We Can Rebuild It: Reconstructive Solutions for Structural Urologic Diseases

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Bladder augmentation and urinary diversion have become standard of care as surgical treatments for structural and functional disorders affecting the bladder, both in children and adults. With improved medical care, long-term survival of these patients is expected. Common medical problems that can occur such as metabolic side effects including acid-base imbalances and nutritional issues need to be anticipated and addressed. In addition, surgical problems caused by impaired urinary drainage, namely stones and urinary tract infections, and mechanical factors related to catheterizable channels and continence also may compound postoperative management. The risk of malignancy after bladder augmentation and substitution, and appropriate surveillance for this, remains to be clearly defined.

Key Words: Bladder, Augmentation, Urinary diversion, Metabolic complications

Reconstructive surgery is needed after bladder extirpation for either congenital conditions or acquired bladder abnormalities. Pediatric conditions such as myelomeningocele or exstrophy affect the structure and function of the bladder, threatening kidney function. This requires pre-emptive medical management from birth, and often these children require surgical remedies including bladder rehabilitation with augmentation or complete replacement. In adults, oncologic indications for removal of the bladder include both muscle invasive bladder cancer and recurrent nonmuscle invasive bladder cancer refractory to conservative treatment.[1] Children may also need radical cystectomy for malignancies, such as rhabdomyosarcoma. In addition, other nononcologic causes may occasionally result in the removal of the bladder and, consequently, a urinary diversion, such as severe neuropathic bladder, treatment refractory overactive bladder, interstitial cystitis, radiation cystitis, and tuberculosis.[1]

SURGICAL APPROACHES

Broadly, reconstruction of the bladder includes augmentation and urinary diversions. In benign conditions with at least part of the bladder available, its capacity can be enlarged (augmentation enterocystoplasty) using a bowel segment. If the bladder is unsuitable for use or has to be removed, as in malignancies, urinary diversion is necessary, which can be either an incontinent or a continent type. In incontinent urinary diversions, urine is diverted to a reservoir constructed from a bowel segment, and the bowel segment is then attached to the body wall for the urine to drain into an external collecting system. In contrast, in continent urinary diversions, urine is collected into a reservoir (neobladder) created from intestine.

Both bladder augmentation and continent diversion require a channel to empty the bladder at periodic intervals and a continence mechanism. If the native urethra is available and usable, the augmented bladder or continent diversion can be emptied through the urethra by intermittent catheterization or rarely by volitional voiding. A bladder made entirely of bowel and attached to the native urethra is termed an "orthotopic neobladder." When the urethra is unavailable or has been removed, a catheterizable channel can be created using a tubular structure such as the appendix (Mitrofanoff principle).[2] Alternatives to the appendix include ureter, fallopian tube, or tubularized segments of small or large bowel.[3] The latter procedure involves isolating a 2-cm segment of bowel, opening it on its antimesenteric border and reconfiguring it lengthwise over a catheter, thus creating an "appendix" (Monti technique).[3] These catheterizable channels connect the augmented or neobladder to the abdominal wall, where they are hidden in the umbilicus or a fold of skin in the lower abdominal wall. To prevent urinary leak through the channel, an antireflux mechanism is also created by plicating it within the bowel or bladder wall.

The above principles can be used in various combinations, and several excellent surgical techniques have evolved over the years, allowing the urological surgeon to choose the appropriate bladder reconstruction to suit the individual needs of any patient. There is an ongoing debate concerning the optimal type of urinary diversion after bladder removal. Various factors including disease stage; patient age, gender, and socioeconomic status; kidney and hepatic function; availability of healthy bowel; adequate sphincter function; and even surgeons' experience all play an important role in the clinical decision making.[4]

With advancement of surgical technique, improved safety profile, and increased number of skilled surgeons, urinary diversion has become the standard treatment for many structural and functional bladder disorders.[4] Despite widespread utilization in the past years, urinary diversions are not a benign reconstructive effort. Depending on the bowel segment used to construct the diversion, patients may confront long-term metabolic complications including acid-base and electrolyte abnormalities, bone demineralization, increased urolithiasis, altered

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absorptive capacity, and deterioration of kidney function.[5] With increased utilization and survival after urinary diversion, it is likely that such long-term complications will be more frequently encountered.[4-6] Keeping this in mind, the goal of this review is to provide an overview of metabolic abnormalities, their causes, evaluation, and management. The focus of the discussion is from the perspective of nephrologists and internists who will often be faced with managing these patients chronically in the outpatient setting and acutely during hospitalization for this or unrelated issues.

METABOLIC ABNORMALITIES

Metabolic abnormalities are common in both continent and incontinent type of diversions. The extent of metabolic abnormalities depends on a number of factors including type and length of bowel segment used and the length of time that urine is in contact with bowel.[6] Virtually, all types of bowel segments can be used in urinary diversions, with ileum and colon being the most commonly used segments.[6]

Acid-Base and Electrolyte Abnormalities

Acid-base and electrolyte abnormalities after urinary diversion depend on the bowel segment used. Stomach and jejunum can be used for bladder augmentation or urinary diversion,[4,6] yet both these bowel segments are considered secondary options because they tend to produce more severe acid-base and electrolyte disorders.[4,6] Indications for using stomach or jejunum include radiation exposure to the bowel, presence of adhesions limiting surgical exposure, and inflammatory bowel disease.[4,6]

CLINICAL SUMMARY

- Use of bowel segments in bladder replacement or augmentation are accompanied by a variety of metabolic and acid-base side-effects.
- The metabolic problems are specific to the type of bowel segment used in reconstruction.
- These abnormalities are well recognized, and can be effectively treated when diagnosed early.

Use of stomach segments has some advantages because the gastric mucosa produces less mucus than more distal bowel segments and is less permeable to urinary solutes, but severe dysuria and hematuria may occur. This complication has led to a significant decrease in use of stomach for lower urinary tract reconstruction in recent years. Urinary diversion with stomach can produce a metabolic alkalosis from secretion of HCl into the urine by the parietal cells through the H^+/K^+ ATPase antiporter, which leads to renal wasting of potassium and hypokalemia.[6,7] Treatment with H_2 blockers and proton pump inhibitors has considerably improved outcomes for patients who have had gastrocystoplasty or reconstruction of the urinary tract using stomach such that use of this tissue remains an option if other surgical strategies cannot be used.[8]

Jejunum can also be used in incontinent urinary diversion although this segment is very rarely used because of severe metabolic complications that may ensue.[4,6] The exact mechanism of electrolyte abnormalities from jejunal segments is not well understood; however, it is believed that the acid-base and electrolyte abnormalities are because of leaky intercellular junctions in the jejunum.[6] Patients who have had urinary reconstruction with a segment of jejunum may develop the "jejunal conduit syndrome." In this setting, patients are acutely ill with evidence of volume depletion likely from urinary losses of sodium, chloride, and bicarbonate. Laboratory data are often notable for hyponatremia, a nonanion gap acidosis, hyperkalemia, and evidence of acute kidney injury. The hyponatremia occurs from volume depletion, oral fluid intake, and appropriate ADH release. In the urologic literature, a "hypochloremic" metabolic acidosis is often described; however, the hypochloremia is seen in concert with hyponatremia and does not represent an anion gap acidosis. Hyperkalemia is also commonly seen, particularly in patients with reduced kidney function at baseline. The constellation of abnormalities seen in the jejunal conduit syndrome improve with administration of saline and are subsequently managed with oral sodium chloride or sodium bicarbonate supplements often indefinitely.[6,9-11]

The most commonly used bowel segments for urinary diversions are terminal ileum and colon, both of which may lead to a nonanion gap metabolic acidosis.[6,12] In elegant studies first using dogs with interposition of ileum in 1 ureter and then kinetic studies using sheets of enterocytes, McDougal and others have clarified the pathophysiology of the metabolic acidosis that may occur in the setting of urinary diversion to an intestinal segment.[13,14] Ammonium produced by the kidney, which has a similar ionic radius to Na^+, competitively inhibits the Na^+/H^+ antiporter, sodium-hydrogen exchanger 3, that is expressed on the apical membrane of the enterocytes. In addition, bicarbonate is lost in the urine in exchange for chloride through the Cl^-/HCO_3^- antiporter.[13,14] The net result is an increase in ammonium chloride reabsorption, and this leads to a nonanion gap metabolic acidosis.[12,14,15]

Most individuals with urinary diversion will not have significant metabolic abnormalities, and current series suggest that only approximately 10% of patients exhibit acid-base and metabolic abnormalities.[6,11] In general, continent urinary diversions lead to a greater degree of metabolic derangement than incontinent urinary diversions. For example, ileal and colonic incontinent diversions have a 10% to 15% risk of acid-base abnormalities, whereas continent diversions may produce these abnormalities in up to 45% of patients.[11] This is primarily because of the increased contact time between urine and the bowel reservoir in continent diversions.[6,11]

Medical practitioners caring for this special population must recognize that the signs and symptoms of acid-base and electrolyte abnormalities are nonspecific and can be