Lecture Code : FE01-S4
Session Name : Fluid & Electrolyte
Session Topic : Fluid and Electrolyte Challenges in Nephrology: from Bench to Bedside
Date & Time, Place : June 20 (Fri) / 10:40-12:20 / Room 2 (GBR 102)

Kidney Stones: Pathogenesis and Treatment

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Kidney stones are crystalline concretions formed within the kidneys when urinary solutes become supersaturated. The pathogenesis begins with supersaturation of stone-forming substances such as calcium, oxalate, or uric acid (Coe et al., 2005). Crystals nucleate either spontaneously (homogeneous nucleation) or on preexisting surfaces like Randall's plaques (heterogeneous nucleation) (Evan et al., 2003). These crystals grow, aggregate, and may adhere to renal tubular epithelium, eventually forming clinically significant stones. Risk factors include metabolic abnormalities (e.g., hypercalciuria, hyperoxaluria), dietary influences (high sodium and animal protein intake), genetic conditions (cystinuria, primary hyperoxaluria), and urinary tract infections (Türk et al., 2016). Dehydration remains a major environmental contributor. Treatment begins with acute management. Pain control using NSAIDs or opioids is essential. Adequate hydration is encouraged to promote spontaneous stone passage. Medical expulsive therapy, primarily using α-blockers like tamsulosin, can aid the passage of small distal ureteral stones (Ferket et al., 2015). Surgical intervention is indicated in cases of persistent obstruction, infection, or stones unlikely to pass naturally. Options include ureteroscopy, shockwave lithotripsy (SWL), and percutaneous nephrolithotomy (PCNL) (Assimos et al., 2016). Long-term prevention focuses on addressing underlying metabolic abnormalities. Increased fluid intake (>2–2.5 L urine/day), dietary modifications (low sodium, moderate calcium intake), and pharmacologic therapies such as thiazide diuretics, potassium citrate, or allopurinol are essential strategies (Pearle et al., 2014). Regular metabolic evaluation and follow-up imaging are critical to prevent recurrence and protect kidney function. References Coe FL, Evan AP, Worcester EM, Lingeman JE. Kidney stone disease. J Clin Invest. 2005;115(10):2598-2608. doi:10.1172/JCI26662 Evan AP, Lingeman JE, Coe FL, Worcester EM. Randall's plaque of patients with nephrolithiasis begins in basement membranes of thin loops of Henle. J Clin Invest. 2003;111(5):607-616. doi:10.1172/JCI16886 Türk C, Petřík A, Sarica K, et al. EAU Guidelines on Interventional Treatment for

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Keywords: Kidney stones, Urolithiasis, Pathogenesis, Treatment, Prevention