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SHORT REVIEW

Bone and metabolic complications of urinary diversions☆

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Abstract Hyperchloremic metabolic acidosis is a complication of urinary diversion using ileum or colon. Its prevalence ranges from 25% to 46% depending on the procedure used and renal function of the patient. It is a consequence of intestinal fluid and electrolyte exchange between intestinal mucosa and urine. The main mechanism is absorption of ammonium and chloride from urine. Long-term chronic metabolic acidosis in these patients may lead to impaired bone metabolism and osteomalacia. Regular monitoring of pH, chlorine, bicarbonate, and calcium–phosphorus metabolism is therefore essential for early diagnosis and treatment.

PALABRAS CLAVE
Acidosis metabólica;
Hipopotasemia;
Derivación urinaria;
Neovejiga;
Conducto ileal

Complicaciones metabólicas y óseas de las derivaciones urinarias

Resumen La acidosis metabólica hiperclorémica es una complicación de las derivaciones urinarias que utilizan ileon o colon, y su prevalencia oscila entre el 25% y 46% de los casos, dependiendo de la técnica utilizada y de la función renal del paciente. Es una consecuencia del intercambio hidroelectrolítico entre la mucosa intestinal y la orina, siendo su mecanismo principal la absorción del amonio y cloruro de la orina. A largo plazo la acidosis metabólica crónica puede conllevar una alteración del metabolismo óseo y producir osteomalacia, por lo que la monitorización del pH, cloro, bicarbonato y metabolismo fosfocálcico es fundamental para un diagnóstico y tratamiento precoz.

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Introduction

The main indication for urinary diversion is radical cystectomy for bladder cancer. Different segments of the gastrointestinal tract may be used for urinary reconstruction. Ileal and colonic segments are the most commonly used. Stomach and proximal bowel are only used when the rest of the intestinal mucosa is not available, as occurs in some patients with inflammatory bowel disease, short bowel, or a history of pelvic radiation. These segments are used to replace the bladder, either as a conduit to drain urine to the abdominal wall, a urinary stoma, or a reconstruction to form a neobladder, i.e. with diversion to the native urethra.

Urinary diversions may be divided into heterotopic and orthotopic depending on the urine outflow tract. In heterotopic diversions, urine will flow out through a duct different from the urethra (skin, rectum, etc.). Heterotopic diversions may in turn be continent or incontinent. The most commonly used diversion is ureteroileostomy, where an ileal conduit is created: ureters will be attached to one end, while the other end will be taken to the skin to construct a stoma. In orthotopic diversions, the bladder is replaced respecting the natural excretory route, i.e. the urethra. A neobladder is created to store urine, which is passed in a voluntary, controlled way (Fig. 1).

The bowel mucosa differs from the urinary mucosa in its semipermeable and metabolically active nature, so that when it comes into contact with urine, metabolic changes may occur through different electrolyte transport and diffusion mechanisms.

The incidence of water and electrolyte disorders has been estimated to range from 21% to 48% in the different series reported.[2–5]

Complications derived from bowel resection itself are very few, due to the use of short bowel segments. Malabsorption is very rare with ileal resections shorter than 60 cm,[6] but vitamin B deficiency$_{12}$ may occur in the long term in up to 21% of patients.[7] That is to say, the risk of the occurrence of these water and electrolyte disorders depends to a large extent on the procedure used, which determines the surface size and contact time of urine with the bowel mucosa.[8]

General physiopathogenic concepts

Each gastrointestinal tract segment (stomach, jejunum, ileum, and colon) is physiologically different in type of solute transport, and different changes occur depending on the segment and length used (Table 1). It should also be noted that the absorption capacity of the bowel segment decreases with time, and atrophic areas occur in the mucosa as a defense mechanism against the different osmolarity, pH, and solute concentration of urine.[6]

On the one hand, transcellular electrolyte and solute transport occurs in the bowel through transmembrane proteins and electrochemical gradients, co-transporters, and exchange channels. Transcellular sodium (Na^+) and hydrogenion (H^+) transport occurs through the Na^+/K^+/ATPase pump and the Na^+/H^+ exchanger in the luminal membrane of the small and large bowel. This process makes it possible for low intracellular sodium levels to be maintained, establishing the electrochemical gradient required for solute transport. This mechanism promotes Na^+ absorption from the intestinal lumen against the concentration gradient existing between the lumen and plasma, with subsequent H^+ secretion into the lumen. The efficiency of Na^+ absorption increases distally.[10] However, potassium transport in the bowel is passive, along

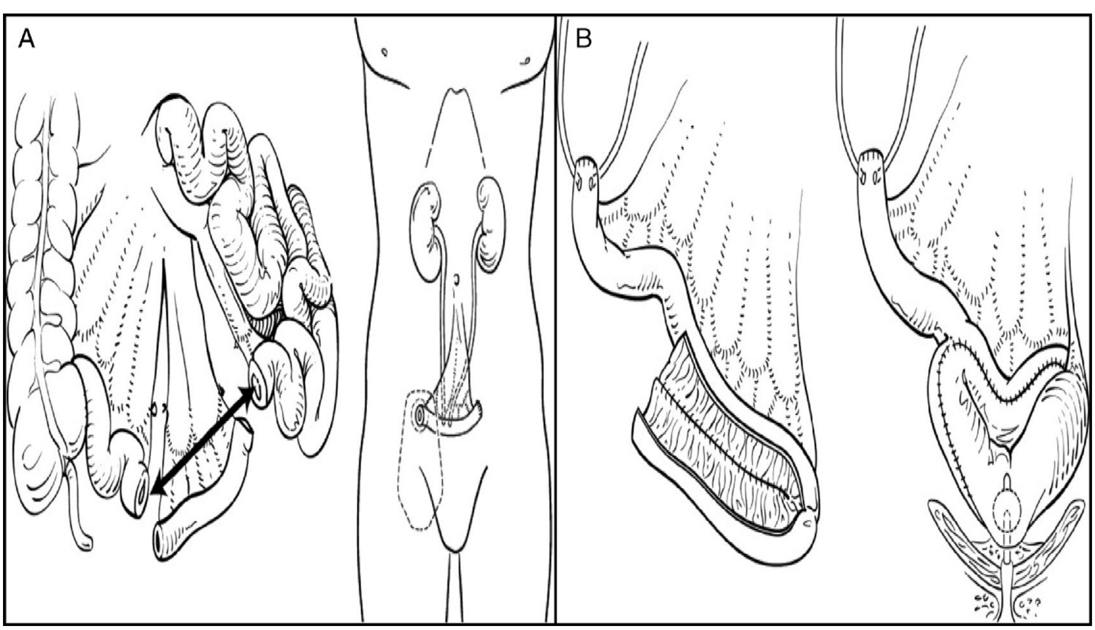

Figure 1 (A) Ileal conduit. (B) Orthotopic ileal neobladder. Taken from Parekh and Donat.[1]