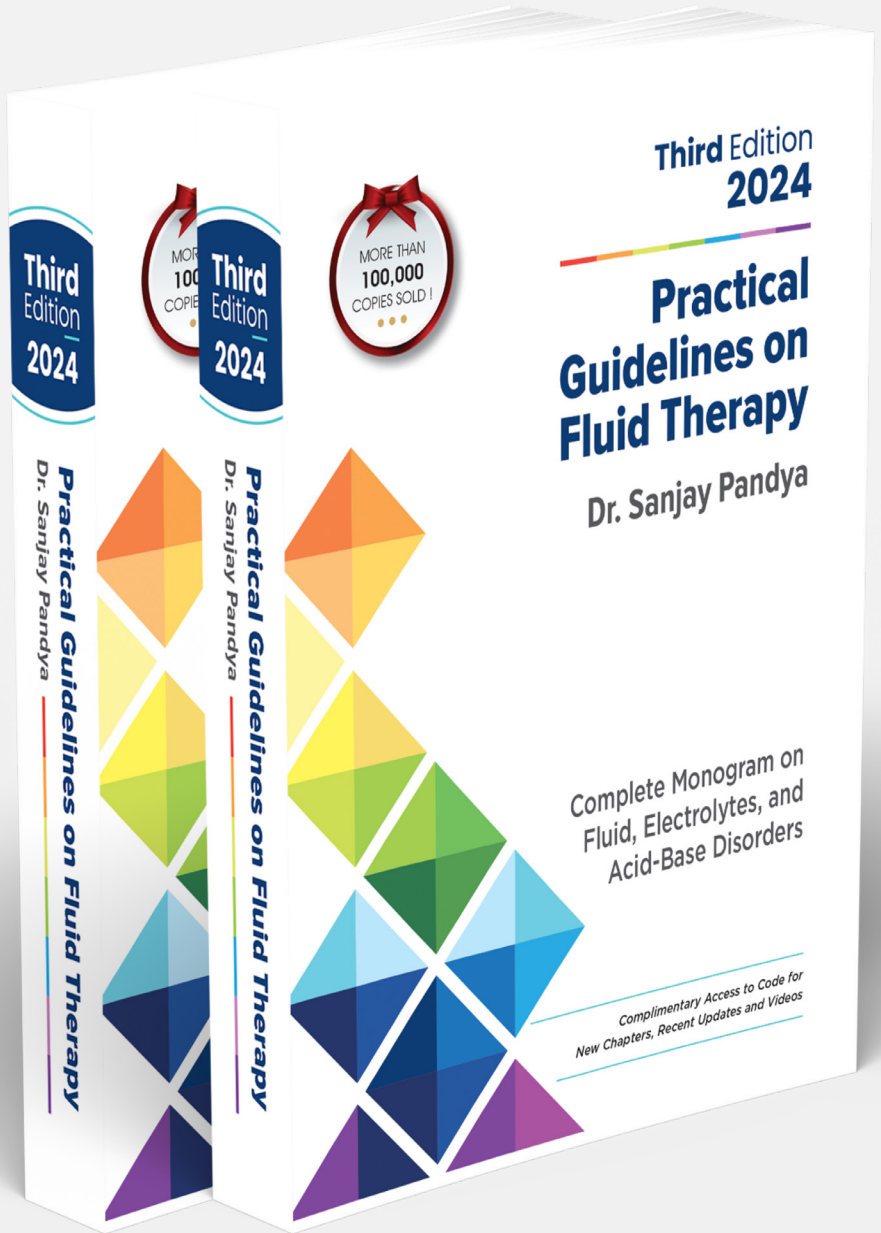
Chapter 47:
Urinary Diversion



To get a copy of the book, visit: **www.fluidtherapy.org**

47 Urinary Diversion

Urinary diversion is a surgical procedure that creates a new route to reroute urine flow from the normal pathway to an alternate path for elimination from the body.

INDICATIONS

Urinary diversion becomes necessary when the bladder can no longer safely store urine due to various conditions, such as:

- Bladder carcinoma: Surgical removal of the bladder for carcinoma.
- Traumatic injuries: Extensive damage to the bladder, urethra, or pelvis.
- Medical conditions: Malfunctioning bladder due to neurologic bladder dysfunction.
- Congenital defect: Bladder exstrophy is a congenital condition in which the bladder is located outside the body.
- Miscellaneous causes: Severe radiation injury to the bladder, intractable urinary incontinence, or vesicovaginal fistulae.

METHODS OF URINARY DIVERSION

There are several types of urinary diversion procedures, and the choice of procedure depends on the specific medical condition, extent of urinary tract involvement, and personal preference [1].

Urinary diversions are classified into two types [2]:

1. Incontinent diversions, where urine is redirected into an intestinal segment and continuously drained into an external collection bag (e.g., ileal conduit).
2. Continent diversions, where urine is collected and stored within the body, requiring either self-catheterization (e.g., catheterizable ileal pouch) or voluntary voiding to empty the reservoir (e.g., orthotopic neobladder).

Four common methods of urinary diversion includes:

A. Ileal conduit urinary diversion

The most commonly performed and simplest type of incontinent urinary diversion with the fewest surgical complications involves attaching the ureters to a segment of the ileum, creating a stoma on the abdomen for continuous urine drainage into an external collecting bag. The drawbacks of ileal conduit urinary diversion include the need for an external collecting bag for continuous urine drainage, potential changes in body image, and the possibility of leakage or odors.

B. Ileal pouch urinary reservoir

In this continent cutaneous diversion method, a pouch is created from portions of the ileum to store urine within the abdomen. The ureters are redirected to drain into this pouch, and intermittent self-catheterization is required several times a day to empty the urine from the pouch. The advantages of the ileal pouch reservoir surgery include not requiring an external bag for urine collection and eliminating the issue of odor associated with external urine drainage.

C. Orthotopic neobladder

This is a form of continent urinary diversion in which a surgically created reservoir is made from a segment of the intestine and placed in the same anatomical position as the original bladder, connected to the urethra. The neobladder closely resembles the storage function of a urinary bladder, allowing patients to void normally by increasing intra-abdominal pressure and significantly improving their quality of life and self-image after radical cystectomy. If voiding is not adequate despite the aforementioned efforts, patients may need to perform self CIC (clean intermittent catheterization).

D. Ureterosigmoidostomy

This urinary diversion procedure involves connecting the ureters to the sigmoid colon, allowing urine to be diverted into the colon and eliminated with bowel movements.

Ureterosigmoidostomy, which was once a common urinary diversion procedure, is now rarely used and considered a last resort option due to potential risks, such as electrolyte imbalances, urinary tract infections, worsening of kidney function, and the risk of colon malignancy. Safer and more advanced methods of urinary diversion have largely replaced it. However, due to advantages such as providing urinary continence without the need for a stoma and external appliances, simplicity, and the relatively quick performance of the procedure, ureterosigmoidostomy is still performed in some complex pediatric cases in resource-limited settings [3].

PATHOPHYSIOLOGY OF METABOLIC AND ELECTROLYTE DISTURBANCES

The factors that determine metabolic derangements and electrolyte imbalances in urinary diversion include the type of bowel segment used for the anastomosis, the length and surface area of the bowel segment exposed to urine, and the duration of contact time between urine and the bowel [4].

Urinary diversions involve using a part of the bowel that is not physiologically designed to handle and withstand urine's fluid composition [5]. Meta-

bolic and electrolyte imbalances are more common in traditional ureterosigmoidostomy compared to other newer methods of urinary diversion. In ureterosigmoidostomy, urine containing chloride, sodium, and ammonium is diverted to the sigmoid colon, which acts as a reservoir. Due to the sigmoid colon serving as a large-sized reservoir and the prolonged transit time of urine in the colonic segment, which leads to a longer contact period in the colon, there is a significant exchange of urinary electrolytes with plasma through the colonic mucosa, resulting in fluid and electrolyte disturbances.

Common abnormalities encountered in ureterosigmoidostomy include metabolic acidosis, hypokalemia, hyperammonemia, diarrhea, urolithiasis, and metabolic bone diseases.

A. Metabolic acidosis

Metabolic acidosis commonly occurs in urinary diversions when urine comes into contact with the bowel wall, leading to the reabsorption of urinary ammonia, hydrogen, and chloride in exchange for the excretion of sodium and bicarbonate ions, which contributes to its development [4].

The absorption of chloride-rich urine by the luminal anion exchange pump of the colonic mucosa leads to the secretion of HCO_3 in exchange, causing bicarbonate loss in the bowel and resulting in hyperchloremia and metabolic acidosis.

An additional mechanism contributing to the development of hyperchloremic acidosis in urinary diversion is the absorption of ammonium [4]. When urine comes into contact with the colonic mucosa, fecal bacteria convert urinary ammonia into a large amount of ammonium ions. This leads to excessive intestinal absorption of urinary ionized ammonium, along with chloride as ammonium chloride, causing a loss of bicarbonate and a gain of chloride, resulting in hyperchloremic metabolic acidosis [6]. Loss of bicarbonate due to diarrhea (due to colon irritation by urine) also contributes to hyperchloremic metabolic acidosis.

B. Hypokalemia

Hypokalemia is more common in sigmoid diversions compared to ileal diversions, possibly because the colon has a lower capacity for potassium absorption than the ileum. In ureterosigmoidostomy, hypokalemia develops due to both loss of potassium in the colonic mucosa (secretion) and renal wasting [7]. Renal potassium wasting plays a relatively major role in potassium depletion, and it occurs due to acidosis, volume depletion, and activation of the renin-angiotensin-aldosterone system [8]. Diarrhea due to colon irritation by urine leads to direct loss of potassium and contributes to hypokalemia.

C. Metabolic bone diseases

In patients with urinary diversion, the long-term risk of metabolic bone diseases is a concern, and the factors contributing to these conditions include [4, 5, 7, 9]:

- Demineralization of bone due to chronic metabolic acidosis.
- Metabolic acidosis impairs the renal activation of vitamin D, which is essential for proper bone mineralization.
- Hypomagnesemia in ureterosigmoidostomy, resulting from urinary loss of magnesium, can lead to hypoparathyroidism by reducing parathyroid hormone (PTH) release and causing PTH insensitivity, adversely affecting bone formation.
- Impaired intestinal absorption of both calcium and vitamin D occurs when

intestinal segments are used during diversion, leading to fat-soluble vitamin D deficiency, further aggravating bone demineralization.

- The associated presence of CKD contributes to metabolic bone diseases.

Metabolic bone diseases are late complications that can lead to osteomalacia and osteoporosis, increasing the risk of fractures [2].

D. Urolithiasis

The risk of renal stone formation is high (3% and 43%) in patients with ureterosigmoidostomy [10], and the mechanisms of its development are as follows [4, 5, 7, 11, 12]:

- Chronic metabolic acidosis leads to the release of bone calcium through the process of demineralization, resulting in hypercalciuria.
- Metabolic acidosis reduces urine citrate levels, and since citrate inhibits stone formation, a low concentration of citrate in the urine predisposes to the development of calcium-containing kidney stones.
- The increased intestinal absorption of bivalent anions, particularly sulfates, leads to elevated serum sulfate concentrations. The excretion of elevated urinary sulfate is coupled with the associated loss of urinary divalent cations, which can cause hypercalciuria and hypermagnesuria and raise the risk of stone formation.
- Dehydration due to diarrhea can lead to concentrated urine, which favors stone formation in the presence of the above predisposing factors.
- Anatomical changes that increase bacterial colonization, urinary stasis, mucus reflux into the upper tract, and exposure to surgical material such as sutures and staples may act as a nidus.

E. Hyperammonemia

In patients with ureterosigmoidostomy, urinary tract infections with urea-splitting organisms and urinary tract obstruction increase ammonia's production and subsequent colonic absorption significantly [13]. In patients with chronic liver disease or acute hepatic dysfunction, the liver cannot metabolize the increased ammonia load efficiently, leading to hyperammonemia and encephalopathy [4].

URINARY DIVERSION METHOD SELECTION

Selecting the appropriate urinary diversion method is crucial to prevent metabolic and electrolyte disturbances. In ureterosigmoidostomy, fluid and electrolyte disturbances are more common due to the larger capacity of the sigmoid colon and longer urinary contact time. To decrease these complications, the ileal conduit method for urinary diversion is effective because the ilial loop serves as a conduit and not as a reservoir. Urine is continuously drained into an external collecting bag, which reduces the contact time between urine and intestinal mucosa, thus minimizing the risk of metabolic and electrolyte disturbances. The incidence of stone formation is significantly lower with the ileal conduit diversion method, ranging from 9% to 11%, compared to 17% to 27% with the Kock pouch diversion [14].

The risk of metabolic and electrolyte disturbances is also reduced when methods like the ileal pouch urinary reservoir and orthotopic neobladder are used for urinary diversion, as they have smaller capacities, resulting in a smaller bowel surface area exposed to urine.

TREATMENT

Treating metabolic derangements and electrolyte imbalances in urinary diversion involves various approaches, depending on the specific imbalance present. Long-term planning with oral agents is desirable, as fluid and electrolyte abnormalities are usually chronic and persistent. In a few patients with severe dehydration, acidosis, and hypokalemia, vigorous hydration, alkalinization, and potassium repletion are needed [15].

A. Metabolic acidosis

Metabolic consequences and disturbances are frequently observed in patients following urinary diversion. Metabolic acidosis can lead to harmful effects in these patients, such as bone loss, increased protein catabolism, and decreased albumin synthesis, resulting in the loss of muscle mass and strength, making them frail and more susceptible to falls and fractures [2]. Initiating prophylactic treatment of metabolic acidosis early, at the stage of subclinical acidosis, and effectively restoring normal acid-base balance is crucial in preventing such metabolic complications [16].

The treatment for hyperchloremic metabolic acidosis requires administering alkalizing agents or blockers of chloride transport [8]. Alkalinizing therapy is commonly initiated with oral sodium bicarbonate (1–2 gm three times a day), but in some patients, it may cause significant gastrointestinal symptoms, such as excessive flatulence. Sodium citrate and citric acid (Shohl's solution) are used together as an effective alternative. Potassium citrate may also be used if excessive sodium administration is a problem (as in cardiac diseases) and potassium supplementation is desirable.

In patients with persistent hyperchloremic metabolic acidosis, where sodium loads are undesirable due to fluid retention/pulmonary edema and hypertension, chlorpromazine or nicotinic acid may be used to reduce the severity of acidosis. These agents impair chloride transport, limit the development of acidosis, and reduce the requirements for alkalizing agents rather than directly correcting the acidosis [8]. Chlorpromazine is usually given at a dose of 25 mg three times a day, whereas nicotinic acid is administered at a dose of 400 mg three to four times a day [17, 18].

Patients with ureterosigmoidostomy are advised to consume a low-sodium chloride diet to minimize their chloride intake and prevent acidosis [10].

B. Hypokalemia

Hypokalemia and total body potassium depletion are common in patients with ureterosigmoidostomies and are usually associated with hyperchloremic metabolic acidosis. Therefore, the treatment must involve simultaneous and cautious correction of both hypokalemia and metabolic acidosis.

In cases of severe potassium deficits, it is crucial to replace potassium before addressing the acidosis to avoid life-threatening hypokalemia. Conversely, correcting hypokalemia without treating metabolic acidosis can lead to hyperkalemia.

For prolonged oral correction, supplementing potassium citrate 15 mEq (approximately 1.6 gm b.i.d. to q.i.d.) helps in correcting both hypokalemia and acidosis in urinary diversion [19].

C. Metabolic bone diseases

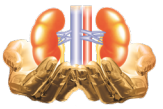
Early and adequate correction of metabolic acidosis is crucial for preventing metabolic bone diseases and improving bone health; if the improvement is not achieved, oral calcium and vitamin D3 supplementation may be considered [9].

D. Hyperammonemia

Hyperammonemia can be managed through a low-protein diet and the administration of medications like oral neomycin and/or lactulose while simultaneously addressing disorders that can increase ammonia production, such as acute or chronic liver disease, urinary tract obstruction, and urinary tract infection [19].

REFERENCE

1. Moon A, Vasdev N, Thorpe AC. Continent urinary diversion. Indian J Urol. 2013;29(4):303–9.
2. Gupta A, Atoria CL, Ehdaie B, et al. Risk of fracture after radical cystectomy and urinary diversion for bladder cancer. J Clin Oncol. 2014;32(29):3291–8.
3. Przydacz M, Corcos J. Revisiting Ureterosigmoidostomy, a Useful Technique of Urinary Diversion in Functional Urology. Urology. 2018;115:14–20.
4. Vasdev N, Moon A, Thorpe AC. Metabolic complications of urinary intestinal diversion. Indian J Urol. 2013;29(4):310–5.
5. Sperling CD, Lee DJ, Aggarwal S. Urinary Diversion: Core Curriculum 2021. Am J Kidney Dis 2021;78(2):293–304.
6. McDougal WS, Stampfer DS, Kirley S, et al. Intestinal ammonium transport by ammonium and hydrogen exchange. J Am Coll Surg 1995;181(3):241–8.
7. Van der Aa F, Joniau S, Van Den Branden M, et al. Metabolic changes after urinary diversion. Adv Urol. 2011;2011:764325.
8. Wintner A, Dahl DM. Use of Intestinal Segments in Urinary Diversion. In: Partin AW, Dmochowski RR, Kavoussi LR, Peters CA, editors. Campbell-Walsh Urology 12th Ed. Elsevier; 2021.p.3200–3201.
9. Cano Megías M, Muñoz Delgado EG. Bone and metabolic complications of urinary diversions. Endocrinol Nutr. 2015;62(2):100–5.
10. Pedrazaa IA, Pérez de la Cruza SM, López de Aldaa A. Percutaneous nephrolithotomy in a patient with a ureterosigmoidostomy diversion. Uro-Technology Journal 2022;6(4):17–20.
11. Terai A, Ueda T, Kakehi Y, et al. Urinary calculi as a late complication of the Indiana continent urinary diversion: Comparison with the kock pouch procedure. J Urol 1996;155(1):66–8.
12. Okhunov Z, Duty B, Smith AD, et al. Management of urolithiasis in patients after urinary diversions. BJU Int. 2011;108(3):330–336.
13. Albersen M, Joniau S, Van Poppel H, et al. Urea-splitting urinary tract infection contributing to hyperammonemic encephalopathy. Nat Clin Pract Urol 2007;4(8):455–58.
14. Zhong W, Yang B, He F, et al. Surgical management of urolithiasis in patients after urinary diversion. PLoS One. 2014;9(10):e111371.
15. Tollefson MK, Elliott DS, Zincke H, et al. Long-term outcome of ureterosigmoidostomy: an analysis of patients with >10 years of follow-up. BJU Int. 2010;105(6):860–3.
16. Stein R, Rubenwolf P. Metabolic consequences after urinary diversion. Front Pediatr. 2014;2:15.
17. Koch MO, McDougal SW. Chlorpromazine: Adjuvant therapy for the metabolic derangements created by urinary diversion through intestinal segments. J Urol 1985;134(1):165–9.
18. Koch MO, McDougal SW. Nicotinic acid: Treatment for the hyperchloremic acidosis following urinary diversion through intestinal segments. J Urol 1985;134(1):162–4.
19. Van der Aa F, De Ridder D, Van Poppel H. Nutrition issues in gastroenterology, SERIES #73. When the Bowel becomes the bladder: Changes in Metabolism After Urinary Diversion. Practical Gastroenterology. 2012;36(37):15–28.

KidneyEducation 

Join the Mission to Fight Kidney Diseases

Explore the world's largest multilingual website created by a global team of over 100 nephrologists.

www.KidneyEducation.com

» Read online or download the 200-page book "Save Your Kidneys" in 40 languages—completely free.

» This comprehensive resource offers valuable information on preventing and managing common kidney problems, tailored for kidney patients and their families.

» It's an authentic guide, prepared by nephrologists and free from any external funding.