Metabolic and Nutritional Consequences of Urinary Diversion Using Intestinal Segments to Reconstruct the Urinary Tract

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KEYWORDS

• Urinary diversion • Urinary reservoirs • Metabolic acidosis • Ileum • Colon
• Metabolic complications • Nutritional complications

KEY POINTS

- The bowel has been used in the urinary tract for many years.
- Urinary diversion with ileum or colon can lead to concentration defects or metabolic acidosis.
- Urinary diversion with jejunum or gastric segments is not recommended for use in urinary tract reconstruction due to metabolic consequences associated with their use.
- Nutritional consequences of urinary diversion with ileum or colon include vitamin B_{12} deficiency, osteoporosis, fat malabsorption, urinary calculi, and ammoniagenic encephalopathy.

INTRODUCTION

Intestinal segments in various forms have been used to reconstruct the urinary tract since the mid-1800s, with most initial reports focusing on the technical aspects of these procedures.[1] In the early twentieth century before 1950, the primary means of urinary diversion (UD) after cystectomy for any indication was bilateral ureterosigmoidostomy. Of all the past and present forms of UD, this particular technique created a conduit where the greatest amount of intestine was exposed to the excreted urine and, to some degree, where it was exposed to the urine for the greatest amount of time.[1,2] Because the metabolic conditions created by UD are directly and proportionally related to these factors, metabolic conditions are seen most commonly in ureterosigmoidostomy patients. In 1950, 2 physicians from the Mayo Clinic, Ferris and Odel,[3] were the first to recognize and publish on the condition of hyperchloremic metabolic acidosis.[3,4]

In the 1950s, there was a great interest in the etiologic factors of this metabolic acidosis and multiple theories were espoused by many of the leaders in urology of that generation. These theories included a renal acidifying defect from ureteral obstruction or pyelonephritis, secretion of bicarbonate (HCO_3^-) by the bowel into the urine, and the generation of ammonia (NH_3) from urea by the action of urease producing organisms in the urine, which was then reabsorbed.[3–5] All 3 of these theories were at least in part accurate and partially explain the abnormalities seen. It was at

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approximately the same time that Eugene Bricker first reported on the ileal conduit[6]. With a lower incidence of surgical and metabolic complications with the ileal conduit, UD via ureterosigmoidostomy was largely abandoned so that the issue of the hyperchloremic metabolic acidosis became a moot point.[7]

In the 1980s, there was a general recognition that patients with ileal conduit UD had significant issues over the long term. With the general acceptance of clean intermittent catheterization techniques, in many children who had neuropathic bladders that had been diverted with conduits, these were undiverted to an augmented bladder with bowel.[8] In addition, driven primarily by body image concerns after cystectomy, both continent cutaneous diversions (eg, Indiana Pouch, Kock pouch) and techniques for continent orthotopic bladder replacements were developed.[9–11] The continent orthotopic replacement was at least in part enabled by the refinement of radical prostatectomy and the ability of urologists to create a urethral-vesical anastomosis.[11] These new continent diversions created systems in which, once again, urine was exposed to large amounts of bowel surface area for protracted periods of time sufficient for the movement of urinary solutes across the intestinal mucosa, thereby leading to electrolyte abnormalities. In addition, the utilization of relatively large segments of ileum to create these diversions resulted in predictable nutritional complications.[12–14]

METABOLIC DERANGEMENTS

Normal urothelium is a highly impermeable barrier to urinary solutes. This property of urothelium allows the kidneys to excrete urinary waste products in concentrations vastly different than that seen in the serum. Specifically, urine is highly concentrated, has a low sodium (Na) content, high potassium content, and it is generally acidic with negligible bicarbonate levels. Whereas normal urine is acidic, normal feces are alkaline. The results seen with various forms of UD are a predictable result of the known physiology of various intestinal segments and the known concentrations of the solutes of urine.[13–16]

Concentration Defect

All forms of UD result in a concentrating defect that is generally well compensated but universally noticeable.[17] Normal urine generally has an osmolality of 500 to 850 mOsm per liter, whereas normal serum and tissue osmolality is around 290 mOsm per liter.[18] Generally, intestinal mucosa is highly permeable to water. As such, when concentrated urine is exposed to a bowel segment, there is movement of water into the intestinal lumen, which negates, in part, the body's ability to preserve volume status. Clinically, this partly accounts for the nocturia seen in these patients and it also results in a chronic prerenal state with an elevated blood urea nitrogen (BUN) to creatinine ratio. Ileum and colon are quite similar in this regard, with jejunum being more severe because of its more loose intraepithelial junctions. Jejunal conduits also create a salt wasting condition, which further exacerbates the volume depletion.[16]

Ileum and Colon: Hyperchloremic Metabolic Acidosis

The apical membrane surface of both ileum and colon contains antiports, which are specific to certain electrolytes and exchange anions or cations in equimolar amounts. Normal ileum and colon absorb urinary chloride and excrete bicarbonate into the intestinal lumen. In the setting in which the urine has no bicarbonate but moderate amounts of chloride, bicarbonate moves into the lumen of the UD while chloride(Cl) is absorbed, thus initiating the observed acidosis.[19]

When an acidosis initially develops, the kidneys respond by excreting acid as free hydrogen (H) ions, which are buffered with phosphate (O4P3-) or, to a lesser degree, sulfate (SO4(2-)) in the urine. This cannot continue indefinitely or the body would become deplete in phosphate (O4P3-), which is an essential part of bone mineralization.[17] The body adapts by creating another buffer in the distal tubule, ammonia, which is very avid for free hydrogen ions. This is readily generated in the kidney by converting glutamine to glutamate, and by deaminating glutamate to 2-oxo-glutarate^{2-} by gamma-glutamyl transferase in the proximal tubule. As abundant amounts of ammonia is generated, it passes through the loop of Henle and distal tubule, where some is reabsorbed and some of it binds to free hydrogen ions to become ammonium (NH_4^+).[20,21]

However, this leads to a second cause of acidosis. The ileum and colon also possess sodium-potassium (Na+/K+) antiports, which in normal circumstances allow for the absorption of sodium. In a patient with a UD, ammonium is reabsorbed in equimolar amounts to the amount of sodium excreted. The net effect of these 2 antiports in the patient with UD is the reabsorption of ammonium and chloride and a resulting hyperchloremic metabolic acidosis.[21,22] Ammonia reabsorption has been found to contribute more to the development of systemic acidosis than either the secretion of bicarbonate or the reabsorption of organic acids.[23–25]