Seminars in Colon and Rectal Surgery 30 (2019) 45–49

Contents lists available at ScienceDirect

Seminars in Colon and Rectal Surgery

journal homepage: www.elsevier.com/locate/yscrs

Fecal incontinence, anal skin irritation, and metabolic concerns associated with pelvic pouches

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ARTICLE INFO

Keywords:
Fecal incontinence
Vitamin B12 deficiency
Osteopenia
Nephrolithiasis

ABSTRACT

Mechanical and inflammatory complications of pelvic pouch surgery have been well described but there has been limited recognition of problems such as fecal incontinence, anal skin irritation and metabolic changes that may develop after surgery. Reported rates of daytime and nighttime fecal incontinence rates are 29% and 47% respectively. We discuss strategies for managing fecal incontinence and associated anal skin irritation that can develop. Metabolic consequences of pelvic pouch surgery are also increasingly being recognized. Patients with IPAA who are doing well with no evidence of pouch inflammation have low rates of nutrient deficiencies but should be monitored for vitamin B12, vitamin D and zinc deficiencies. In contrast, patients with IPAA who develop pouchitis, diarrhea, and/or rectal bleeding are more susceptible to nutrient deficiencies and should have a broader assessment of nutrient status. In additions, there appears to be an increased risk of bone loss among patients with IPAA and thus these patients should undergo appropriate risk screening and testing. Finally, patients with IPAA have physiologic changes that predispose to formation of calcium oxalate and uric acid renal stones.

Restorative proctocolectomy has become the standard of care for the majority of patients with ulcerative colitis (UC) requiring surgery. Although complications such as mechanical issues, pouchitis, and a delayed diagnosis of Crohn's disease have been well described after surgery, there is increasing recognition of other problems such as fecal incontinence, anal skin irritation and metabolic changes that may develop after creation of a pelvic pouch.

Fecal incontinence

Fecal incontinence is a symptom that can be debilitating to a patient with a pelvic pouch. Reported incidences vary with daytime and nighttime incontinence rates of 29% and 47%, with a median follow-up of 11 years. This causality is because most often the stool consistency is soft to liquid and bowel movements can vary between 6 to more than 20 in a 24 h period.[1]

As part of preoperative patient counselling a face to face discussion of bowel habits after full functioning of the pouch is very important. Patients need to understand that they may never have “normal bowel movements” and that although the pouch now represents the rectal reservoir, it is quite different from the rectum in capacity, sensation and the evacuation mechanism. In a study from our institution we did not find incontinence to increase with the age of the pouch,[2] however aging anal sphincter musculature may contribute to leakage in a pouch with borderline incontinence.[3,4] Risk factors for incontinence include older age at surgery, low resting pressures prior to surgery and damage to the internal anal sphincter muscle. Other factors that may impact continence is the presence of pouch complications like abscesses, fistula or frequent pouchitis.[5]

The management of continence starts from the preoperative time point in assessment of the anal sphincter as part of an initial work up. This includes an anal manometry and a clinical evaluation of the anal sphincters.[6] Women who are multiparous may require additional evaluation of the sphincters with an endoanal ultrasound. Fecal incontinence may manifest as a mild skin irritation, seepage at night, urgency to frank leakage of soft to liquid stool.

Treatment of fecal incontinence in patients with a pelvic pouch

After a detailed history and examination the first line of therapy is medical management. This may include a session with a dietician and a physical therapist.

Skin care

Exposure of anal skin to excessive moisture and irritants such as feces, results in skin erosion, erythema, scaly damaged skin with

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https://doi.org/10.1053/j.scrs.2019.01.011
1043-1489/

poorly defined borders. This skin condition is referred to as irritant dermatitis from exposure to feces. Because of its known rapid onset the skin damage occurs within hours after exposure.[7,8]

Irritant dermatitis is confined to the contact area and leads to complains of burning and an itching sensation. Fungal infection, which can present as a diffuse red rash with defined margin and/or a yeast (Candida) infection which can presents as a rash with satellite papules and pustules are common presentations of irritant dermatitis caused by exposure of skin to feces.[7]

The normal skin flora stays in balance when the skin pH remains within the usual acid range of 4–6. Repeat exposure of skin to moisture and feces causes the skin pH to move into an alkaline range of more than 7. The pathologic bacteria multiply in the alkaline environment and the skin becomes vulnerable to a breakdown. Moist skin is more susceptible to mechanical trauma from aggressive cleansing and excessive friction. As the skin ages its function deteriorates and it has increased tendency toward alkalinity therefore, older adults are at higher risk to develop perianal skin irritation from moisture and passage of frequent loose stools. Soaps and cleansers with preservatives, fragrances and dyes increase the chance for skin irritation in a susceptible population. Identifying the causes of irritant dermatitis and differentiating from other skin conditions are imperative to guide prevention and treatment.[8]

The skin should be washed daily with a pH balanced cleanser to maintain its acidity and dried using a soft cloth to reduce friction and the risk for breakdown from repeated trauma. In addition to cleansing the skin and keeping it dry, the perianal skin should to be protected from exposure to feces and moisture. Skin barrier products containing zinc oxide, petrolatum, or dimethicone provide a physical barrier to irritants and moisture.[7] Before choosing the skin barrier product the clinician should review its ingredients, learn how easily it can be spread on skin application, find out the product availability in the region it is prescribed and the form in which it is supplied i.e., cream, lotion, ointment or liquid. It is of utmost importance that the skin barrier product is reapplied to the skin after each bowel movement. Excessive wiping with tissue paper should be avoided. A better choice to wipe with, is moistened tissue with plain water, to reduce friction.

Topical antifungals such as clotrimazole and miconazole are recommended if fungal and/or yeast infection is suspected. The medication should be applied first to the clean skin followed by the protective barrier.[7,8]

Dietary concerns

In addition to proper cleaning and protection of the perianal skin, other measures such as dietary modifications, use of bulking agents and bowel stoppers should be considered. Foods to bulk up stool include apple sauce, banana, pasta, smooth peanut butter and potatoes. Avoidance of greasy, spicy foods, and foods high in simple sugars which make the stools looser result in more irritation to the skin and are harder to control. Bulk-forming fiber supplements, such as psyllium products may improve stool consistency and reduce frequency and leakage.

Controlling bowel frequency and leakage

Changing bowel consistency is not easy in this patient category. Most patients with frequent bowel movements will need the aid of anti-diarrheals like loperamide or diphenoxylate and atropine. Other options are a small dose of a fiber supplement and dietary modifications which are individualized to the patient. Other treatment options include Kegel exercises if the anal pressures are low and electrical stimulation of the anal sphincters. Anal plugs (Renew insert™), though efficient, are not easy to use in this patient population with multiple bowel movements but can be used at night to prevent seepage.

Surgical options

Sacral neuromodulation has been used in patients with pelvic pouches very infrequently. A study found only 12 patients in 3 studies reporting on outcomes. All the studies reported good outcomes with improvement of continence. However no long term results or large cohort studies are available. The clinician should keep close contact with patient and adjust the plan of care as indicated.

Other possible therapies which are currently not available include the Fenix magnetic anal sphincter and the artificial bowel sphincter. Injection therapies and not usually considered. Likewise, the SECCA procedure due to the thin ileal wall that could possibly lead to complications is also not a consideration for treatment. Severe refractory incontinence in some patients may lead to a permanent ileostomy.

In summary, patients with an ileal pouch require a thoughtful customized approach if they experience postoperative skin and fecal incontinence difficulties. A multidisciplinary approach which involves the surgeon, a gastroenterologist, nursing, physical therapists, and dieticians may be necessary to address concerns. This is a lifelong commitment and as these patients age recommendations may need to be adjusted based on changing problems. Peer groups may also be beneficial to give support and guidance.

Metabolic concerns

Metabolic sequelae of nutrient deficiencies, bone loss and nephrolithiasis among patients with inflammatory bowel disease (IBD) who have undergone total proctocolectomy with ileal pouch-anal anastomosis (IPAA) are important and often forgotten.

Nutrient deficiencies

Although normal physiology is altered after IPAA surgery, one would still expect normal nutrient absorption as this occurs predominantly in the small intestine. In a study of 23 patients with UC and IPAA and 23 patients with UC and end ileostomy, there were no differences in overall low rates of deficiency of vitamin B12, folate, iron and trace elements between the two groups.[9]

Potential risk factors for development of nutrient deficiencies after IPAA include inflammation in the pouch or in the small bowel proximal to the pouch, rapid intestinal transit and development of small intestinal bacterial overgrowth. In a prospective study of 104 patients with UC who had undergone IPAA, 42% developed pouchitis and 33% had evidence of subtotal or total villous atrophy on biopsies of the pouch and proximal ileum.[10] Villous atrophy was associated with significantly lower levels of albumin, calcium, cholesterol, triglycerides, and vitamin E.[10] In another long-term follow up study of patients with IPAA, iron deficiency anemia was noted in 7% but was associated with bleeding and chronic pouchitis.[11] Similarly low magnesium levels were noted in 11% of patients but seemed related to pouchitis and associated diarrhea.

In addition to concerns about pouch inflammation, there may be independent increased risks for nutrient deficiencies in patients with IPAA. The best studied nutrients in patients with IPAA are vitamin B12, vitamin D, and trace elements.

Vitamin B12

The normal absorptive process for vitamin B12 is complex and requires several steps. After initial ingestion, vitamin B12 binds to haptocorrin in the stomach and then intrinsic factor in the duodenum to shield it from digestive enzymes. The intrinsic factor-B12 complex travels through the small intestine until it is taken up by a specific receptor in the terminal ileum.[12] Risk factors for vitamin B12

Table 1
Frequency of vitamin B12 and D deficiencies in patients with IPAA.

Study	N	Vitamin B12	Vitamin D deficiency (<20 ng/ml)
Coull et al.[15]	171	22%	–
M'Koma.[16]	83	5%	–
Kusima et al.[10]	104	5%	11%
Khanna et al.[20]	157	–	22%
Miller HL et al.[21]	74	–	22%

deficiency including resection of the terminal ileum and use of the ileum in reconstructive surgeries.[13] Vitamin B12 deficiency can lead to megaloblastic anemia, glossitis and demyelinating neurologic disease.[13]

Patients with IPAA may be at increased risk of vitamin B12 deficiency due to factors such as loss of ileal absorptive surface and development of small intestinal bacterial overgrowth.[14] Reported rates of vitamin B12 deficiency in patients with IPAA range from 5% to 22% (Table 1).[10,15,16] In a study of 171 patients with IPAA (150 of whom had UC as the underlying indication) who underwent sequential vitamin B12 measurements, 22% developed B12 deficiency after IPAA surgery.[15] The median time from surgery to first abnormal B12 measurement was 2.9 years.[15] Further testing in the B12 deficient patients revealed a normal Schilling test (indicating normal B12 absorption) in 94% and an abnormal hydrogen breath test (indicating small intestinal bacterial overgrowth) in only 1 patient.[15] Eight patients who had vitamin B12 deficiency and a normal Schilling test were treated with oral vitamin B12 replacement for 12 months and all 8 had normalization of their vitamin B12 levels. Other studies have demonstrated evidence of malabsorption of vitamin B12 in 20%–33% of patients although rates of B12 deficiency were only 5% in those studies.[10,16]

Vitamin D

Vitamin D plays an important role in several homeostatic mechanisms including immune function and bone metabolism.[17] Up to 60% of IBD patients have vitamin D deficiency, compared to 41% of the general population in the United States.[18,19] Replacement of vitamin D has been shown to be of benefit especially in patients with Crohn's disease.[17]

A study of 157 patients with UC and IPAA and 155 controls (UC patients without surgery) showed that 69% of IPAA patients had vitamin D insufficiency (level <31 ng/ml) compared to 42% of UC controls ($p < 0.001$) and 22% of IPAA patients had vitamin D deficiency (level <20 ng/ml) compared to 11% of UC controls ($p = 0.06$).[20] Inflammation of the pouch was not associated with low vitamin D levels. The only factor associated with low vitamin D levels on multivariable analysis was a low hemoglobin level (odds ratio [OR] = 3.37, 95% confidence interval [CI] - 1.41, 8.06). Another group found that 80% of patients with UC and IPAA had low vitamin D levels with 58% having vitamin D insufficiency and 22% having vitamin D deficiency.[21] Similar to the prior study, there was no correlation between low vitamin D levels and pouchitis.

Trace elements

Trace elements such as copper, manganese, zinc and selenium, are derived from the diet, required in small amounts, and are absorbed in the small bowel. Several studies have evaluated these trace elements in patients with IPAA. In a study of 23 patients with UC and IPAA and 23 patients with UC and end ileostomy, both groups had very low rates of trace elements deficiencies ranging from 0% to 9% for copper, manganese and selenium.[9] Zinc deficiency was a little more frequent occurring in 9% of IPAA patients and 13% of end ileostomy patients.[9] Another study by Pironi et al. compared 36 patients with IPAA who still had a diverting ileostomy, 18 patients with IPAA at least 3 months after closure of diverting ileostomy and 13 patients at least 3 months from proctocolectomy with end ileostomy.[22] The authors found significant differences in rates of zinc deficiency: 0% in the end ileostomy group; 25% in the diverted group and 61% in the IPAA group.[22] Ma et al. compared 40 hospitalized UC patients who were being prepared for surgery to 35 IPAA patients who were doing well after surgery and found that the IPAA patients had higher levels of zinc and manganese but lower levels of selenium compared to the pre-operative patients.[23]

El Muhtaseb et al. performed the only study comparing IPAA patients ($N = 55$) to healthy controls ($N = 46$) and found no significant differences in plasma copper, zinc, and selenium levels.[24] However, concentrations of blood manganese were significantly higher (median of 178.5 nmol/l versus 140 nmol/l; $p = 0.004$) in IPAA patients and 4 patients had manganese levels more than 3 standard deviations above the control group mean. Dietary assessment showed no differences between groups in trace element intake so the reason for the differences in manganese levels were not clear.

In summary, patients with IPAA who are doing well with no evidence of pouch inflammation have low rates of nutrient deficiencies but should be monitored for vitamin B12 deficiency (can develop in 5%–22%), vitamin D deficiency (can develop in 11%–22%), and zinc deficiency (can develop in 9%–60%). In contrast, patients with IPAA who develop pouchitis, diarrhea, and/or rectal bleeding are more susceptible to nutrient deficiencies and should have a broader assessment of nutrients including iron, calcium, magnesium, vitamin E and cholesterol levels.

Bone loss

It is well recognized that patients with IBD are at increased risk for low bone mineral density (BMD) with estimated rates of osteoporosis ranging from 14% to 42%.[25] The American College of Gastroenterology Preventive Care in IBD Guidelines recommend that patients with IBD who have conventional risk factors for bone loss should undergo BMD testing at the time of diagnosis and periodically afterwards.[25] In addition, the guideline recommends BMD testing for IBD patients who are initiated on corticosteroids and especially if patients have been treated with a prednisone equivalent dose of 7.5 mg or greater for longer than 3 consecutive months.[25]

Given that many patients with UC who undergo IPAA have likely been exposed to corticosteroids prior to surgery and may have additional risk factors for bone loss afterwards, it is important to assess their bone health. Among patients with UC and IPAA, rates of osteopenia range from 26% to 55%, rates of osteoporosis range from 13% to 32%, and the rates of fragility fractures range from 7% to 15%.[26] There has been some question in the literature as to whether restorative proctocolectomy in patients with UC should lead to increased or decreased risk of bone loss after surgery. In a large study of 267 patients with UC and IPAA compared to 119 patients with UC and no surgery, 31% of the IPAA patients had low BMD compared to 15% of the control group ($p = 0.001$).[27] Fragility fractures were noted in 8.1% of IPAA patients compared to 2.5% in the control group. On multivariable analysis combining both the study and control groups, IPAA was an independent risk factor for low BMD with an odds ratio of 6.02 (95% CI - 2.46, 14.70).[27] Other factors associated with low BMD were advanced age (OR = 1.51 per 5 years; 95% CI - 1.34–1.71) and low body mass index (OR = 2.37 per 5 kg/m^2 decrease; 95% CI - 1.68–3.36). In contrast, a study of a national cohort of Danish patients comparing 1757 patients with UC and IPAA to 8785 age and gender matched controls from the general population, found that fracture risk prior to IPAA was similar to the control group but decreased to half that of the general population (hazard ratio = 0.49; 95% CI - 0.43, 0.55) after IPAA.[28] The authors note that there was a short duration of disease prior to IPAA and low rates of systemic steroid use as likely explanations for the lack of increased fracture risk prior to IPAA.

In summary, patients with IPAA should be monitored for evidence of bone loss. Based on limited data, it appears that patients with IPAA may have a higher risk of bone loss compared to UC patients without surgery. However, when compared to non-IBD controls, fracture risk may be decreased after IPAA.

Nephrolithiasis

Patients with IBD are at increased risk of developing kidney stones with prevalence rates as high as 30% to 40% in some studies.[29–32] The risk is particularly increased in IBD patients who have undergone bowel resection due to factors such as dehydration with decreased urine output, malabsorption, and urinary composition changes including low pH and increased urinary uric acid and oxalate levels.[31,32] In particular among patients with colonic resection, lower urinary output, increased urinary uric acid levels and low urine pH have been described as leading to an increased risk of uric acid stones.[31,32] This increased stone risk has long been recognized in patients with ulcerative colitis who underwent proctocolectomy with end ileostomy.[33]

There are limited studies assessing nephrolithiasis risk among patients with IBD and IPAA, but IPAA patients seem to have similar physiologic changes to patients with proctocolectomy with end ileostomy. In study of urine composition among 13 UC patients with end ileostomy, 15 UC patients with IPAA, and 17 controls, the end ileostomy and IPAA patient groups had lower urinary volumes and pH and increased uric acid and calcium oxalate supersaturation compared to controls.[34] The only difference between the two patient groups was an increased predicted risk of calcium stones in the end ileostomy group but not in the IPAA group.[34]

The prevalence of nephrolithiasis after IPAA is not well described but in one study, 37% of patients with IPAA had renal stones after surgery.[30] However, it is important to note that it is not known whether some of these patients may have had stones prior to surgery that were not detected until after IPAA. These patients were compared to a control group of IBD/IPAA patients with no history of kidney stones and no stones on abdominal imaging following surgery. On multivariable analysis, factors associated with nephrolithiasis were presence of extra-intestinal manifestations consisting predominantly of arthralgia/arthropathy (OR = 2.9, 95% CI - 1.4, 5.8), no antibiotic use (OR = 3.2; 95% CI - 1.5, 6.5) and low serum bicarbonate levels (OR = 0.9; 95% CI - 0.8, 1.0).[30] No association was found between inflammation of the pouch and nephrolithiasis suggesting that factors other than mucosal inflammation predispose to kidney stone formation. Urine stone analysis data was available for eight patients: 2 had uric acid stones, 2 had calcium oxalate stones, 2 had mixed calcium oxalate and calcium phosphate stones, 1 had calcium oxalate crystals, and 1 had uric acid crystals.[30] In a follow up study from the same group, 40 patients with UC and IPAA with and without kidney stones underwent analysis of serum and urine metabolic factors.[35] IPAA patients with kidney stones had higher levels of 24-h urinary supersaturation of calcium oxalate and calcium phosphate.[35]

In summary, patients with IPAA have physiologic changes that predispose to formation of calcium oxalate and uric acid renal stones. Because of an increased predisposition to dehydration and decreased urinary volumes, all patients with IPAA should be counseled on the importance of maintaining adequate fluid intake. In addition, among patients with IPAA who develop renal stones, characterization of the stone type is important as dietary changes and use of supplements can be implemented.

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