Test Your Knowledge: Hypertension in CKD

A recent *AJKD* Core Curriculum by Ku et al reviews the pathophysiology, diagnosis, and management of hypertension in patients with chronic kidney disease (CKD). Test your knowledge on this topic with the quiz below.

1. All of the following factors contribute to hypertension in CKD EXCEPT:

    A. Reduced blood flow in peritubular capillaries downstream of sclerosed glomeruli leading to activation of the renin-angiotensin system
    B. Reduced nephron mass
    C. Increased nitrous oxide production
    D. Excess extracellular volume

2. All of the following are proposed mechanisms for hypertension in patients with CKD EXCEPT:

    A. Secondary hyperparathyroidism leads to increased intracellular calcium levels
    B. Vascular calcification leads to systolic hypertension
    C. Uremia leads to vasodilation via endothelial dysfunction
    D. Erythropoietin and erythropoiesis stimulating agents cause vasoconstriction

3. Which of the following are true regarding ambulatory blood pressure monitoring (ABPM)?

    A. Blood pressure should decrease by > 20% during sleep, which is appropriate nocturnal physiologic dipping
    B. Masked hypertension is not associated with increased risk for cardiovascular disease in CKD
    C. ABPM entails measurements every hour while awake and every 2 hours while sleeping
    D. A standard automated BP of 140/90 mm Hg correlates with an ABPM 24-hour average BP of 130/80 mm Hg

- ***Quiz prepared by Nimra Sarfaraz, AJKDBlog Contributor. Follow her @DrNimraS.***

To view Ku et al (FREE), please visit AJKD.org.

Title: Hypertension in CKD: Core Curriculum 2019
Authors: Elaine Ku, Benjamin J. Lee, Jenny Wei, and Matthew R. Weir
DOI: 10.1053/j.ajkd.2018.12.044

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1. **C. Increased nitrous oxide production**

   Endothelial dysfunction impairs nitrous oxide production, which is implicated in the pathogenesis of hypertension in CKD.

   Reduced blood flow to peritubular capillaries downstream of sclerosed glomeruli leads to reduced effective (perceived) blood flow, leading to renin being secreted from the juxtaglomerular apparatus. Reduced nephron mass implies that the fewer functioning glomeruli in CKD muse increase its GFR via increasing systemic arterial pressure and increased perfusion pressure. Excess extracellular volume expansion in peripheral tissues stimulates vasoconstriction, raises peripheral vascular resistance, and increases blood pressure.

2. **C. Uremia leads to vasodilation via endothelial dysfunction**

   Uremia may impair vasodilation by inhibiting nitric oxide synthase, which leads to vasoconstriction. All of the other mechanisms are factors that also may contribute to the high prevalence of hypertension in CKD.

3. **D. A standard automated BP of 140/90 mm Hg correlates with an ABPM 24-hour average BP of 130/80 mm Hg**

   Average daytime BP of 135/85 mm Hg and nighttime BP of 120/70 mm Hg average to about 130/80 mm Hg. SPRINT showed that routine clinic BPs with unobserved automated devices could be 5-10 mm Hg higher than BPs obtained in clinical trials.

   Appropriate nocturnal physiologic dipping shows that blood pressure should decrease by approximately 10% during sleep. Masked hypertension is defined by normal office BPs but elevated out-of-office BPs increase CV risk for patients with CKD, as do nocturnal hypertension and nondipping status. ABPM measures readings every 15-20 minutes during the day, and every 30-60 minutes during sleep.

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