

Prediction and Prevention of High-Output Stoma After Colorectal Surgery

Marcos Almanza Ramírez^{*1}, Mariana Damian Aldama¹, Daniela Gómez Gómez¹, Ana Isabel Cadena Carreón¹, Gustavo Quihuis García¹, Gabriel Hafner Vázquez Navarro¹

¹Hospital General Rubén Leñero.

ABSTRACT

High-output stoma (HOS) is the most frequent functional complication after the construction of a diverting or terminal ileostomy in colorectal surgery, affecting roughly 15-30% of patients and accounting for a substantial proportion of unplanned readmissions for dehydration, electrolyte derangement and acute kidney injury. Although the condition is largely preventable, its diagnosis is still delayed in routine practice because output measurement is inconsistent and diagnostic thresholds remain heterogeneous (1000-2000 mL/24 h). This narrative review summarizes current evidence on the definition, pathophysiology, risk factors, predictive models and preventive strategies for HOS after colorectal surgery. Consistently reported predictors include male sex, extremes of age, alcohol intake, perioperative use of angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, postoperative intestinal obstruction or ileus, intra-abdominal sepsis, hypoalbuminemia, leukocytosis and electrolyte abnormalities. Published prediction tools - from decision-tree models to multivariable nomograms - achieve areas under the curve of 0.79-0.91 but share important methodological limitations, including single-center retrospective design, inconsistent outcome definitions and scarce external validation. Preventive care is best delivered as a bundle: preoperative risk stratification and nutritional optimization, review of renin-angiotensin blockade and diuretics, systematic measurement of stoma output, early exclusion of sepsis and outlet obstruction, restriction of hypotonic fluid with glucose-saline rehydration solutions, stepwise antimotility and antisecretory therapy, structured education with output diaries, and protocol-driven follow-up after discharge. Adoption of a uniform definition and prospective multicenter validation of risk models are the priorities for future research.

KEYWORDS: High-output stoma; ileostomy; colorectal surgery; dehydration; risk prediction; enhanced recovery.

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I. INTRODUCTION

Colorectal cancer remains one of the most frequently diagnosed malignancies worldwide, and sphincter-preserving surgery has progressively replaced abdominoperineal resection for tumours of the middle and lower rectum [1]. Because anastomotic leakage after low or ultra-low anastomosis carries substantial morbidity and mortality, a diverting loop ileostomy is constructed in a large proportion of these patients; ileostomies now account for the majority of all stomas created in colorectal units [2,3]. Although a defunctioning ileostomy does not abolish leakage, it reliably reduces its clinical severity and the need

for urgent reoperation, and it is preferred to a colostomy because of the simpler reversal procedure [3,4].

The protective benefit of the ileostomy is not free of cost. Diverting the faecal stream above the colon removes the principal site of water and electrolyte absorption, and small-bowel effluent, with a sodium concentration of approximately 100 mmol/L, is delivered directly to the appliance [5,6]. When the volume of that effluent exceeds the compensatory capacity of the patient, the result is a high-output stoma (HOS): a state of progressive water, sodium and magnesium depletion that may culminate in dehydration, acute kidney injury, delayed adjuvant therapy and unplanned readmission [5,6,7].

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HOS is common, expensive and, to a large extent, preventable. Reported incidences vary between 8% and 31% depending on the definition used and on the phase of the postoperative course studied, and dehydration related to stoma output is the single most frequent reason for readmission after ileostomy creation [30,31,34,35]. Despite this, output measurement is often replaced in day-to-day practice by subjective reports from the patient or the family, and surveys of colorectal nurses show important gaps in knowledge and practice regarding the recognition and early management of HOS [49]. Many episodes therefore come to attention only when the patient is already thirsty, oliguric, hypotensive or azotaemic.

Over the last decade two complementary lines of work have matured. The first is predictive: several groups have derived statistical models - decision trees, logistic regression equations and nomograms - that identify, within the first postoperative days, the patients most likely to develop HOS [9,10,11]. The second is preventive: physiology-based fluid and drug regimens, structured nutritional intervention and standardized discharge pathways have all been shown to reduce output, dehydration and readmission [6,7,39,41,42]. The purpose of this narrative review is to bring these two lines together and translate them into practical recommendations for the general and colorectal surgeon, with particular attention to the early postoperative window in which prevention is still possible.

II. DEFINITION AND EPIDEMIOLOGY

There is still no universally accepted definition of HOS, and this single fact explains most of the heterogeneity in the literature. Thresholds used in published series include an output of at least 1000 mL/24 h for three consecutive days, at least 1500 mL/24 h, at least 2000 mL/24 h, and a peak output above 2000 mL/day combined with more than three days above 1000 mL/day [5,9,10,12]. The UK Association of Stoma Care Nurses and the most widely cited physiological reviews favor a threshold of 1500-2000 mL/24 h, on the grounds that water and sodium depletion becomes clinically relevant above this level in a patient with a normal oral intake [6,7].

Nightingale has argued convincingly that any fixed volume is an approximation, because the clinical consequence of a given output depends on what the patient drinks and absorbs: an output of 2 L is harmless in someone ingesting 4 L but produces dehydration in someone ingesting 0.5 L [6]. For clinical purposes it is therefore reasonable to define HOS as a stoma output that produces, or would predictably produce, water and sodium depletion; and to use 1500 mL/24 h as the operational alert threshold for the inpatient setting, since it identifies patients before biochemical decompensation.

TABLE I. DEFINITIONS AND REPORTED INCIDENCE OF HIGH-OUTPUT STOMA

Source / setting	Definition used	Reported incidence
ASCN UK guidelines; Baker et al. [5,7]	Output >1500-2000 mL/24 h causing water and sodium depletion	Up to 31% of small-bowel stomas
Nightingale [6]	>1500-2000 mL/24 h, interpreted against oral intake	16% early (<3 weeks); 4% persistent
Fujino et al. [10]	Peak output >2000 mL/day plus >1000 mL/day for >3 days	31% of the training cohort
Lee et al. [12]	Output ≥2000 mL/day during admission	15.9% (48/302)
Chen H et al. [9]	Maximum output ≥1500 mL/24 h during admission	27.7% (derivation), 28.9% (validation)
Zhang et al. [13]	>2000 mL/24 h, persistent, before stoma reversal	8.1% in the late/outpatient phase

The temporal distribution of HOS is bimodal. An early form appears within the first three postoperative weeks in approximately 16% of patients and is usually secondary to sepsis, ileus or medication; a late or chronic form persists beyond three weeks in about 4% and is more often related to a short residual bowel or to intermittent obstruction from strictures [6]. In colorectal cancer cohorts, in which the ileostomy is created in an otherwise intact small bowel, the early form predominates and typically peaks between the fourth and eighth postoperative day; in one recent cohort of 657 patients HOS occurred on average 5.8 days after surgery [9]. Table I summarizes the definitions and incidences reported in the studies analyzed for this review.

III. PATHOPHYSIOLOGY

A mature ileostomy produces 600-1200 mL of effluent per 24 h once adaptation has occurred [6]. Adaptation of the distal ileum, which allows progressive concentration of intraluminal contents, develops over 2 to 16 weeks after stoma formation; in the interval, and particularly in the first postoperative week, the patient is physiologically vulnerable [6]. Because jejunal and ileal mucosa are relatively leaky, ingestion of hypotonic fluid provokes a net efflux of sodium from plasma into the lumen until a luminal concentration of 90-100 mmol/L is reached; that sodium is then lost through the stoma. The classic vicious circle follows sodium depletion produces thirst, thirst produces further intake of

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hypotonic fluid, and stomal sodium losses increase further [6].

Superimposed on this baseline physiology, four mechanisms account for most cases of early HOS after colorectal surgery. First, sepsis and systemic inflammation. Intra-abdominal or organ/space infection produces hypoalbuminemia, reduced plasma colloid osmotic pressure and oedema of the bowel and stoma, leading to functional outlet obstruction with proximal accumulation and subsequent high-volume discharge; a raised white blood cell count on the first postoperative day is one of the earliest signals of this pathway [6,10,20]. Second, obstruction. Partial or intermittent obstruction, whether from adhesions, from kinking and rotation at the fascial aperture or from oedema of the stoma itself, generates bacterial overgrowth in the proximal segment and impaired absorption; the surge in output classically occurs during recovery from the obstructive episode, which is why HOS and postoperative ileus are so tightly correlated [12,20].

Third, medication. Prokinetic drugs, the abrupt withdrawal of opioids or corticosteroids, and blockade of the renin-angiotensin-aldosterone system all increase output. The intestinal renin-angiotensin system participates in fluid, electrolyte and nutrient absorption, and pharmacological inhibition with angiotensin-converting enzyme inhibitors (ACEI) or angiotensin II receptor blockers (ARB) impairs that absorptive capacity while simultaneously blunting the renal compensatory response to volume depletion [21,22,23,24]. Fourth, luminal and microbial factors. Analysis of ileostomy effluent shows dysbiosis in patients with HOS, and *Clostridioides difficile* enteritis of the small bowel is a recognized, if uncommon, cause of very high output [27,28]. In patients with extensive small-bowel resection, loss of the normal daily secretory load together with rapid gastric emptying, gastric acid hypersecretion and low circulating peptide YY and glucagon-like peptide 2 levels reproduce the physiology of short-bowel syndrome [6,29].

The clinical consequences follow directly from the mechanism. Sodium and water depletion cause thirst, cramps, postural hypotension and oliguria; magnesium depletion, reported in up to 78% of patients with a jejunostomy, produces fatigue, weakness and, when severe, tetany and arrhythmia; and unrecognized chronic depletion causes progressive renal impairment [6].

IV. RISK FACTORS

A. Demographic and lifestyle factors

Male sex is the most consistently reported non-modifiable risk factor. In a cohort of 470 patients undergoing ileostomy for colorectal cancer, male sex carried an odds ratio of 4.41 for early HOS, an association attributed to higher rates of smoking and alcohol consumption and to poorer bowel function after ileostomy in men [9,30]. A documented history of alcohol intake was independently predictive in the

same series (odds ratio 2.44) and in earlier work [9,11]; ethanol inhibits water and sodium absorption and is independently associated with postoperative diarrhea [52].

Age behaves inconsistently across studies and deserves particular caution. Two Asian series identified younger age as a risk factor, hypothesizing that younger patients resume oral intake and physical activity sooner, thereby stimulating intestinal motility [9,10]. In contrast, other authors report older age as predictive, on the basis that elderly patients tolerate rapid fluid shifts poorly and have less renal reserve [14,26]. Both mechanisms are plausible and probably operate in different populations; age should therefore be interpreted within the local context rather than treated as a universal predictor.

B. Comorbidity and medication

Perioperative use of ACEI or ARB has emerged as one of the strongest and, importantly, one of the few modifiable predictors. In a multivariable model it conferred an odds ratio of 6.68 for early HOS [9]; in an independent cohort it was the major risk factor for readmission with dehydration after ileostomy creation [21]; and population-based data link renin-angiotensin blockade to acute kidney injury after colorectal cancer surgery [22]. Diuretics carry a comparable logic and should generally be avoided in patients with an ileostomy who have had episodes of high output [6]. Diabetes mellitus, hypertension, chronic kidney disease and corticosteroid use appear in several models, although with weaker and less reproducible effects [9,15,18].

C. Disease and operative factors

Anatomical determinants matter. The risk of significant fluid loss increases when the stoma is fashioned from a proximal segment, when a substantial length of small bowel has been resected, when the whole colon has been removed, and in inflammatory bowel disease, in which chronic inflammation and immunosuppression add to the burden [5,6]. Conversely, problems are rare when more than half of the colon remains in continuity and more than 50 cm of functioning small bowel is present [6]. Series confined to rectal cancer have identified advanced T stage and a thick rectus abdominis muscle - a surrogate for a difficult, angulated stoma - as predictors of outlet obstruction and consequent high output [17]. Most conventional operative variables (operative time, blood loss, laparoscopic versus open approach) have not shown a consistent association with HOS [10].

D. Postoperative and laboratory variables

Postoperative intestinal obstruction and ileus are powerful predictors: obstruction carried an odds ratio of 5.90 for early HOS in one multivariable model [9], and 45.8% of patients with HOS developed postoperative ileus compared with 7.5% of those without [12]. Among laboratory variables, hypoalbuminemia, leukocytosis and hypocalcemia were independently associated with HOS in the largest recent nomogram study, with each 1 g/L increase in albumin

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reducing the odds by approximately 29% and each unit increase in white cell count increasing them by 47% [9]. Serum sodium, potassium and magnesium abnormalities are frequent in these patients and, although not always retained in multivariable models, remain clinically important monitoring targets [6,9]. Table III summarizes the risk factors by domain.

TABLE III. RISK FACTORS FOR HIGH-OUTPUT STOMA BY DOMAIN

Domain	Factors associated with high-output stoma
Demographic and lifestyle	Male sex; extremes of age (younger age in Asian cohorts, older age in others); regular alcohol intake; smoking [9,10,16,25]
Comorbidity and medication	Diabetes mellitus; hypertension; chronic kidney disease; perioperative ACEI/ARB; diuretics; corticosteroids; prokinetics; abrupt opioid withdrawal [6,9,15,21,22]
Disease and operative factors	Loop ileostomy sited close to the ileocaecal valve; extensive small-bowel resection; total colectomy; inflammatory bowel disease; advanced T stage; thick abdominal wall; open surgery [5,14,16,17]
Postoperative events	Intra-abdominal or organ/space sepsis; postoperative ileus; partial or intermittent obstruction and stoma outlet obstruction; Clostridioides difficile enteritis; dysbiosis [12,20,27,28]
Laboratory markers	Hypoalbuminemia; leukocytosis; raised C-reactive protein; raised creatinine; hyponatremia, hypokalemia, hypomagnesaemia and hypocalcemia [9,11,18,25]

V. PREDICTION MODELS

Three published models address the early prediction of HOS specifically after colorectal surgery, and a fourth addresses the related outcome of readmission for dehydration (Table II). Fujino and colleagues used recursive partitioning in 50 patients to build a decision tree based on sex, age and the white blood cell count on the first postoperative day, classifying patients into five strata with HOS rates ranging from 0% to 88%; the area under the curve was 0.838 in the derivation set and 0.792 in an independent validation set of 11 patients [10]. Chen and co-workers subsequently constructed a nomogram in 140 patients incorporating hypertension, alcohol consumption and leukocytosis, but the model showed only moderate discrimination and was never externally validated [11].

The most robust model published to date is a nomogram derived from 470 consecutive patients and temporally validated in a further 187, using a threshold of 1500 mL/24 h [9]. Eight predictors were retained: sex, age, drinking history, ACEI/ARB use, postoperative intestinal obstruction, albumin, white blood cell count and serum calcium. Discrimination was excellent (area under the curve 0.906 in derivation and 0.888 in validation), calibration was close to ideal, and decision-curve analysis demonstrated net clinical benefit across a wide range of thresholds. The events-per-variable ratio of 16 exceeds the conventional minimum of 10, which supports the stability of the coefficients.

TABLE II. PUBLISHED PREDICTION MODELS FOR HIGH-OUTPUT STOMA

Model	Predictors	Performance
Fujino et al. 2017, decision tree (n=50 derivation / 11 validation) [10]	Sex, age, white blood cell count on POD 1	AUC 0.838 derivation; 0.792 validation
Chen Y et al. 2023, nomogram (n=140) [11]	Hypertension, alcohol consumption, elevated white blood cell count	Moderate discrimination; no external validation
Chen H et al. 2026, nomogram (n=470 / 187) [9]	Male sex, younger age, alcohol intake, ACEI/ARB use, intestinal obstruction, albumin, white blood cell count, serum calcium	AUC 0.906 derivation; 0.888 temporal validation; good calibration
DRAI score 2018, readmission for dehydration (n>4000) [40]	Age, comorbidity, procedure and perioperative variables	Externally derived risk score for dehydration readmission

These models should nonetheless be interpreted critically. All are retrospective and single center; all were developed in East Asian populations whose body composition, dietary sodium intake and perioperative protocols may differ from those of other regions; and none has undergone geographical external validation or an impact study demonstrating that its use changes outcomes rather than merely predicting them. The temporal validation used in the largest study is a partial safeguard against overfitting but does not test transportability. Reporting also remains incomplete when judged against the TRIPOD statement, particularly regarding handling of missing data, treatment of continuous predictors and presentation of the full model equation.

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The most fundamental obstacle, however, is the outcome definition itself. A model developed with a 1500 mL threshold cannot be applied unchanged in a unit that defines HOS at 2000 mL, and comparisons of discrimination between models built on different outcomes are not meaningful. Until an international consensus definition is adopted, prediction models should be regarded as tools for internal risk stratification and for structuring surveillance, not as diagnostic instruments. In practical terms, their principal value is that they concentrate attention on a small number of variables that are available at the bedside within 72 hours of surgery - sex, age, alcohol history, renin-angiotensin blockade, albumin, white cell count, calcium and the presence of ileus - and that can be reviewed on a daily ward round without additional cost.

VI. CLINICAL CONSEQUENCES

The immediate consequence of uncontrolled output is water and sodium depletion, with thirst, weight loss, postural hypotension and oliguria. Dehydration is the leading indication for readmission after ileostomy creation, accounting for a substantial share of the 13-30% readmission rates reported in the literature, and meta-analyses confirm that dehydration-related readmission is both frequent and largely preventable [30,31,33,34,35]. Renal consequences are not trivial: temporary loop ileostomy measurably impairs renal function, and ileostomy creation is associated with an increased risk of both acute kidney injury and subsequent chronic kidney disease [36,37]. Acute kidney injury after ileostomy is particularly likely in patients with diabetes or a raised creatinine at discharge [12,15].

HOS also interacts with the surgical course itself. In a series of 302 patients undergoing rectal cancer surgery with diverting ileostomy, HOS was strongly associated with postoperative ileus (45.8% versus 7.5%) and with all-cause readmission (27.1% versus 15.0%); ileus in turn was an independent risk factor for readmission overall and for readmission with ileus [12]. Oncological care is affected as well: high-output states delay or interrupt adjuvant chemotherapy through dehydration, renal impairment and malnutrition [38]. Finally, evidence is emerging that the functional legacy of HOS extends beyond stoma closure. In 653 patients with a preventive ileostomy, those who experienced HOS before reversal had a threefold increased risk of major low anterior resection syndrome after closure (odds ratio 3.10, 95% confidence interval 1.56-6.14), an association attributed to electrolyte-induced sphincter dysfunction, autonomic disturbance and altered colonic motility [13]. Prevention of HOS is therefore not only a matter of fluid balance but potentially of long-term functional outcome.

VII. PREVENTION

A. Preoperative assessment and optimization

Prevention begins before the operation. Nutritional screening and correction of hypoalbuminemia, treatment of anemia, and control of diabetes address several of the variables that later appear in predictive models [9,18]. Medication review is the most concrete opportunity: ACEI, ARB and diuretics should be identified, and a plan agreed with the prescribing physician for withholding or reducing them in the perioperative period and for the interval during which the ileostomy is in place, resuming them only when output is stable and renal function normal [6,21,22]. Preoperative stoma siting by a specialist nurse, avoiding skin folds and the belt line, reduces leakage and appliance failure and facilitates accurate output measurement. Education delivered before surgery, when the patient is not fatigued or in pain, is retained far better than education delivered on the day of discharge and should explicitly cover the concepts of measuring output and of avoiding excessive plain water.

B. Operative considerations

Technical details influence subsequent output. The loop should be fashioned from terminal ileum, conventionally 15-25 cm from the ileocecal valve, preserving as much absorptive length as possible; a stoma constructed from a more proximal segment behaves physiologically as a jejunostomy [5,9]. The trephine should be adequate and the mesentery orientated to avoid rotation or kinking, both of which cause functional outlet obstruction and paradoxically high output during resolution [12,20]. Meticulous technique to minimize anastomotic leakage and organ/space infection is preventive, since sepsis is the commonest cause of early HOS [20].

C. Systematic measurement and early detection

No preventive strategy works without measurement. Stoma output should be charted from the first postoperative day, with every emptying recorded and a 24-hour total calculated, together with daily weight and fluid balance. An output of 1000 mL/24 h should trigger review; 1500-2000 mL/24 h defines HOS and mandates action. Serum creatinine, urea, sodium, potassium, magnesium and albumin should be checked every one to three days initially, and a random urinary sodium concentration below 10 mmol/L is a sensitive indicator of sodium depletion that precedes changes in serum values [6]. When output rises, a cause other than short bowel must be sought actively: examination for sepsis, cross-sectional imaging when a collection is suspected, digital examination of the stoma for stenosis, review of the drug chart, and stool testing for C. difficile [6,20,28].

D. Fluid and nutritional strategy

The counterintuitive core of treatment is fluid restriction. Patients must never be told to drink more to compensate for their output, because hypotonic fluid increases stomal

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sodium loss and worsens both dehydration and thirst [6]. Total hypotonic fluid should be limited to 500-1000 mL/24 h, and the remainder of the fluid requirement supplied as a glucose-saline oral rehydration solution with a sodium concentration of at least 90 mmol/L, sipped throughout the day. Commercial sports drinks and single-strength standard rehydration sachets contain too little sodium to be effective. Randomized and crossover data support both palatability-optimized rehydration solutions and oral rehydration salts as means of reducing ileostomy output [44,45]. Early involvement of a dietitian is associated with a significant reduction in HOS-related readmission and should be considered part of routine stoma care rather than a rescue measure [7,39].

E. Pharmacological measures

When fluid manipulation alone is insufficient, drug therapy is added in a stepwise fashion (Table IV). Loperamide, given 30 minutes before meals and at bedtime, is the first-line ant motility agent; because enterohepatic recirculation is disrupted and transit is rapid, doses considerably higher than those used for simple diarrhea are often required, although the total daily dose should generally remain below 80 mg and high-dose therapy warrants electrocardiographic surveillance because of the risk of QT prolongation [6,43]. Codeine phosphate may be added, accepting its sedative and addictive potential. In net secretors, in whom output exceeds oral intake, a proton pump inhibitor reduces the volume of gastric secretion delivered to the small bowel. Somatostatin analogues are effective but expensive and are reserved for refractory cases, and clonidine has been used successfully in small series [6,46,47]. Prophylactic probiotics have been proposed on the basis of their effect on postoperative inflammation, but the evidence in this specific setting remains preliminary [48].

TABLE IV. STEPWISE MEASURES FOR THE CONTROL OF STOMA OUTPUT

Measure	Practical prescription and caveats
Restriction of hypotonic fluid	Limit water, tea, coffee, juice and dilute drinks to 500-1000 mL/24 h; never advise the patient to "drink more" to match output [6]
Glucose-saline oral rehydration	Sip 1 L/day of a solution containing at least 90-120 mmol/L sodium (St Mark's or double-strength standard solution); chilling and flavouring improve compliance [6,44,45]
Salt supplementation	Added table salt or sodium chloride capsules when losses are 1200-2000 mL/day; nausea limits high doses [6]

Measure	Practical prescription and caveats
Loperamide	4-16 mg, 30 min before meals and at bedtime, titrated; total daily dose generally <80 mg; consider ECG before high-dose therapy [6,43]
Codeine phosphate	30-60 mg four times daily as an add-on; sedative and addictive, caution in the elderly [6]
Proton pump inhibitor	Omeprazole 40 mg daily in net secretors; discontinue if output does not fall [6]
Somatostatin analogues	Octreotide or long-acting lanreotide reserved for refractory output; cost, gallstones and injection-site effects [6,46]
Clonidine	Transdermal 0.3 mg in selected refractory cases; monitor blood pressure [47]
Nutrition consultation	Early dietitian review reduces HOS-related readmission; protein-energy support corrects hypoalbuminaemia [7,39]

F. Care pathways, discharge and follow-up

The most convincing evidence that HOS is preventable comes from structured pathways rather than from individual drugs. Standardized ileostomy pathways that combine preoperative education, in-hospital output monitoring, a written oral rehydration prescription and early post-discharge contact reduce readmission for dehydration, and their effect has been shown to persist over time [41]. Regimented follow-up with oral rehydration instructions likewise decreases readmissions after colorectal surgery that includes ileostomy [42], and multidisciplinary programmed combining nursing, dietetic and surgical input have reproduced these results [54]. Practical components include a patient-held intake-output diary, written thresholds at which the patient should contact the unit, a telephone review within the first week, and clinic assessment with electrolytes at two to four weeks [50]. Where HOS is persistent, earlier stoma closure after exclusion of anastomotic complications restores colonic absorption and resolves the problem definitively; when reversal is planned, stabilizing output, electrolytes and nutrition before closure is advisable, and may also mitigate subsequent bowel dysfunction [6,13].

VIII. A PRACTICAL BEDSIDE ALGORITHM

The evidence reviewed above can be condensed into a seven-step sequence applicable in any colorectal unit without additional technology (Figure 1). The sequence deliberately places measurement first, because the commonest failure in practice is not the absence of effective therapy but the absence of a reliable output chart; and it

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places the search for a treatable cause before symptomatic control, because treating sepsis or obstruction resolves the majority of early cases.

STEP 1 - Measure. Chart every stoma emptying from POD 1; record 24-hour volume, consistency and daily weight.

STEP 2 - Detect. Output ≥ 1000 mL/24 h = alert; ≥ 1500 -2000 mL/24 h = high-output stoma.

STEP 3 - Exclude a cause. Sepsis, intra-abdominal collection, ileus or outlet obstruction, *C. difficile* enteritis, prokinetics, opioid withdrawal, ACEI/ARB and diuretics.

STEP 4 - Correct. Restrict hypotonic fluid to <1000 mL/day, start glucose-saline solution, replace sodium, potassium and magnesium, review albumin and nutrition.

STEP 5 - Slow transit. Loperamide before meals, add codeine phosphate, then a proton pump inhibitor in net secretors; somatostatin analogues only if refractory.

STEP 6 - Protect the kidney. Daily creatinine and urine output; hold nephrotoxic and renin-angiotensin blocking drugs until output is controlled.

STEP 7 - Prepare discharge. Output diary, written thresholds for contacting the unit, oral rehydration prescription, early telephone review and clinic follow-up; consider earlier stoma closure.

Figure 1. Proposed stepwise algorithm for the detection, work-up and control of high-output stoma in the early postoperative period after colorectal surgery.

IX. LIMITATIONS AND FUTURE DIRECTIONS

This review is narrative rather than systematic, and the literature it summarizes has recognizable weaknesses. The absence of a consensus definition remains the dominant methodological problem, affecting incidence estimates, risk-factor analyses and model performance alike; an international Delphi process to agree a threshold, a time window and a requirement for persistence would do more to advance the field than any single new cohort. Most available series are retrospective, single-center and modest in size, with heterogeneous perioperative protocols, and the two largest prediction models originate from a small number of high-volume Asian centers. Follow-up is usually confined to the index admission, so the incidence of late and community-managed HOS is almost certainly underestimated.

Four priorities can be identified for future research. First, prospective multicenter cohorts using a standardized definition and standardized output measurement. Second, geographical external validation and prospective impact evaluation of existing models, ideally embedded in the electronic record so that risk is calculated automatically from routinely captured variables. Third, randomized evaluation of preventive bundles - perioperative interruption of renin-angiotensin blockade, protocolized glucose-saline

rehydration, prophylactic low-dose loperamide in high-risk patients - rather than of single components. Fourth, exploration of the longer-term consequences of HOS, including its relationship with renal function, completion of adjuvant chemotherapy and post-closure bowel function [13,36,38].

CONCLUSIONS

High-output stoma is a frequent, costly and largely preventable complication of colorectal surgery with ileostomy. Its pathophysiology is well understood loss of colonic absorption, delayed ileal adaptation, and a sodium-losing vicious circle aggravated by sepsis, obstruction and drugs that block the renin-angiotensin-aldosterone system. Risk factors identified consistently across cohorts - male sex, alcohol intake, ACEI or ARB use, postoperative obstruction or ileus, hypoalbuminemia, leukocytosis and electrolyte disturbance - are available at the bedside within the first postoperative days, and several prediction models, the best of which achieves an area under the curve close to 0.90, allow these variables to be combined into an individualized probability. Their promise remains conditional on external validation and on a shared definition of the outcome.

Prevention, in contrast, does not require further evidence to be implemented. Systematic measurement of stoma output, active exclusion of sepsis and outlet obstruction, restriction of hypotonic fluid with glucose-saline rehydration, stepwise antimotility and antisecretory therapy, early dietetic involvement, and a structured discharge and follow-up pathway together constitute a bundle that has repeatedly been shown to reduce dehydration and readmission. Incorporating these elements into routine postoperative care - and treating the stoma output chart with the same seriousness as the vital signs chart - is the most effective contribution the surgical team can make to the safety of a patient with a new ileostomy.

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