Renal-Friendly ORS: Evidence-Based Personalisation In Acute And Chronic Kidney Disease

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Abstract

Oral Rehydration Solution (ORS) is a life-saving therapy widely used for the management of dehydration, especially in diarrhoeal illnesses. However, in patients with acute kidney injury (AKI) or chronic kidney disease (CKD), including those with hypertension, oliguria, or fluid overload, standard ORS formulations may cause harm. These patients often present with impaired sodium and potassium handling, low urine output, and increased risk of volume overload or hyperkalaemia. This review summarises the limitations of conventional ORS in this population and provides evidence-based, stage-wise recommendations for renal-safe ORS formulations that consider fluid status, serum potassium levels, GFR, and comorbid conditions like hypertension.

Keywords: Acute kidney injury, Chronic kidney disease, Electrolyte imbalance, Fluid overload, Hyperkalemia, Hypertension, Potassium, Sodium

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Introduction

ORS is one of the simplest yet most impactful interventions in medicine. Developed to combat fluid losses in diarrhoeal illness, especially in children, ORS leverages the sodium-glucose co-transporter mechanism (SGLT1) in the small intestine to promote water and electrolyte absorption.[1] According to the revised WHO formulation (2006), the standard ORS contains 75 mEq/L sodium, 20 mEq/L potassium, 75 mmol/L glucose, 65 mEq/L chloride, and 10 mmol/L citrate, with a total osmolarity of approximately 245 mOsm/L.[2] These concentrations are designed for the general population to rapidly correct dehydration without causing hypernatraemia or osmotic diarrhoea.

However, the same formulation may be problematic when used in individuals with compromised renal function. In patients with AKI or CKD, especially stages 4 and 5, the kidneys' ability to handle sodium, potassium, and water loads is impaired. These patients frequently face overlapping challenges such as vomiting, poor oral intake, oliguria or anuria, electrolyte imbalances, and concurrent hypertension.[3,4] In such scenarios, the indiscriminate use of standard ORS could lead to worsening of fluid overload, hyperkalaemia, or uncontrolled blood pressure. Therefore, a nuanced, personalized approach to ORS therapy is warranted in the setting of renal dysfunction.

Fluid and Electrolyte Homeostasis in AKI and CKD

The kidneys play a pivotal role in regulating homeostasis of fluids, sodium, potassium, and acid-base status. A healthy kidney filters approximately 180 litres of plasma and handles over 25,000 mEq of potassium daily, reabsorbing, and excreting electrolytes precisely to meet physiological needs[5]. However, in patients with reduced glomerular filtration rate (GFR) due to AKI or progressive CKD, these finely tuned mechanisms falter. Sodium retention may occur due to impaired filtration and reabsorption dynamics, while potassium excretion is markedly reduced once GFR falls below 30 mL/min/1.73 m^2. The capacity to eliminate free water is also diminished, contributing to hyponatraemia and fluid overload. Additionally, many CKD patients- especially in stage 5- develop oliguria (urine output < 400 mL/day) or even anuria, severely limiting their ability to clear daily fluid and electrolyte intake.[6,7]

These alterations set the stage for complications when standard ORS is administered without modifications. Even a modest ORS intake (e.g., 1 litre containing 20 mEq of potassium) could be hazardous for a patient with minimal or no urinary output. Moreover, sodium intake exceeding 100–150 mEq/day can contribute to hypertension and volume expansion, particularly in salt-sensitive individuals or those with pre-existing oedema.

Clinical Implications of ORS Use in Renal Patients

To better understand these physiological mismatches, Table 1 presents a comparative view of normal electrolyte handling versus changes in AKI and CKD, along with their

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