



Metabolic and Intestinal Complications Following Urinary Intestinal Diversion: Mechanisms, Clinical Impact, and Management

Metabolni in intestinalni zapleti po derivaciji urina: mehanizmi, klinični pomen in zdravljenje

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Keywords: Hyperchloremic metabolic acidosis; small intestinal bacterial overgrowth; vitamin B12 deficiency; chronic kidney disease; osteoporosis.

ABSTRACT

Urinary intestinal diversion is an essential reconstructive procedure after radical cystectomy and in selected patients with complex lower urinary tract disorders. Incorporation of bowel segments into the urinary tract may cause metabolic, renal, nutritional, and gastrointestinal complications that require lifelong surveillance.

This narrative review summarises current evidence on the mechanisms, clinical impact, diagnosis, and long-term management of these complications. We performed structured PubMed/MEDLINE search. Publications focused primarily on toxicity attributable to neoadjuvant or adjuvant chemotherapy or pelvic

POVZETEK

Urinarna črevesna preusmeritev je pomemben rekonstruktivni poseg po radikalni cistektomiji in pri izbranih bolnikih s kompleksnimi boleznimi spodnjih sečil. Vključitev črevesnega segmenta v sečila lahko povzroči presnovne, ledvične, prehranske in gastrointestinalne zaplete, ki zahtevajo vseživljenjsko spremljanje.

Namen preglednega članka je povzeti sodobne dokaze o patofizioloških mehanizmih, kliničnem pomenu, diagnostiki in dolgoročni obravnavi teh zapletov. Izvedli smo strukturirano iskanje v zbirki PubMed/MEDLINE.

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radiotherapy were excluded when diversion-specific outcomes could not be separated.

Chronic hyperchloremic metabolic acidosis is a central mechanism linking electrolyte disturbances, renal dysfunction, and skeletal demineralisation. Chronic diarrhoea, malabsorption, vitamin B12 deficiency, and small intestinal bacterial overgrowth (SIBO) may further compromise nutritional status. In symptomatic patients, assess SIBO with standardised hydrogen-methane breath testing or, in selected cases, small-bowel aspirate culture; treat confirmed symptomatic disease while correcting underlying anatomical, motility, and nutritional factors. Chronic liver disease and sex-specific endocrine or functional consequences require individualised preoperative assessment and follow-up. Long-term outcomes depend on structured, risk-adapted, multidisciplinary surveillance of renal function, acid-base status, nutritional parameters, bone health, gastrointestinal symptoms, and selected endocrine outcomes.

INTRODUCTION

Urinary intestinal diversion (UD) is a fundamental component of reconstructive urologic surgery. It is most commonly performed after radical cystectomy (RC) for muscle-invasive bladder cancer, but it may also be required during anterior or total pelvic exenteration for selected locally advanced or recurrent pelvic malignancies and, less frequently, for complex benign lower urinary tract disorders. Therefore, consider the indication for surgery, prior pelvic treatment, comorbidity burden, and anticipated bowel and renal reserve when selecting the diversion technique (1–4).

Depending on patient characteristics and surgical indications, reconstruction may use incontinent techniques, such as an ileal conduit, or continent diversions, including orthotopic neobladders and continent cutaneous reservoirs. Although these procedures improve urinary function and quality of

Raziskave, osredotočene predvsem na toksičnost neoadjuvantne ali adjuvantne kemoterapije oziroma medeničnega obsevanja, so bile izključene, kadar njihovih učinkov ni bilo mogoče ločiti od posledic same urinske derivacije. Kronična hiperkloremična presnovna acidoza je osrednji mehanizem elektrolitskih motenj, napredovanja ledvične okvare in izgube kostne mase.

Kronična driska, malabsorpcija in prekomerna razrast bakterij v tankem črevesu (SIBO) lahko dodatno poslabšajo prehranski status. Pri simptomatskih bolnikih je priporočljivo objektivno testiranje za SIBO, potrjene primere pa je treba zdraviti ob sočasni odpravi osnovnega vzroka in prehranskih pomanjkljivosti.

Posebno oceno potrebujejo bolniki s kronično jetrno boleznijo ter bolniki s funkcionalnimi ali hormonskimi posledicami operacije. Dobri dolgoročni rezultati so odvisni od strukturiranega, tveganju prilagojenega in multidisciplinarnega spremljanja.

life, they expose patients to a distinct spectrum of long-term metabolic, renal, gastrointestinal, nutritional, and functional complications that require lifelong surveillance (1–3).

The pathophysiological basis of many complications is the continued absorptive and secretory activity of the intestinal mucosa after incorporation into the urinary tract. Exposure of ileal or colonic segments to urine promotes exchange of chloride, ammonium, bicarbonate, and other solutes across the bowel wall. The magnitude of these changes depends on the bowel segment and length used, urine-bowel contact time, baseline kidney function, hydration, and follow-up duration (1, 5).

Bowel resection and functional exclusion may also disrupt bile acid recycling, intestinal transit, nutrient absorption, and the gut microbial environment. Patients may consequently develop chronic diarrhoea, malabsorption, vitamin and micronu-

trient deficiencies, and SIBO. These manifestations can reduce quality of life even when the urinary functions satisfactorily (2, 3, 6, 7).

Long-term complications often develop gradually and may remain clinically silent until organ dysfunction is advanced. Symptom-driven assessment alone is therefore insufficient. Contemporary guidance supports structured functional follow-up that includes renal function, acid-base balance, vitamin B12 status, upper urinary tract assessment, and evaluation of patient-reported functional outcomes (4, 8).

Functional outcomes differ by sex and by the extent of organ and nerve preservation. Men may experience erectile, orgasmic, or ejaculatory dysfunction and daytime or nocturnal incontinence. Women may develop impaired neobladder emptying requiring clean intermittent catheterisation, incontinence, pelvic organ prolapse, dyspareunia, or vaginal stenosis. These outcomes justify sex-specific assessment of urinary, sexual, and quality-of-life domains during long-term follow-up (4).

Endocrine consequences require separate consideration when RC or pelvic exenteration includes bilateral salpingo-oophorectomy, causes premature loss of ovarian function, or is followed by persistent symptomatic hypogonadism in men. Menopausal hormone therapy and testosterone replacement are not routine treatments for every patient after UD; they should be considered only after confirmation of the relevant endocrine condition, assessment of oncologic and cardiovascular contraindications, and shared decision-making (9, 10).

The present review focuses on diversion-related metabolic and intestinal consequences. Oncologic outcomes and toxicities primarily attributable to systemic anticancer treatment or pelvic radiotherapy are outside its scope, although these exposures remain clinically relevant when interpreting an individual patient's postoperative risk.

This literature review summarises current evidence on metabolic, renal, intestinal, nutritional, skeletal, hepatic, and selected endocrine consequences associated with urinary intestinal diversion, with particular emphasis on pathophysiological mechanisms, clinical significance, diagnostic evaluation, and risk-adapted long-term management.

LITERATURE SEARCH AND NARRATIVE SYNTHESIS

We conducted narrative literature search was conducted in PubMed/MEDLINE to identify publications addressing metabolic and intestinal complications associated with urinary intestinal diversion. Medical Subject Headings (MeSH) and free-text terms included urinary diversion, ileal conduit, orthotopic neobladder, metabolic acidosis, electrolyte imbalance, renal function, chronic kidney disease, ureteroenteric stricture, small intestinal bacterial overgrowth, malabsorption, vitamin B12 deficiency, anaemia, osteoporosis, cirrhosis, chronic liver disease, surgical menopause, hormone therapy, hypogonadism, and testosterone replacement therapy. Boolean operators (AND and OR) were used to combine terms.

We prioritised adult studies, clinical guidelines, systematic and narrative reviews, and original research addressing metabolic, renal, gastrointestinal, nutritional, skeletal, hepatic, or functional outcomes after urinary intestinal diversion. We excluded paediatric studies, case reports, conference abstracts without full manuscripts, and publications unrelated to diversion-associated consequences. We excluded publications focused primarily on neoadjuvant or adjuvant chemotherapy or pelvic radiotherapy when diversion-specific outcomes could not be separated.

Because of substantial heterogeneity in study design, patient populations, diversion techniques, follow-up duration, and reported outcomes, we synthesised the evidence narratively and organised it into thematic sections. This article is a narrative review and does not constitute a systematic review or meta-analysis.

METABOLIC AND INTESTINAL COMPLICATIONS

Acid-Base and Electrolyte Disturbances

Metabolic acid–base abnormalities are among the best documented long-term complications of urinary intestinal diversion. Their development is primarily related to the persistent exposure of bowel mucosa to urine, which allows continuous exchange of electrolytes and solutes across the intestinal epithelium. Ileal and colonic segments retain their absorptive and secretory functions after reconstruction, promoting chloride and ammonium absorption together with bicarbonate secretion into the intestinal lumen. This process predisposes patients to hyperchloremic metabolic acidosis, particularly when large bowel segments are incorporated into the urinary tract or when renal function is impaired (1, 5).

The severity of metabolic acidosis varies according to the type of diversion, the length of bowel used, baseline kidney function, and duration of postoperative follow-up. In most patients, the disturbance remains mild, but persistent acid retention may contribute to fatigue, reduced exercise tolerance, muscle weakness, and progressive deterioration of mineral metabolism. Patients with chronic kidney disease have diminished capacity to compensate for the increased acid load and are therefore at substantially greater risk of clinically significant biochemical abnormalities (1).

Electrolyte disturbances frequently accompany acid–base imbalance. Hypokalemia may occur secondary to gastrointestinal and renal potassium losses, whereas alterations in sodium and chloride concentrations reflect ongoing ion exchange between urine and intestinal mucosa. Although severe electrolyte disorders are uncommon in patients with preserved renal function, regular laboratory surveillance is recommended because abnormalities may develop gradually and remain asymptomatic for prolonged periods (1, 2).

The choice of bowel segment also influences metabolic risk. Colonic urinary diversions generally pro-

duce greater chloride absorption and bicarbonate loss than ileal reconstructions, increasing the likelihood of clinically relevant acidosis. Conversely, shorter ileal conduits tend to produce fewer metabolic consequences than continent reservoirs or orthotopic neobladders constructed from longer bowel segments, although long-term monitoring remains necessary regardless of diversion type (3, 11).

Taken together, current evidence indicates that hyperchloremic metabolic acidosis is the central metabolic disturbance after urinary intestinal diversion and provides a pathophysiological basis for several renal, nutritional, muscular, and skeletal sequelae. Periodic assessment of serum bicarbonate, electrolytes, and renal function is therefore an essential for follow-up (1, 5). Table 1 summarises the principal complication categories.

Renal and Urinary Tract Complications

Renal impairment is one of the most important long-term concerns after urinary intestinal diversion. Deterioration is usually multifactorial and may reflect chronic acid load, recurrent infection, urinary obstruction, ureteroenteric stricture, nephrolithiasis, dehydration, and pre-existing chronic kidney disease. A longer bowel segment and prolonged urine–bowel contact may increase metabolic burden, whereas obstruction and infection can cause potentially preventable structural loss of renal function (1, 4, 8, 11).

Baseline renal function strongly modifies postoperative risk. Patients with reduced glomerular filtration rate have less capacity to excrete the additional acid load and are more susceptible to persistent metabolic acidosis and progressive kidney dysfunction. Even patients with preserved renal reserve require surveillance because clinically important abnormalities may emerge years after surgery (1, 4, 8).

The type of diversion also influences renal outcomes. Ureteroenteric obstruction, recurrent pyelonephritis, upper urinary tract dilatation, and morphological renal changes occur after both incontinent

Table 1. Comparative overview of metabolic and intestinal complications after urinary intestinal diversion

Complication category	Main mechanisms	Clinical manifestations	Key findings	Supporting references
Acid–base disturbances	Chloride and ammonium absorption, bicarbonate loss	Fatigue, weakness, asymptomatic acidosis	Hyperchloremic metabolic acidosis is the central metabolic disturbance after urinary intestinal diversion	Sperling et al. (1); Hautmann (5)
Electrolyte abnormalities	Intestinal ion exchange and renal compensation	Hypokalemia, altered chloride and bicarbonate levels	Electrolyte abnormalities often accompany chronic acid–base disturbance	Sperling et al. (1)
Renal complications	Chronic acid load, impaired renal reserve, urinary tract changes	eGFR decline, nephrolithiasis, renal deterioration	Baseline renal function strongly influences the severity of metabolic complications	Sperling et al. (1); Tinoco et al. (11)
Intestinal complications	Bowel resection, altered bile acid handling, changed transit	Diarrheal, bloating, malabsorption	Gastrointestinal symptoms are influenced by the bowel segment used and diversion type	Chesnut et al. (2); Barone et al. (3)
Nutritional deficiencies	Ileal resection, impaired absorption, chronic diarrhoea	Vitamin B12 deficiency, anaemia, malnutrition	Long-term follow-up should include nutritional assessment, especially after ileal diversion	Sperling et al. (1); Chesnut et al. (2)
Bone metabolism disorders	Chronic acidosis, calcium buffering, vitamin D disturbance	Osteopenia, osteoporosis, fractures	Chronic metabolic acidosis may contribute to skeletal demineralization over time	Sperling et al. (1); Hautmann (5)

Abbreviations: SIBO, small intestinal bacterial overgrowth.

and continent reconstruction. Imaging should be interpreted together with serum creatinine and estimated glomerular filtration rate, because serum creatinine alone may underestimate functional decline in patients with low muscle mass (4, 8, 11).

Stone formation is another clinically relevant complication. Altered urinary pH, chronic bacteriuria, urinary stasis, mucus, foreign material, and metabolic abnormalities may promote calculi. Adequate hydration, correction of acidosis, investigation of recurrent infection, and prompt treatment of obstruction are central preventive measures (1, 11).

Preservation of renal function therefore depends on both surgical and medical follow-up: early recognition of obstruction, infection, stones, dehydration,

and metabolic acidosis; serial biochemical assessment; and risk-adapted upper urinary tract imaging throughout long-term follow-up (1, 4, 8). Table 2 compares the metabolic and intestinal consequences of the principal diversion types.

Intestinal Complications and Small Intestinal Bacterial Overgrowth (SIBO)

Using intestinal segments for urinary reconstruction alters gastrointestinal anatomy and physiology. Most patients adapt, but some develop persistent diarrhoea, bloating, abdominal discomfort, altered bowel habits, or malabsorption. Severity depends on the segment and length of bowel resected or excluded, baseline gastrointestinal function, motility, medication exposure, and postoperative adaptation (1–3).

Table 2. Metabolic and intestinal consequences according to the type of urinary intestinal diversion

Type of urinary diversion	Intestinal segment used	Major metabolic considerations	Potential intestinal consequences	Long-term monitoring priorities
Ileal conduit	Short ileal segment	Hyperchloremic metabolic acidosis may occur but is generally less pronounced than in continent diversions	Mild bowel dysfunction; possible vitamin B12 deficiency after long-term follow-up	Renal function, electrolytes, acid–base status, vitamin B12
Orthotopic neobladder	Longer ileal segment	Increased risk of metabolic acidosis due to prolonged urine–bowel contact	Malabsorption, diarrhoea, vitamin B12 deficiency, possible SIBO	Renal function, serum bicarbonate, nutritional status, vitamin B12
Continent cutaneous reservoir	Ileum with or without colon	Electrolyte disturbances and metabolic acidosis may develop depending on bowel length and renal reserve	Altered bowel function and nutritional deficiencies in selected patients	Electrolytes, renal function, nutritional assessment
Colon-based diversion	Colonic segment	Greater chloride absorption and bicarbonate loss may increase the risk of hyperchloremic metabolic acidosis	Changes in bowel habit, diarrhoea, electrolyte imbalance	Acid–base status, electrolytes, renal function, gastrointestinal symptoms

Abbreviations: SIBO, small intestinal bacterial overgrowth.

Chronic diarrhoea is one of the most common gastrointestinal complaints. Ileal resection or exclusion may impair bile acid reabsorption and accelerate transit, increasing stool frequency and fluid loss. More extensive bowel utilisation may further reduce absorptive reserve and contribute to electrolyte depletion, dehydration, and nutritional imbalance (2, 6).

The preferred non-invasive diagnostic approach is standardised glucose or lactulose hydrogen-methane breath testing. Before testing, generally avoid antibiotics for four weeks, pro-motility agents and laxatives for one week for four weeks, promotility agents and laxatives for one week, fermentable foods for the preceding day, and food for 8–12 hours. Standard substrates are 75 g glucose or 10 g lactulose. A rise in hydrogen of at least 20 parts per million within 90 minutes supports SIBO, while methane of at least 10 parts per million at any time is more accurately termed intestinal methanogen overgrowth. In selected cases, quantitative duodenal aspirate culture of at least 10^3 colony-forming units/mL may sup-

port the diagnosis, but it is invasive and not a universally available gold standard (7).

Treat confirmed symptomatic SIBO rather than observe it without intervention. Management includes correcting the underlying anatomical or motility disorder where feasible, replacing nutritional deficiencies, providing hydration and dietary support, and an individualised course of antibiotics.

Rifaximin is the most studied minimally absorbed agent; systemic alternatives may be used according to clinical context, local antimicrobial policy, contraindications, and suspected microbial pattern. Avoid empirical repeated antibiotic courses without reassessment because recurrence is common and evidence for a single universal regimen is limited. Consider repeat breath testing when symptoms persist or recur (7, 12).

SIBO can contribute to malabsorption of vitamin B12 and fat-soluble vitamins, anaemia, weight loss, and chronic fatigue. Small observational studies

have also linked SIBO to reduced bone mineral density, suggesting that long-standing malabsorption may act as a cofactor in metabolic bone disease rather than an isolated direct cause. Recent evidence also shows a higher prevalence of SIBO in chronic liver disease, although causality and the benefit of routine screening in asymptomatic patients remain uncertain (13, 14).

Nutritional Deficiencies and Anaemia

Nutritional complications after urinary intestinal diversion primarily relate to ileal segment use altered in gastrointestinal physiology, and changes in nutrient absorption. Although many patients remain clinically stable for years after surgery, deficiencies may develop gradually and become apparent only during long-term follow-up. Their recognition is particularly important because several of these abnormalities are preventable or readily treatable when identified early (1, 8).

Vitamin B12 deficiency is the best recognised micronutrient disorder. Absorption occurs in the terminal ileum, and resection or functional exclusion of this segment may impair uptake. Because hepatic stores are substantial, deficiency may become clinically apparent only several years after surgery. Untreated deficiency can cause megaloblastic anaemia, cognitive impairment, peripheral neuropathy, and irreversible neurological injury, supporting regular laboratory assessment in patients with ileal diversion (4, 8, 15).

Iron deficiency and anaemia may also occur, although their ethology is usually multifactorial. Reduced intake, chronic inflammation, recurrent infection, occult blood loss, chronic kidney disease, and impaired intestinal absorption may all contribute. Persistent diarrhoea or SIBO may further compromise uptake of micronutrients required for erythropoiesis (1, 6, 7).

Patients with extensive ileal resection or exclusion may additionally develop impaired bile acid recyc-

ling and fat absorption. These alterations can cause chronic diarrhoea, weight loss, and deficiencies of vitamins A, D, E, and K. Risk increases with the amount of bowel affected and with coexisting motility disorders, SIBO, pancreatic disease, or chronic liver disease (6, 7, 14).

Long-term follow-up should include complete blood count and vitamin B12 measurement, with additional iron studies, folate, albumin, magnesium, and fat-soluble vitamin assessment guided by symptoms, diversion type, bowel length, liver disease, and nutritional risk. Correct deficiencies promptly, and evaluate unexplained abnormalities for malabsorption, SIBO, infection, bleeding, or renal disease (4,6–8,15).

Bone Metabolism, Osteoporosis, and Fracture Risk

Long-term alterations in bone metabolism have been increasingly recognised as clinically relevant complications of urinary intestinal diversion. Chronic metabolic acidosis, impaired renal function, and nutritional deficiencies interact to promote skeletal demineralisation and increase the risk of osteoporosis and fragility fractures. Although these changes often develop gradually, they may substantially affect morbidity in patients with prolonged postoperative survival (1, 11, 16).

A key mechanism underlying bone loss is persistent hyperchloremic metabolic acidosis. To buffer excess hydrogen ions, bone releases alkaline salts, mobilising calcium and phosphate from the skeleton. Sustained acid retention may therefore stimulate bone resorption, reduce bone mineral density, and impair normal mineralisation. In parallel, urinary calcium excretion may increase, further contributing to negative calcium balance and progressive skeletal weakening (1, 5).

Vitamin D deficiency and impaired intestinal absorption may amplify the skeletal effects of chronic acidosis. Patients with ileal resection, chronic diarrhoea, SIBO, or chronic liver disease may have

reduced absorption or altered vitamin D and calcium metabolism. Evidence linking SIBO directly to osteoporosis is limited to small observational studies; therefore, it should be regarded as a potential cofactor through malabsorption and poor nutritional status rather than as an established independent cause (6, 13, 14).

Population-based evidence supports the clinical relevance of skeletal complications. Patients with intestinal urinary diversion have a higher reported incidence of bone loss and fragility fractures than matched controls, consistent with the combined effects of chronic acidosis, renal impairment, age, hypogonadism, and nutritional deficiency (4, 16).

Bone-health surveillance should be risk-adapted. Assess serum calcium, phosphate, alkaline phosphatase, renal function, and 25-hydroxyvitamin D in patients with persistent acidosis, malabsorption, chronic liver or kidney disease, premature menopause, symptomatic hypogonadism, low-trauma fracture, or other osteoporosis risk factors. Dual-energy X-ray absorptiometry is appropriate when standard clinical criteria or these additional risks are present (1, 4, 9, 10, 16).

Chronic Liver Disease and Perioperative Risk

Chronic liver disease is not a consequence of urinary diversion, but it substantially modifies the safety of radical cystectomy and bowel-based reconstruction. Cirrhosis increases vulnerability to bleeding, infection, acute kidney injury, malnutrition, impaired wound healing, hepatic decompensation, and postoperative mortality. Contemporary radical cystectomy data demonstrate higher morbidity and mortality with increasing Model for End-Stage Liver Disease (MELD) score, and recent population-based evidence identifies chronic liver disease as an independent risk factor for adverse in-hospital outcomes (17, 18).

Preoperative assessment should establish whether liver disease is compensated or decompensated and

should include bilirubin, albumin, international normalised ratio, creatinine, sodium, platelet count, nutritional status, and evidence of portal hypertension, ascites, encephalopathy, or prior variceal bleeding. MELD and Child-Pugh classification provide useful baseline information, but current hepatology guidance recommends multidimensional surgery-specific risk assessment rather than reliance on a single score (19).

Elective surgery should be planned jointly by urology, anaesthesia, hepatology, nutrition, and, when needed, nephrology. Modifiable factors such as ascites, infection, alcohol use, malnutrition, electrolyte abnormalities, and renal dysfunction should be optimised. A MELD score of 10 or greater is a clinically relevant warning signal in radical-cystectomy cohorts, but it is not an absolute contraindication; the decision should consider cancer urgency, hepatic reserve, portal hypertension, frailty, and alternative oncologic strategies (17, 19).

Urinary diversion should be individualised. In patients with limited hepatic, renal, or nutritional reserve, a shorter and technically simpler bowel segment may reduce physiological burden, but direct comparative evidence in cirrhosis is insufficient to mandate one diversion for all patients. Postoperative surveillance should include liver function, coagulation, renal function, nutrition, infection, and signs of hepatic decompensation in addition to standard diversion follow-up (18, 19).

CLINICAL MONITORING AND MANAGEMENT STRATEGIES

Long-term success after urinary intestinal diversion depends on structured surveillance rather than surgical technique alone. Because many abnormalities are initially asymptomatic, follow-up should combine scheduled core assessments with more intensive, symptom- and risk-triggered testing. No single universally validated schedule exists; the protocol below synthesises available urological, nephrological, gastroenterological, hepatological, and endocrine guidance (1, 4, 7–10, 12, 19).

Metabolic and Renal Monitoring

Routine laboratory assessment should include serum electrolytes, bicarbonate, creatinine, estimated glomerular filtration rate, and, when bicarbonate is low or symptoms are disproportionate, venous blood gas analysis. Persistent hyperchloremic metabolic acidosis should prompt evaluation of hydration, renal function, infection, and obstruction. Oral alkali therapy may be indicated when acidosis is sustained, with dose and monitoring individualised to renal function and electrolyte status (1,8,11).

Upper urinary tract imaging should be integrated with oncologic surveillance and continued long term to identify hydronephrosis, ureteroenteric stricture, stones, or progressive structural deterioration. New renal dysfunction, recurrent infection, flank pain, or reduced stoma/neobladder drainage warrants expedited imaging and urological assessment (4,8,11).

Nutritional and Gastrointestinal Follow-up

Long-term nutritional surveillance is recommended, particularly after ileal diversion or extensive bowel surgery. Measure Vitamin B12 annually, as recommended by current European urological guidance. Add a complete blood count, iron studies, folate, albumin, magnesium, and fat-soluble vitamins based on symptoms and risk. Persistent diarrhoea, bloating, weight loss, or unexplained deficiency should trigger evaluation for bile acid malabsorption, SIBO, pancreatic disease, infection, and other causes (4, 6–8,1 5).

Dietary counselling and individualised nutritional support should address hydration, stool losses, energy and protein intake, and documented deficiencies. SIBO testing is symptom-triggered rather than routine. Treat confirmed symptomatic SIBO and address the underlying predisposing factor; recurrence should prompt reassessment rather than indefinite empirical antibiotic therapy (7, 12).

Bone Health Assessment

Patients with chronic acidosis or additional osteoporosis risk factors should undergo periodic assessment of calcium, phosphate, alkaline phosphatase, 25-hydroxyvitamin D, renal function, and relevant endocrine status. Dual-energy X-ray absorptiometry should follow standard osteoporosis criteria and be considered earlier in patients with fragility fracture, premature surgical menopause, confirmed hypogonadism, chronic liver or kidney disease, persistent malabsorption, or recurrent acidosis (4, 9, 10, 13, 16).

Chronic Liver Disease Monitoring

Patients with known or suspected chronic liver disease require preoperative and postoperative hepatology input. Monitoring should include bilirubin, albumin, international normalised ratio, aminotransferases, alkaline phosphatase, platelet count, renal function, sodium, nutrition, and clinical evidence of ascites, encephalopathy, infection, or portal-hypertensive complications. Frequency should be determined by disease severity and recent decompensation (17–19).

Sex-Specific Functional and Endocrine Follow-up

Sexual function, continence, voiding or emptying, pelvic-floor symptoms, and quality of life should be assessed in both women and men. Women who undergo bilateral oophorectomy before the expected age of natural menopause should be evaluated for vasomotor symptoms, genitourinary syndrome of menopause, cardiovascular and bone risk, and candidacy for menopausal hormone therapy. Systemic therapy may be appropriate after individualised oncologic and vascular risk assessment, particularly in non-hormone-sensitive malignancy, while low-dose vaginal estrogen is an effective option for isolated genitourinary symptoms. Testosterone therapy in women should not be used routinely for cardiovascular or muscle protection and is reserved for carefully assessed hypoactive sexual desire disorder (9).

In men, testosterone deficiency should be diagnosed only when compatible symptoms or signs coexist with consistently low morning total testosterone on two separate measurements. A value below 300 ng/dL is a reasonable diagnostic support threshold in the AUA guideline. Testosterone replacement is therefore not mandatory after RC or UD; it is an individualised treatment for confirmed symptomatic deficiency after assessment of fertility plans, haematocrit, cardiovascular status, prostate or breast cancer risk, sleep apnea, and other contraindications, with appropriate on-treatment monitoring (10).

Proposed Risk-Adapted Screening Protocol

The following schedule is a pragmatic synthesis rather than a validated universal standard. Modify it

according to diversion type, bowel length, baseline kidney and liver function, age, frailty, oncologic surveillance, and previous complications. Table 3 presents the proposed risk-adapted schedule.

Multidisciplinary Care

Optimal care may require urologists, nephrologists, gastroenterologists, hepatologists, endocrinologists or gynaecologists, dietitians, physiotherapists, and primary care clinicians. A coordinated plan is particularly important for patients with renal or liver disease, persistent gastrointestinal symptoms, malnutrition, recurrent acidosis, osteoporosis, surgical menopause, symptomatic hypogonadism, or complex functional problems.

Table 3. Proposed risk-adapted screening protocol after urinary intestinal diversion

Time point	Core assessment	Additional risk- or symptom-triggered assessment
Preoperative baseline	CBC; sodium, potassium, chloride, bicarbonate; creatinine/eGFR; calcium; albumin; urinalysis as indicated; upper-tract imaging; nutritional and functional history.	Liver panel, INR, platelets, MELD/Child-Pugh and hepatology review if liver disease; vitamin B12 and 25-OH vitamin D in high-risk patients; sex-specific endocrine history.
Approximately 3 months	Electrolytes, bicarbonate, creatinine/eGFR, hydration and stoma/neobladder function; review infection, obstruction, bowel symptoms, weight, and medications.	Venous blood gas if bicarbonate is low; imaging for renal deterioration, pain, infection, or poor drainage; nutritional tests if diarrhoea or weight loss.
6–12 months	Repeat renal and metabolic laboratory profile; upper-tract assessment coordinated with oncologic imaging; CBC and nutritional review.	SIBO breath test for persistent bloating/diarrhoea or unexplained deficiency; liver monitoring according to disease severity; DXA if clinically indicated.
Annually lifelong	Renal function, electrolytes and bicarbonate; annual vitamin B12; review infections, stones, bowel function, hydration, weight, continence/emptying, sexual function, and quality of life.	Iron/folate/fat-soluble vitamins, 25-OH vitamin D, DXA, hormone testing, or more frequent imaging according to risk and prior abnormalities.
At any new symptom or abnormal result	Prompt evaluation rather than waiting for the next scheduled visit.	Assess obstruction, infection, acidosis, dehydration, SIBO/malabsorption, hepatic decompensation, nutritional deficiency, or endocrine dysfunction as clinically appropriate.

Abbreviations: CBC, complete blood count; DXA, dual-energy X-ray absorptiometry; eGFR, estimated glomerular filtration rate; INR, international normalized ratio; MELD, Model for End-stage Liver Disease; 25-OH vitamin D, 25-hydroxyvitamin D; SIBO, small intestinal bacterial overgrowth.

DISCUSSION

This review confirms that metabolic and intestinal complications remain central to long-term care after urinary intestinal diversion. The physiological consequences of exposing bowel mucosa to urine interact with obstruction, infection, hydration, renal reserve, nutritional status, liver disease, age, and endocrine factors. Many complications are preventable or modifiable when surveillance begins before symptoms become advanced (1, 3, 4, 8).

Hyperchloremic metabolic acidosis is the most consistent unifying mechanism. It should not be regarded as an isolated laboratory finding, because persistent acid retention can aggravate muscle weakness, renal dysfunction, calcium loss, and skeletal demineralisation. The association supports routine measurement of bicarbonate and renal function, with investigation and treatment of sustained abnormalities (1, 5, 8).

Risk is highly individualised. Baseline kidney function, bowel segment and length, urine-bowel contact time, obstruction, infection, age, frailty, chronic liver disease, and nutritional reserve all modify outcomes. A uniform protocol is therefore less appropriate than a core lifelong schedule intensified according to risk and previous complications (1, 4, 8, 11, 17–19).

Gastrointestinal symptoms deserve systematic attention. SIBO is clinically plausible after anatomical and motility changes, but symptoms alone are not diagnostic. Standardised breath testing before treatment improves diagnostic discipline, while management should address the underlying cause and documented deficiencies in addition to antibiotic therapy. Evidence connecting SIBO with osteoporosis or liver disease supports clinical vigilance but does not establish a direct causal pathway in every patient (7, 12–14).

Nutritional deficiencies may remain silent for years. Vitamin B12 depletion is especially important because neurological injury may be irreversible. Annual measurement is a practical minimum after

bowel diversion, while broader testing should be guided by diarrhoea, weight loss, anaemia, bowel length, SIBO, chronic liver disease, and other indicators of malabsorption (4, 8, 15).

Bone health reflects the cumulative effects of acidosis, renal impairment, vitamin D and calcium deficiency, malabsorption, age, inactivity, surgical menopause, and confirmed hypogonadism. Hormone therapy may contribute to bone preservation in appropriately selected patients, but neither menopausal hormone therapy nor testosterone replacement should be presented as mandatory or as a universal preventive strategy after cystectomy (9, 10, 16).

This review has several limitations. The literature is heterogeneous in design, diversion type, baseline risk, and follow-up. Evidence specifically addressing SIBO, chronic liver disease, and endocrine therapy after urinary diversion is limited, and several recommendations are extrapolated from broader gastroenterology, hepatology, and endocrine guidelines. We excluded publications focused primarily on chemotherapy- or radiotherapy-related toxicity when diversion-specific effects could not be separated therefore, the review does not quantify the independent contribution of these oncologic treatments. The proposed screening protocol is a pragmatic synthesis and requires prospective validation.

Overall, the evidence supports a multidisciplinary model in which metabolic monitoring, renal function preservation, upper-tract surveillance, nutritional assessment, symptom-directed SIBO evaluation, bone health, hepatic risk, and sex-specific functional or endocrine care are integral components of long-term management.

CONCLUSION

Urinary intestinal diversion remains indispensable after radical cystectomy and in selected complex pelvic or lower urinary tract conditions. Diversion type, bowel length, renal and hepatic reserve, nutritional status, infection, obstruction, and individual

patient characteristics influence its long-term consequences.

Hyperchloremic metabolic acidosis is a key mechanism linking electrolyte abnormalities, renal impairment, muscle symptoms, and skeletal deterioration. Gastrointestinal disorders, including bile acid diarrhoea, malabsorption, and SIBO, may further compromise nutrition and bone health. Test SIBO objectively in symptomatic patients where feasible, and treat confirmed symptomatic disease alongside underlying causes and deficiencies.

Successful management requires lifelong, risk-adapted assessment of renal function, acid-base balance, upper urinary tract integrity, vitamin B12 and other nutritional markers, gastrointestinal symptoms, and bone health. Patients with chronic liver disease require multidisciplinary perioperative risk assessment, while hormone replacement therapy should be individualised for documented surgical menopause or symptomatic hypogonadism rather than used routinely in all patients.

Prospective studies are needed to validate surveillance intervals and clarify the incidence and clinical impact of SIBO, hepatic comorbidity, and endocrine dysfunction across diversion types. Until such evidence is available, structured multidisciplinary follow-up and early correction of modifiable abnormalities offer the best strategy to reduce morbidity and preserve long-term quality of life.

Use of Generative Artificial Intelligence

During manuscript preparation, OpenAI ChatGPT was used for language editing, translation support, and assistance in organising literature searches and manuscript structure. The human authors reviewed and approved all scientific statements, references, and final wording and take full responsibility for the manuscript's accuracy, integrity, and originality.

Conflict of Interest

Conflict of Interest: none declared.

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