alterations can have when these elderly patients are

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confronted with illness or injury. As patients age, several accompanying histologic alterations in renal functioning occur. The onset of these architectural and anatomic changes leads to subsequent physiologic perturbations, which can affect the care and prognosis of the elderly patient. Geriatric patients with chronic kidney disease (CKD) are particularly predisposed toward the development of acute kidney injury (AKI) and its adverse clinical sequelae. Understanding the mechanisms by which aging renal function deteriorates can lead to a better comprehension of how clinicians should approach the care of the injured or critically ill elderly patient.

THE EFFECTS OF AGING ON RENAL STRUCTURE

Several structural changes occur in the aging kidney, starting with histologic alterations in the renal vasculature (**Box 1**). Microangiopathic examination shows arteriolar deposition of subendothelial collagen fibers and hyaline, in addition to proliferation of elastic tissue, the sum of which leads to intimal thickening of both the afferent and efferent renal arterioles. These histologic changes are often associated with the development of atherosclerosis and atrophy of the smooth muscular media.[2] There is also a decreased ability in the capacity of the aging kidney to preserve the autonomic renal vascular reflex, which is termed renovascular dysautonomy; this diminished ability to maintain homeostasis can modify the ability to preserve renal function in both hypotensive and hypertensive states. Expansion of the mesangial matrix in the aging kidney induces destruction and extirpation of juxtamedullary nephrons, producing direct configurations and communication between the afferent and efferent arterioles. This structural process gives rise to a glomerular circulation, in which normal renal blood flow is compromised; the average decrease in renal blood flow is approximately 10% per decade starting at age 40 years.[3]

Box 1
Histologic changes in the aging kidney

Renal vasculature

- Subendothelial deposition of collagen fibers and hyaline in arterioles
- Proliferation of elastic tissue
- Atherosclerosis
- Smooth muscular medial atrophy

Mesangial matrix expansion

Aglomerular circulation

Glomerulosclerosis

Renal tubular atrophy, fibrosis and loss

Interstitial fibrosis

Decreased availability of functional sodium-potassium-chloride transporters

Nuclear changes

- Decreased tubular cell proliferation
- Increased susceptibility to apoptosis
- Telomere shortening
- Aberrant DNA methylation