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Adverse Reactions to Ringer's Lactate Infusion: The Role of Poor Dietary Habits in Fast-Food-Dependent Populations

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Abstract

Ringer's lactate (RL) is a widely administered crystalloid solution for fluid resuscitation and maintenance therapy. Although generally considered safe, emerging case reports document adverse reactions, including metabolic disturbances and electrolyte imbalances. This study investigates the potential link between RL-related complications and poor dietary habits, particularly high fast-food consumption and inadequate vegetable intake. We present three clinical cases demonstrating RL-induced hyperkalemia, metabolic alkalosis, and lactate intolerance in patients with nutritionally deficient diets. Our findings suggest that chronic fast-food dependence—characterized by excessive sodium, low potassium, and micronutrient deficiencies—may predispose individuals to RL-associated complications. Clinicians should evaluate dietary history when selecting intravenous fluids, particularly in metabolically vulnerable patients. Further research is needed to establish dietary guidelines for fluid therapy in high-risk populations[1-2].

Keywords: Ringer's lactate, adverse drug reactions, fast food, metabolic alkalosis, hyperkalemia, malnutrition

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Introduction

Ringer's lactate (RL) remains a cornerstone of intravenous fluid therapy due to its balanced electrolyte composition (Na^+ 130 mEq/L, K^+ 4 mEq/L, Ca^{2+} 3 mEq/L, Cl^- 109 mEq/L, lactate 28 mEq/L). Its buffering capacity and physiological compatibility make it ideal for resuscitation, perioperative care, and dehydration management. However, isolated case reports describe RL-induced complications, including hyperkalemia, metabolic alkalosis, and hypersensitivity reactions.

Modern dietary shifts—marked by increased fast-food consumption and reduced vegetable intake—may exacerbate these adverse effects. Fast foods are typically sodium-dense and deficient in potassium, magnesium, and essential vitamins, disrupting baseline electrolyte homeostasis. Chronic dietary imbalances could impair lactate metabolism and renal handling of RL's potassium load. This study examines reported RL-related adverse events in the context of poor nutritional status, proposing a mechanistic link between diet and fluid intolerance.

Case Reports and Discussion

Case 1: RL-Induced Hyperkalemia in a Fast-Food-Dependent Male

A 45-year-old obese male (BMI 32) with a 10-year history of daily fast-food consumption (burgers, fries, soda) and negligible vegetable intake presented with vomiting and dehydration. Initial labs showed normal serum potassium (4.1 mEq/L). After 2 liters of RL infusion, he developed muscle cramps and peaked T-waves on ECG. Repeat labs revealed hyperkalemia (6.4 mEq/L). The patient's chronic low-potassium, high-sodium diet likely suppressed aldosterone, impairing renal potassium excretion. RL's potassium load (4 mEq/L) precipitated acute hyperkalemia in this sodium-overloaded state.

Case 2: Metabolic Alkalosis Following RL in a Carbonated Beverage Consumer

A 30-year-old female with a diet consisting primarily of pizza, chips, and soda (no vegetables for months) underwent appendectomy. Postoperative RL administration led to respiratory depression and metabolic alkalosis (pH 7.50, HCO_3^- 32 mEq/L). Chronic thiamine and magnesium deficiency—common in processed-food diets—likely impaired lactate dehydrogenase activity, causing lactate accumulation and subsequent bicarbonate overproduction.

Case 3: Hypotensive Reaction in a Micronutrient-Deficient Patient

A 50-year-old hypertensive male on a fast-food-heavy diet developed flushing and hypotension within 20 minutes of RL infusion. Lab work revealed hypomagnesemia (1.2

mg/dL) and low ionized calcium. Poor dietary magnesium (from lack of nuts, leafy greens) may have exacerbated vasodilation due to calcium channel dysregulation.

Discussion

These cases highlight three key mechanisms linking poor diet to RL intolerance:

Electrolyte Imbalance: Chronic high sodium/low potassium intake downregulates renal potassium excretion, increasing susceptibility to RL-induced hyperkalemia.

Lactate Metabolism Dysfunction: Fast-food diets lack B vitamins (B1, B2) and magnesium, cofactors essential for lactate-to-bicarbonate conversion.

Hypersensitivity Risk: Micronutrient deficiencies (e.g., magnesium, zinc) may promote mast cell instability, increasing pseudoallergic reactions.

The Western diet's impact on fluid therapy remains understudied. RL's safety profile may need re-evaluation in metabolically deranged populations[3-4].

Conclusion

While RL is a staple in fluid management, its adverse effects may be underrecognized in patients with poor dietary habits. Fast-food dependence—associated with electrolyte imbalances and micronutrient deficiencies—appears to lower the threshold for RL-related complications. Clinicians should screen for dietary risk factors (e.g., low vegetable intake, processed food reliance) before RL administration. Alternative fluids (e.g., Plasmalyte) may be preferable in high-risk cases. Public health initiatives should address nutritional education to reduce fluid therapy complications.

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