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REVIEW PERSPECTIVE

Nephrology key information for internists

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ABSTRACT

Hospitalists and primary care physicians encounter renal disease daily. Although most cases of acute kidney injury (AKI) are secondary to dehydration and resolve by giving fluids, many cases of AKI are due to not uncommon but unfamiliar causes needing nephrology evaluation. Common indications to consult a nephrologist on an emergency basis include hyperkalemia or volume overload in end stage renal disease patients (ESRD). Other causes of immediate consultation are cresenteric glomerulonephritis / rapidly progressive glomerulonephritis in which renal prognosis of the patient depends on timely intervention. The following evidence-based key information could improve patient care and outcomes.

Abbreviations: AKI: Acute kidney injury ESRD: End stage renal disease patients

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Hospitalists and primary care physicians encounter renal disease daily. Although most cases of acute kidney injury (AKI) are secondary to dehydration and resolve by giving fluids, many cases of AKI are due to not uncommon but unfamiliar causes needing nephrology evaluation. Common indications to consult a nephrologist on an emergency basis include hyperkalemia or volume overload in end stage renal disease patients (ESRD). Other causes of Immediate consultation are crescentic glomerulonephritis / rapidly progressive glomerulonephritis in which renal prognosis of the patient depends on timely intervention. The following list is evidence-based key information, which could improve patient care and outcomes.

(1) Leukemia, causing high white blood cell counts (cell lysis leads to fragility), can cause spurious hyperkalemia [1]. This should be confirmed by arterial blood gas analysis so that patients are not inappropriately treated, causing arrhythmias and possibly death as a consequence. The use of vacuum tubes, pneumatic tube transportation, prolonged incubation, and tourniquet use have been implicated as causing cell lysis and pseudo-hyperkalemia. Arterial blood gas analysis is an extremely quick and reliable test [2].

(2) The most common cause of inadequate increase in hemoglobin in patients on erythropoietin includes coexisting iron deficiency. Iron stores should be replenished before increasing erythropoietin dosage. Among all hemodialysis patients who have transferrin saturation (TSAT) ≤20 percent and ferritin ≤200 ng/mL, the use of intravenous iron is preferred, provided an underlying infection has been excluded, regardless of the hemoglobin and regardless of whether patients are being treated with an ESA. Typical iron loss during hemodialysis may be as much as 2 g per year, which is highly likely to produce iron deficiency without supplementation.

(3) Spot urine protein creatinine ratio is good for monitoring progression of renal disease but cannot substitute a 24-hour urine protein for initial diagnosis of proteinuria. Higher gamma gap is a strong predictor for a positive serum or urine protein electrophoresis [3]. Total Protein Creatinine Ratio (TPCR) is a more sensitive screening test than Albumin Creatinine Ratio (ACR) to predict clinically relevant proteinuria and total proteinuria cannot be adequately predicted from ACR [4]. Utilizing total protein-to-creatinine ratio is acceptable if albumin-to-creatinine ratio is high (>500–1000 mg/g) [5].

(4) Hypertonic saline (3% HTS) can be administered via a peripheral inserted catheter [6]. Modern evidence does not support the concern for HTS-induced intravascular hemolysis with peripheral vein administration in humans [7,8].

(5) Patients on Peritoneal dialysis (PD) may not have abdominal symptoms with peritonitis. If

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febrile, a cell count and culture of peritoneal fluid should be sent. Cell count of more than 100 is significant with PD compared to 250 with spontaneous bacterial peritonitis. PD peritonitis commonly follows invasive interventional procedures (e.g., colonoscopy, hysteroscopy, cholecystectomy). Systematic review recommends prophylactic use of intravenous (IV) ampicillin plus an aminoglycoside, with or without metronidazole [9].

(6) Always check for presence of red blood cells (RBCs) casts or dysmorphic RBC in the urine in patients with AKI. If elevated, this should raise suspicion for crescentic glomerulonephritis [10,11]. This requires urgent consultation. Crescentic glomerulonephritis/rapid progressive glomerulonephritis (RPGN) is usually caused by one of the three following mechanisms: anti-GBM antibody disease with or without pulmonary hemorrhage, pauci-immune glomerulonephritis, or severe immune complex glomerulonephritis [10].

(7) Denosumab given for osteoporosis can cause severe hypocalcemia in chronic kidney disease stage 4 and 5, which could present three weeks to months following denosumab causing fatal arrhythmias. These patients need vitamin D and calcium replacements with regular follow-up of serum calcium levels [12].

(8) Calciphylaxis should be suspected in higher CKD stages/End stage Kidney disease patients presenting with painful, non-ulcerating, subcutaneous nodules or plaques, nonhealing ulcers, and/or necrosis. Lesions are most commonly seen in abdomen, thigh, and any areas of increased adiposity. Histologically by calcification of dermal arterioles [13].

(9) Work up for resistant hypertension should be sent only if patient is on three or more drugs at adequate dosage, out of which one should be a diuretic. Resistant hypertension is defined in the 2008 American Heart Association and in the 2013 Guidelines of the European Societies of Hypertension and Cardiology as blood pressure that remains above goal in spite of concurrent use of three antihypertensive agents of different classes. Use of ultrasound doppler to detect renal artery stenosis has poor sensitivity, especially if the patient is obese or has increased bowel gas. It is time consuming, technically difficult (particularly in large patient), and is operator dependent [14].

(10) Sickle cell disease can lead to a variety of both glomerular and tubular pathologies, and been estimated to develop in anywhere from 5% to 40% of cases. Tubular secretion of creatinine is higher in sickle cell disease and patients usually have lower muscle mass, which tends to falsely lower measured serum creatinine levels [15,16]. In these patients a 24-hour urine for creatinine and protein is preferred over serum creatinine. They can also present with microalbuminuria or macroalbuminuria and need nephrology referral as soon as albumin is detected in urine [17].

Disclosure statement

The authors alone are responsible for the content and writing of this paper. Dr. Venkataraman Palabindala, is currently an Assistant professor at the University of Mississippi medical center; the remaining authors have no disclosures or conflict of interest to report. This study did not receive any research funding.

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