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Pelvic pouch cancers associated with inflammatory bowel disease

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ABSTRACT

Patients with inflammatory bowel disease who undergo ileal pouch-anal anastomosis are at finite risk of developing neoplasia of various parts of the pouch. In this review article, we will describe different forms of pelvic pouch-related cancers, with a focus on diagnosis and management of small bowel adenocarcinomas of the pouch body, rectal neoplasia of the rectal cuff and anal transition zone, small intestinal lymphoma of the pouch, and anal dysplasia and squamous cell cancer of the anal canal.

Pouch-body cancer

Small bowel adenocarcinoma is a rare intestinal malignancy with an incidence in the United States of approximately 10,000 new cases per year, representing only 0.6% of cancers overall.[1] Small bowel adenocarcinoma has traditionally been associated with a poor survival, but improved survival have been observed, and recently published overall 5-year survival is now 67.6%.[1] Overall, this type of cancer is most commonly seen to arise from the duodenum as periampullary neoplasia, with only 17% being in the ileum.[2] Small bowel adenocarcinoma represents 28.6% of all patients with pouch-related adenocarcinoma while the majority are rectal cancer from the anal transition zone.[3]

In general, there is association of Crohn's disease and small bowel adenocarcinoma based on several population-based studies, likely secondary to mucosal proliferative pressure from chronic inflammation of small bowel and cellular injury-healing-injury cycle.[4–6] Adenocarcinomas (*i.e.* gland-forming) are tumors associated with mucosal injury, as opposed to small bowel sarcomas and other tumors discussed below which are not necessarily mucosal-derived gland forming, but rather stromal, tumors. Chronic histological changes in ileal pouch mucosa has been described in the literature and follows an initial phase of villous atrophy followed by normalization with "colonification" and blunting of the villi.[7] However, pathological changes of pouch mucosa can occur with chronic inflammation including severe villous atrophy and increased goblet cells.[8,9] Nonetheless, dysplastic changes may occur after ten years in patients with chronic pouchitis.[10]

As it relates to the pelvic pouch, small bowel adenocarcinoma may arise anywhere in the pouch including the afferent limb, efferent limb, and pouch body (Fig. 1). Pelvic pouch patients with pouch body cancers may be present early and be asymptomatic if on a surveillance pouchoscopy program, while symptoms may mimic pouchitis including new onset bloody stools, tenesmus or mass like sensation. This type of cancer may present late with small bowel obstructive symptoms and obstipation. Diagnosis is made by flexible pouchoscopy with biopsies. Staging includes computed tomographic (CT) scanning of the chest, abdomen, and pelvis looking for metastases, as well as contrast-enhanced 3-Tesla (3T) pelvic magnetic resonance imaging (MRI) for local staging (MR pouchogram), similar to as in rectal cancer.

Treatment is radical surgical extirpation with pouch excision and regional lymphadenectomy. Small bowel cancer staging follows the American Joint Commission on Cancer (AJCC) and Union for International Cancer Control (UICC) Tumor Nodes Metastasis (TNM) classification, and adjuvant chemotherapy is reserved for cases at high-risk of recurrence including those with nodal involvement or distant metastases. Adjuvant chemotherapy typically uses a combination approach including agents such as capecitabine and oxaliplatin (CAPOX), 5-flurouracil (5-FU), leucovorin with oxaliplatin (FOLFOX), and 5-FU, leucovorin with irinotecan (FOLFIRI). In the case of positive margins, adjuvant radiotherapy may have a role, while heated or pressurized intraperitoneal chemotherapy (HIPEC/PIPEC) may have a

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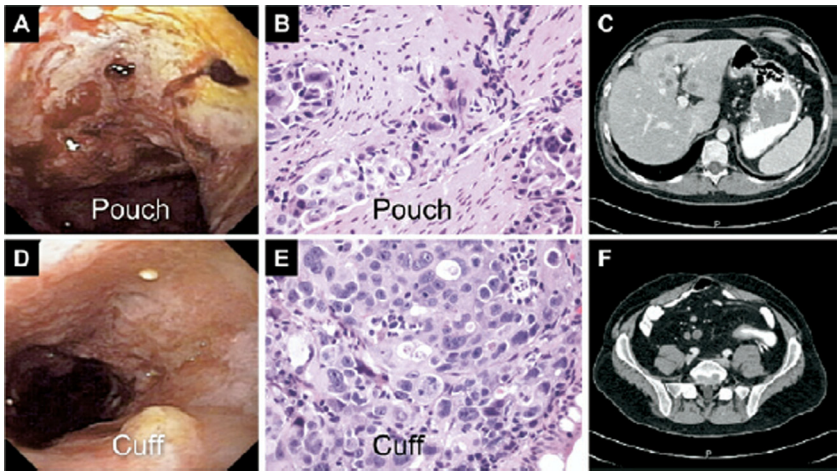

Fig. 1. Endoscopy, histopathology and CT findings in a patients who had both pouch (A & B) and anal transitional zone adenocarcinoma (D & E) with liver (C)and intra-abdominal lymph node involvement (F). Reprinted with permission Clinical gastroenterology and hepatology 2008; 6:145–158.

role in those with peritoneal involvement. Other novel treatments include checkpoint inhibitors (in the case of mismatch repair [MMR] mutations or microsatellite instability). Given the rarity of small bowel adenocarcinoma, clinical trials may be considered for this group of patients.

One difficultly in interpreting the data on pouch body adenocarcinomas is the need for differentiation between true pouch cancer versus cancer arising from ATZ. Additionally, after mucosectomy there may be "retained islands of mucosa" where microscopic areas of mucosal cells are inadvertently left behind during mucosal stripping. When the pouch is advanced down and a handsewn IPAA constructed, these islands of cells are covered with the pouch outlet and discovery of brewing cancer can be exceedingly difficult until a critical mass of cells erode into the pouch. Differentiation of cancer from the small bowel versus ATZ or mucosectomy area may also be difficult as histologic examination and staining may not necessarily differentiate small bowel and rectal adenocarcinomas. In this setting these cancers are often well-differentiated and thus the mucosa/glands of both small- and large-bowel adenocarcinomas will be positive for cytokeratin 20 (CK20) and mucin.[11] The key to differentiating the source includes location in the upper 2/3rd of the pouch (more likely small bowel) vs. lower 1/3rd (near pouch-anal anastomosis). Another clue is adjacent dysplasia, for example adjacent ileal dysplasia would suggest ileal source (Ilyssa Gordon MD, Cleveland Clinic Pathologist, personal communication).

Aside from small bowel adenocarcinoma, other cancers may arise in the pouch body including gastrointestinal stromal tumors (GIST, which arise from the interstitial cells of Cajal and to our knowledge have never been reported after IPAA),[12] neuroendocrine tumors (midgut NETs) – formerly called carcinoids, sarcomas (mesenchymal cancers), and lymphoma (discussed separately below) but the principles for work-up and treatment of these follow the above algorithmic oncologic approach with the following exceptions. For patients with GIST tumors, the c-*kit* inhibitor imatinib has a role in the neoadjuvant setting to down-stage large tumors and is indicated in those with a high-mitotic index, and also in the adjuvant setting in select cases at high-risk of recurrence due to tumor size, location, margin and nodal status.

Midgut NETs follow a different paradigm, and readers are strongly recommended to review the recently updated North American Neuro Endocrine Tumor Society (NANETS) guideline for mid- and hindgut NETs.[13,14] Briefly, small (<1 cm) pouch-body NETs may be managed endoscopically while larger tumors or those with poor outcome risk factors require radical surgery with lymphadenectomy. Staging and surveillance of midgut NETs utilizes specialized imaging with Octreoscans (which exploit the high degree of somatostatin receptors found in these tumors) and serial serum chromogranin A levels. Of note the recent 2017 revisions from NANETS includes updated diagnosis and treatment recommendations, based on 5 new positive randomized trials and treatment for midgut NETs. This field is currently undergoing a paradigm shift, with the recent FDA approval of ^{68}Ga-DOTATATE (Netspot©) nuclear medicine scan for identification of somatostatin receptor–positive tumors, ^{177} Lu-DOTATATE – a somatostatin receptor directed cellular-level radiotherapy, and everolimus in patients who progress radiographically, telotristat ethyl (a tryptophan dehydroxylate inhibitor) for refractory diarrhea from carcinoid syndrome.[15,16] The appropriate use of somatostatin receptor positron emission tomography for this type of tumor is discussed in an article from Hope, *et al.* and the reader is referred there for more in depth discussion of this topic.[17]

Rectal cuff/anal transition zone

Dysplasia and neoplasia of the anal transition zone (ATZ) represents rectal dysplasia and adenocarcinoma of the rectal cuff (Fig. 2). Cancer in this area, must be thought in the context that colorectal cancer is very common in the United States, with an incidence of >140,000 new cases per year, and is the 3rd most common malignancy in the USA, representing 8.1% of cancers overall.[1] Overall rectal cancer represents 1/3 of all colorectal cancers.[18] Secular trends, including the global focus on screening colonoscopy and high-quality total mesorectal excision, 3T pelvic MRI, and improvements in neoadjuvant and adjuvant therapies, the prognosis for patients with rectal cancer has improved and presently stage-for-stage is better than colon cancer.[18] The oncologic work-up and treatment follows the algorithmic approach listed above.

However there are two special considerations for ATZ and rectal cuff management/surveillance that differ from the general non-IPAA population. The first is that the indication for surgery in ~18% of patients who have a restorative proctocolectomy is high-grade dysplasia (HGD) or cancer, placing them in a high-risk subgroup. The other is that patients with primary sclerosing cholangitis (PSC) are also at increased risk of primary colorectal cancer (as well as pouchitis). The risk of progression from dysplasia to cancer is well-established.[19] Several studies have shed light on the risk of dysplasia after IPAA in PSC patients.[20] For these patients who are at increased risk of future rectal cuff neoplasia, there has long been controversy whether these patients should optimally be managed with an initial (prophylactic) mucosectomy and handsewn anastomosis versus double-stapled technique which is associated with improved long-term

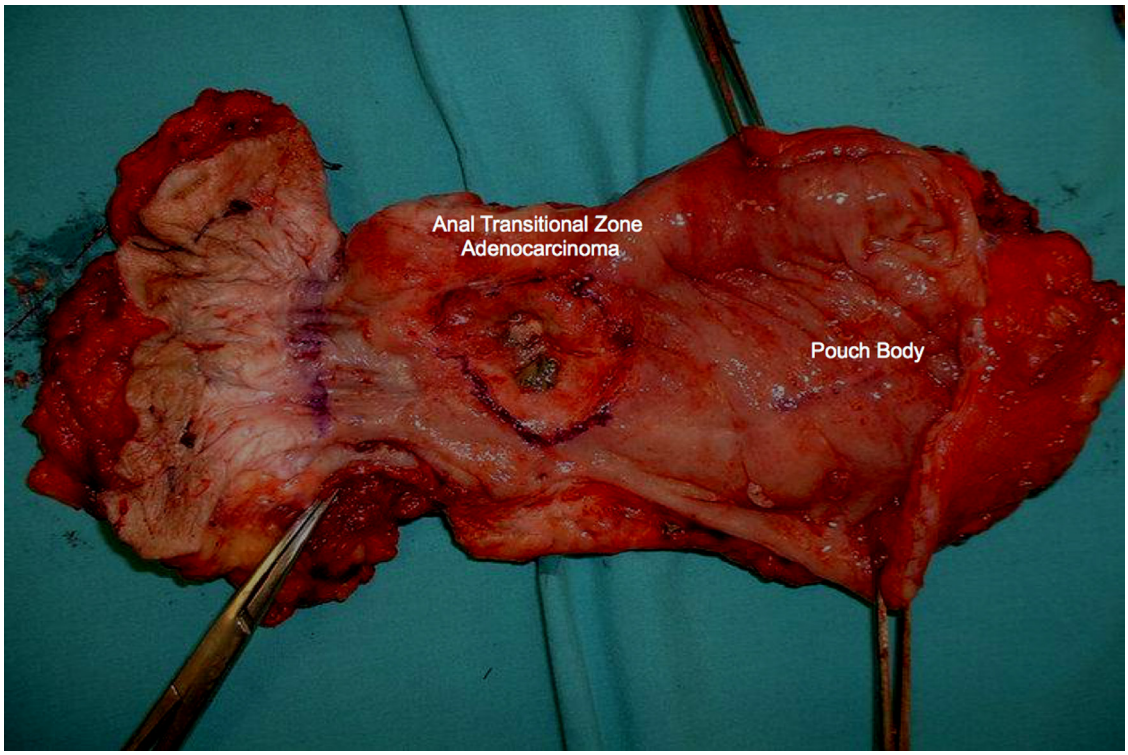

Fig. 2. Anal transitional zone adenocarcinoma outlined in pouch excision specimen. (Reprinted with permission from Dr. Tracy Hull).

functional outcomes.[21] Keeping in mind that a mucosectomy even when tediously performed may leave some rectal cells that are covered with the pelvic pouch as mentioned above, our preoperative approach to these patients is to preform extensive biopsies of the low rectum, in the setting of an examination under anesthesia if necessary. Lacking the presence of HGD or cancer in the mid to low rectum, the patient is offered a double-stapled anastomosis with at least yearly pouchoscopies with meticulous biopsy to the ATZ. When HGD or cancer is found on from preoperative distal rectal biopsies, the options discussed with the patient include a mucosectomy with handsewn IPAA; total proctocolectomy with end-ileostomy or Kock pouch. Finally, remembering that patients with PSC who initially undergo subtotal colectomy are at increased risk of rectal cancer of the retained rectal stump, they should be counseled to undergo the next step in surgery (completion proctectomy +/− reconstruction) or yearly biopsies of the retained rectum. Although rectal cancer overall is common, literature review demonstrates that rectal cancer of the ATZ pelvic pouch has rarely been reported.[22]

Pouch lymphoma

Overall, primary small intestinal lymphoma is also rare.[23] To date, there is no clear evidence that IBD by itself increases patient's risk of developing lymphoma.[24] The thiopurines, 6-mercatopurine (6-MP) and azathioprine (AZA), have been implicated in development of lymphoma in IBD patients as demonstrated in several large population studies.[25–27] Once the medication is discontinued, the age-adjusted risk of developing lymphoma reduces to that of general population.[26] Patient can still develop lymphoma similar to post-transplant lymphoproliferative disease (PTLD) with long-term usage of thiopurines, typically in individuals with previous Epstein-Barr Virus (EBV) infection.[28] Overall, treatment related lymphoma (AZA/6MP/anti-tumor necrosis factor [TNF]) is uncommon. (<0.1)[29] However, the addition of anti-TNFs to thiopurines seems to amplify the risk of developing lymphoma based on a meta-analysis of older studies,[24] although recently published data from post-marketing registry for adalimumab did not demonstrate increased risk of lymphoma during a mean of 3-years follow-up.[30]

In reviewing the literature, pouch lymphoma has rarely been reported. In a retrospective series spanning over 25 years from the Cleveland Clinic, a total of 3,203 IBD patients underwent restorative proctocolectomy with IPAA and only one case of pouch lymphoma was identified (0.03%). There are several case reports of ileal pouch lymphoma all in males with age range from 29–64.[31–35] Pouchitis (diarrhea, urgency and pain) and pouch dysfunction were the predominant symptoms felt to be from the lymphoma whereas some patients also had significant weight loss which is a classical symptom of lymphoma.[31,34] Diffuse large B-cell lymphoma (DLBCL) is the most common histological subtype amongst the case reports.

Diagnosis of pouch lymphoma can usually be made with endoscopic biopsy aided by imaging modalities such as endorectal ultrasound, CT and MRI. CT of chest, abdomen and pelvis can be utilized for staging, however, PET-CT may be more advantageous as it has a potential role in monitoring treatment response after chemotherapy and has been advocated in recent guideline.[36] Traditionally, the modified Ann Arbor Staging System is commonly used for staging due to its simplicity, however, other classification systems that are tailored to primary intestinal lymphoma have been reported in recent years. (i.e. Lugano and Paris classification)[37,38]

Although a multimodal approach is typically employed with surgery, radiation, and chemotherapy all playing a role,[23] chemotherapy remains the mainstay treatment for ileal pouch lymphoma similar to other primary intestinal lymphomas. Rituximab, an anti-CD20 antibody, can be used as first-line therapy to target B-cells specifically in combination with cyclophosphamide, doxorubicin vincristine, prednisolone (R-CHOP).[39] Some patients achieved good clinical response avoiding pouch excision[33–35] whereas patients who have minimal response require pouch excision.[32] There is no data in the literature in regards to long term outcome and recurrence rate after chemotherapy for pouch lymphoma.

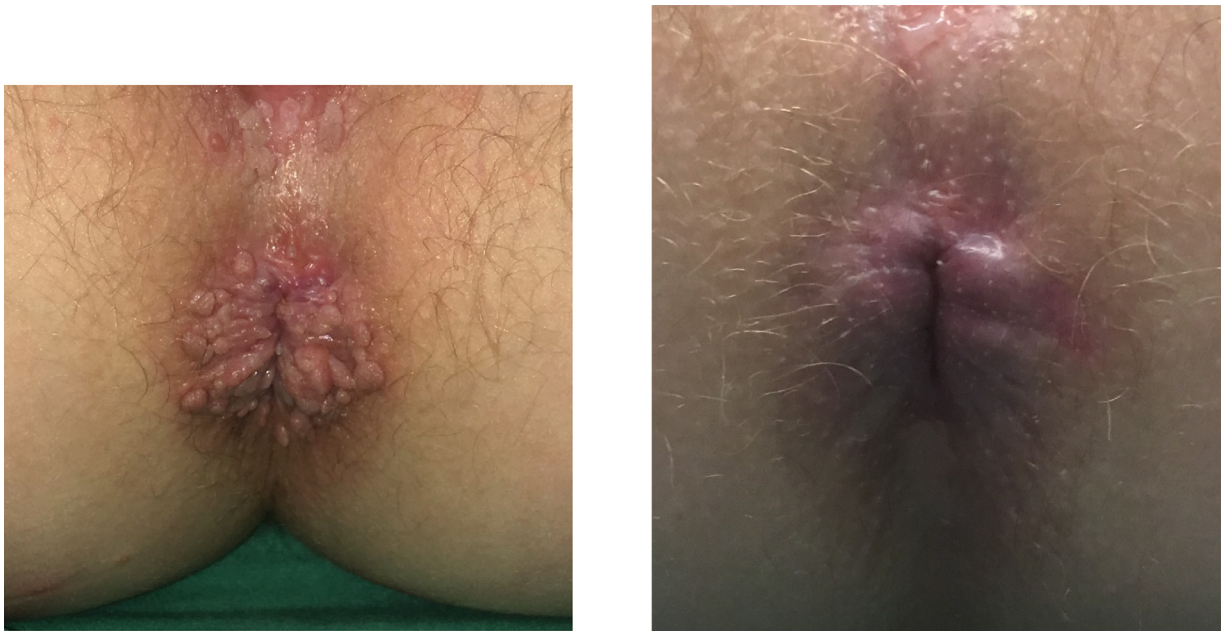

Fig. 3. Perianal condylomatous disease in an ulcerative colitis patient on high-dose immunosuppression, with spontaneous resolution after subtotal colectomy and withdrawal of immunosuppression (left panel perianal and vulvar condyloma in an acute colitis on steroids, right panel shows complete spontaneous regression). (Reprinted with permission from Dr. Stefan Holubar).

Anal dysplasia and squamous cell carcinoma of the anal cancer

Human papilloma virus (HPV) commonly results in perianal and anal canal condyloma, and rarely anal cancer. In the United States, the incidence of anal cancer is 16.3 per 1,000,000 person per year.[40] Globally, the estimated prevalence of HPV infection is 11.7% with the first peak at age 25 (or younger) and second peak after age 45.[41] Currently HPV is not routinely being screened for in IBD patients. Results from a retrospective study suggested a high prevalence of anal HPV in a small cohort of IBD patients (89.1%) with the majority diagnosed with dysplasia.[42] Patients who are receiving immunosuppressive therapy are at risk for new development or exacerbation of condylomatous disease (Fig. 3). Multiple risk factors for developing squamous cell carcinoma (SCC) have been studied including history of receptive anal intercourse, multiple sexual partners, men who have sex with men (MSM), history of anogenital cancer (including cervical, vulvar and vaginal), immunosuppression and the presence of oncogenic HPV type 16 and 18.

To date, only a handful cases of ileal pouch related SCC have been reported.[43–47] Most SCC occurred in the ATZ and rectal cuff. Only two cases of pouch body SCC have been reported.[44,45] Clinical presentation includes pouch obstruction, iron deficiency anemia and chronic pouchitis. Local and distant staging should be completed with CT chest, abdomen and pelvis and MRI pelvis. The management principles are similar to treatment of anal SCC. Radiation in additional to mitomycin and 5- Fluorouracil (FU) based chemotherapy should be offered to these patients. Pouch excision can be offered if patients have poor response to chemotherapy. Patients that require radiation therapy to their pelvis, typically have extremely poor pouch function afterwards and may require pouch excision or rediversion.

Summary

Pouch-associated cancers are thankfully rare. However, providers caring for IBD patients with pelvic pouches should be aware of the different forms of pouch-associated cancers that may develop. When conducting long-term surveillance with pouchoscopy, providers should always have a high-index of suspicion if any abnormality is found. Additionally in high-risk patients such as those with prior malignancy, PSC patients, patients who received long-term thiopurines, and patients who already had HPV-related perianal disease the index of suspicion should be much higher. Work-up includes the aforementioned pouchoscopy and otherwise follows standard algorithmic approaches. Treatment is multimodal but may require pouch excision with end-ileostomy, conversion to a Kock pouch, or neo-IPAA.

Conflicts of interest and source of funding

None.

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