Malignancy after gastrointestinal augmentation in childhood

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***Abstract*:**
Introduction: To review the incidence and risks of bladder cancer following gastrointestinal augmentations done for congenial anomalies in childhood.
Materials and Methods: A literature search using PubMed and Ovid Medline search engines was performed. MeSH terms evaluated were; bladder augmentations, enterocystoplasty, gastrocystoplasty, spina bifida, spinal dysraphism, myelodysplasia, neural tube defects, posterior urethral valves and bladder exstrophy were cross referenced with the terms, bladder cancer and urinary bladder neoplasm. All patients who developed a bladder cancer following a bladder augmentation for a congenital anomaly were reviewed
Results: A total of 20 cases of bladder cancer following augmentations for congential anomalies, were identified, 9 arose following ileal cystoplasty, 3 following colocystolasty and 8 following gastrocystoplasty. The incidence of cancer developing per decade following surgery was 1.5% for ileal/colonic and 2.8% for gastric bladder augmentations. The majority of cancers developing within the augmented bladder are at advanced stages at the time of diagnosis (60%; 12/20 cases were $\geq$T3 at diagnosis). Several of the cases that developed occurred in patients exposed to known carcinogenic stimuli and/or arose in bladders with a known predisposition to carcinoma.
Conclusion: Patients augmented with ileal or colonic segment for a congenital bladder anomaly have a 7–8 fold and gastric augments a 14–15 fold increased risk for the development of bladder cancer over standard norms. Published data is however unable to determine if gastrointestinal bladder augmentation is an independent risk factor for cancer over the inherent risk of cancer arising from a congenitally abnormal bladder.

***Keywords*:** bladder augmentation, urinary bladder neoplasia, exstrophy

Introduction

The association of bladder cancer and enteric bladder augmentation is well-documented, however, the vast majority of patients that developed cancer following augmentation had undergone a enterocystoplasty for tuberculosis or shistosomal cystitis, both of these latter entities are known to be associated with the development of bladder cancer.[Ali-El-Dein *et al*. 2002; Filmer and Spencer, 1990; Golomb *et al*. 1989] The incidence of malignancy following bladder augmentation performed for treatment of congenital etiologies is relatively unknown. The purpose of this paper is to delineate the risk of cancer development in a patient population who has undergone bladder reconstruction as a consequence of a congenital anomaly.

Materials and methods

A literature search using PubMed and Ovid Medline search engines was performed. Key terms evaluated were bladder augmentations, enterocystoplasty, gastrocystoplasty, spina bifida, spinal dysraphism, myelodysplasia, neural tube defects, posterior urethral valves and exstrophy were cross referenced with the terms, bladder cancer and urinary bladder neoplasm.

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Table 1. Bladder cancer following augmentation for congenital anomalies, review of published literature–enteric augments.

Etiology of bladder dysfunction	Augmenting segment	Length of time from augment to cancer/polyp development: years	Type and stage of cancer at diagnosis
Spina bifida [Husmann and Rathbun, 2008]	Ileum	52	Transitional cell carcinoma T2 N0 M0
Spina bifida [Husmann and Rathbun, 2008]	Ileum	53	Transitional cell carcinoma T2 N0 M0
Spina bifida [Soergel *et al.* 2004]	Ileum	21	Transitional cell carcinoma T2 N0 M1
Spina bifida [Soergel *et al.* 2004]	Ileum	13	Transitional cell carcinoma T3 N2 M1
Spina bifida [Soergel *et al.* 2004] AUS in place	Colon	21	Transitional cell carcinoma T3 N2 M0
Spina bifida [Austin *et al.* 2007]	Ileum	8	Carcinoma type not stated
Spina bifida [Barrington *et al.* 1997a]	Ileum	11	Adenocarcinoma T3 N0 M0
Spina bifida with renal Tx immunosupression [Husmann and Rathbun, 2008]	Colon	15	Adenomatous polyp
PUV with renal Tx immunosupression [Husmann and Rathbun, 2008]	Ileum	25	Adenocarcinoma T3 N2 M0
PUV with renal Tx immunosupression [Husmann and Rathbun, 2008]	Colon	22	Adenocarcinoma T3 N1 M0
Exstrophy[Husmann and Rathbun, 2008] AUS in place	Colon	32	Adenocarcinoma T3 N2 M0
Exstrophy [Husmann and Rathbun, 2008] AUS in place	Ileum	47	Adenocarcinoma T3 N2 M0
Exstrophy [Husmann and Rathbun, 2008]	Ileum	22	Adenocarcinoma T4, N2 M1

Only articles in the English language were reviewed. Two hundred and thirty one papers were read in abstract form, 68 papers were found to include and/or reference case reports. Only eight publications identified 21 patients that had bladder cancer or adenomatous polyps develop following augmentation for congenital anomalies. These articles were reviewed in-depth, patient information extracted and if patients were duplicated in various reports the duplicated patient reports were omitted.

Results

In our review of the literature we were able to abstract data on a total of 20 patients that had undergone a vesical augmentation for a congenital anomaly and subsequently developed a bladder neoplasm and 1 that had developed an adenomatous polyp in the augmenting segment, see Tables 1 and 2.

Enterocystoplasty was performed in 13 patients, the ileum was used for augmentation in 9 and colon in 4 (3 cancers and 1 polyp) [Husmann and Rathbun, 2008; Austin *et al.* 2007; Soergel *et al.* 2004; Barrington *et al.* 1997a]. The median time to tumor occurrence post enteric augmentation was 22 years with a range of 8–55 years. Cancer occurred in patients with ileal-colonic bladder augmentation at a rate of approximately 1.5% per decade post augment [Husmann and Rathbun, 2008]. The underlying congenital anomaly that resulted in the need for bladder augmentation, the type of cancer that arose, stage at diagnosis and latency from time of transplant to the development of the tumor is found in Table 1.

Gastrocystoplasty was performed in 8 patients [Vemulakonda *et al.* 2008; Castellan *et al.* 2007; Fernandez *et al.* 2005; Qui *et al.* 2003]. The median time to tumor occurrence post gastric augmentation was 14 years with a range of 11–15. Cancer occurred in gastric augments at a rate of approximately 2.8% per decade of follow-up [Vemulakonda *et al.* 2008]. The underlying congenital anomaly that resulted in the need for bladder augmentation, the type of cancer that arose, stage at diagnosis and latency from time of transplant to the development of the tumor is found in Table 2.

Discussion

General comments regarding the incidence of malignancy following gastrointestinal bladder augmentations

To determine the risk for the development of neoplasia in an augmented bladder three separate

Table 2. Bladder cancer following augmentation for congenital anomalies, review of published literature–gastric augments.

Etiology of bladder dysfunction	Augmenting segment	Length of time from augment to cancer/polyp development: years	Type and stage of cancer at diagnosis
Spina bifida [Castellan *et al.* 2007]	Gastric	12	Adenocarcinoma T3 N0 M0
Spina bifida [Castellan *et al.* 2007]	Gastric	14	Adenocarcinoma T1 N0 M0
Spina bifida [Castellan *et al.* 2007]	Gastric	11	Adenocarcinoma T2 N0 M0
Spina bifida [Fernandez *et al.* 2005]	Gastric	12	Adenocarcinoma T2 N0 M0
Spina bifida [Qui *et al.* 2003]	Gastric	14	Transitional cell carcinoma T2 N0 M0
Spina bifida [Vemulakonda *et al.* 2008] AUS in place	Gastric	13	Adenocarcinoma T3 N2M1
PUV with renal Tx immunosupression [Castellan *et al.* 2007]	Gastric	14	Transitional cell carcinoma T4 N2 M1 Pulmonary and peritoneal mets
PUV with renal Tx immunosupression [Vemulakonda *et al.* 2008]	Gastric	15	Adenocarcinoma T4 N2 M1 Pulmonary and peritoneal mets

groups of questions must be answered. (1) What is the incidence of bladder cancer in a normal population compared to individuals with an augmented bladder? (2) What is the normal incidence of cancer in the augmenting segment and is this risk altered by using this segment as an augment? (3) Does the bladder we are augmenting have an inherent increased risk for developing a malignancy?

What is the incidence of bladder cancer in a normal population compared to individuals with an augmented bladder?

The incidence for the development of a bladder neoplasm following ileocystoplasty or colocystoplasty is approximately 1.5% per decade post augmentation. [Husmann and Rathbun, 2008] The risk for cancer following a gastric cystoplasty appears to be elevated above this level at a risk of approximately 2.8% per decade post augmentation [Vemulakonda *et al.* 2008; Balachandra *et al.* 2007].

By extrapolation of the current data by the 6th decade of life (>50 yrs of age) the incidence of bladder cancer in patients with an enteric bladder augmention will be 5–6%, and in gastric augments will be 10–12%. Based on US statistics the incidence of bladder cancer in this age range would be 0.7%. These findings suggests patients will have a 7–8 fold increased risk of malignancy following an ileal or colonic augmentation and a 14–15 fold increased risk following gastric augmentation [Ries *et al.* 2008].

What is the normal incidence of cancer in the augmenting segment?

Patients in the 6th decade of life with colonic bladder augmentations will have a 5–6% risk of developing a cancer, this incidence is identical to the 5–6% risk of developing a colonic adenocarcinoma of the bowel by that age [Husmann and Rathbun, 2008; Ries *et al.* 2008; Barrington *et al.* 1997a]. Based on this information it appears that the colon, when used for bladder augmentation, will not be at an increased risk for neoplasia above that of the normal. In contrast, approximately 5–6% of patients with an ileal augmentation will develop a malignancy of the bladder, since the incidence of carcinoma developing in the small bowel is relatively low – approximately 0.1% – this represents a 40–50 fold increased risk [Husmann and Rathbun, 2008; Ries *et al.* 2008; Barrington *et al.* 1997a]. Long-term follow-up of gastric bladder augmentations suggests the incidence of malignancy in these augmentations should be 10–12% by 6th decade of life, the risk of gastric neoplasia in the US population in this age range is 0.3%, representing a 30–40 fold increased risk [Ries *et al.* 2008; Vemulakonda *et al.* 2008; Balachandra *et al.* 2007]. The current published data suggests that the segment of bowl used in augmenting the bladder may impact the incidence of subsequent bladder malignancy. This finding is not novel and has been documented for several decades. [Filmer and Spencer, 1990; Husmann and Spence, 1990] Indeed, the physician must be aware that the risk of malignancy in gastrointestinal bladder

augmentations will be a composite of all tissues involved.

In our review of the literature we also became concerned that gastrocystoplasty maybe an independent risk factor for the development of bladder cancer. This concern is based on the relatively high incidence of malignancy reported in this patient population during relatively short follow-up intervals [Vemulakonda *et al*. 2008; Castellan *et al*. 2007]. The concern is heightened by the frequent finding of gastric atrophy in surgical specimens and biopsies [Vemulakonda *et al*. 2008; Castellan *et al*. 2007; Take *et al*. 2007; Baydar *et al*. 2005; Ngan *et al*. 1993]. Gastric atrophy is a known premalignant condition that arises as a consequence of the Correa cascade. The latter phenomena is a multi-step process by which inflammation stimulates the host's immune response, the immunologic response can lead to gastric atrophy, intestinal metaplasia, epithelia dysplasia and eventually the development of invasive carcinoma [Take *et al*. 2007; Vajda *et al*. 2005; Qui *et al*. 2003; Vajda and Kaiser, 2002]. An increased risk of cancer is also of concern in composite gastic enteric bladder augments, this procedure mimics that of Bilroth II procedures where gastroenterotomies (gastric jejunal anastamosis) were performed for benign disease, these procedures were later found to be associated with frequent cancer development [Husmann and Rathbun, 2008; Baydar *et al*. 2005; Schafer *et al*. 1983].

Is the bladder we are augmenting at the normal risk for developing cancer or does it have an inherent increased risk for malignancy?

Our ability to assess if bladder augmentation is an independent risk factor for developing a malignancy is severely impact by inadequate information regarding the inherent risk of malignancy that may exist in the congenitally aberrant bladders [Woodhouse *et al*. 2006; Smeulders and Woodhouse, 2001; Shapiro *et al*. 1998]. Our assumptions are that the bladders we are augmenting are at normal risk for developing a malignancy; the increased frequency of malignancy following a bladder augment would then be attributed to the performance of the bladder augmentation. This assumption appears to be invalid. Indeed it is well-documented that the exstrophic bladder and, it is strongly suggested, the neurogenic bladder are at a significantly increased risk for developing bladder cancer [Austin *et al*. 2007; Michaud, 2007; Woodhouse *et al*. 2006; Smeulders and Woodhouse, 2001]. Additional problems exist in the calculating the risk for the development of cancer due to failure of reporting authors to report on multiple associated factors that may have played a role in cancer development. Specifically, genetic predisposition to the development of cancer, tobacco/immunosuppressive usage, exposure to of the patient to environmental toxins, the incidence of chronic bacteriuria, and recurrent bladder calculi may all impact the incidence of bladder cancer. Unfortunately the published studies rarely, if ever, report on the presence of these independent risk factors [Jankovic and Radosavljevic, 2007; Michaud, 2007; Pelucchi *et al*. 2006]. In essence, published data is unable to determine if gastrointestinal augmentation is an independent risk factor for cancer over the inherent risk of cancer arising from a congenitally abnormal bladder.

Etiology of cancer in bladder augmentations

It is believed that cancer arises within the augmented bladder due to either one or a combination of several of the following. (1) Chronic bacteriuria inducing nitrosamines and producing toxic oxygen radicals, both of which can induce DNA mutagenesis. (2) A direct toxic effect of the urine on the enteric epithelium. (3) The removal of the enteric epithelium from a intraluminal nutritional supplies (diversion enteritis or starvation enteritis). (4) Aberrant intercellular cell signaling mechanisms arising from mesenchymal-epithelial interactions of the disparate intestinal and urothelial tissues [Husmann and Rathbun, 2008; Dyer *et al*. 2005; Ali-El-Dein *et al*. 2002].

In general it is believed that a chronic infection is the primary stimulus for the development of bladder cancer. This hypothesis is based on the high incidence of chronic bacteriuria, found within this patient population [Husmann and Rathbun 2008]. In fact, it was originally hypothesized that gastrocystoplasties would be more resistant to malignant transformation than enteric cystoplasties due to the relative absence of chronic bacteriuria, 13% *versus* 55% respectively [Husmann and Rathbun, 2008; Baydar *et al*. 2005; Vajda *et al*. 2005]. Unfortunately as noted previously, this has not been found to be true. Also refuting the hypothesis that chronic bacteriuria is the etiology of cancer is the finding that there is no significant correlation between chronic bacteriuria and cancer development. Specifically, in patients undergoing a enteric

augmentation where no cancer developed 56% of all urine cultures were positive, in comparison in the patients where cancer developed bacteriuria was noted on 54% of the urine cultures which shows no significant difference between the two patient populations [Husmann and Rathbun, 2008]. Further supporting the fact that bacteriuria may not play a major role in cancer development and also aiding in the substantiation that an inherent bladder abnormality within dysfunctional bladders could possibly be leading to a malignancy is an excellent study by Barrington and associates [Barrington *et al.* 1997a]. This paper investigates the hypothesis that enterocystoplasty would be associated with cancer development due to the deficiency in intracellular antioxidant activity. This antioxidant deficiency would lead to increased toxic oxygen radical production and thereby increased DNA mutagenesis. These authors investigated 3 patient populations' neuropathic bladders with and without augmentation and normal controls. This study found significant antioxidant deficiencies of equal nature in patients following enterocystoplasty and in the non augmented neuropathic bladders compared to normal controls, ($p < 0.001$) [Barrington *et al.* 1997b]. This data certainly supports the concept that patients with congenital bladder dysfunction are at high risk for mutagensis whether or not an enterocystoplasty is present.

Immunosupression and the risk of bladder cancer

The association of immunosupression and the increased risk of bladder cancer does merit discussion. Four patients developed a malignancy and one an adenomatous polyp while on immunosuppressives, of note is that at least three of the five patients had a documented history of cytomegalo and/or Epstein-Barr viral infections post transplant. The development of urologic malignancies post transplant is known to be directly related to immunosupression, the presence of oncogenic viruses (Epstein-Barr, cytomegalic virus) and the loss of T suppressor function [Husmann and Rathbun, 2008; Besarani and Cranston, 2007]. It is worth mentioning that viral induced proteins mimic the actions of growth/transcription factors and can block apoptosis thereby altering cellular growth and aggravating any underlying cellular aberrancies. When bladder cancer does arise within patients on immunosuppressives it is usually rapidly progressive, poorly differentiated, multifocal and results in the patient's death [Husmann and Rathbun, 2008; Besarani and Cranston, 2007]. Current data suggest that when the physician finds a patient with the triad findings of: bladder augmentation, immunosupression and a history of cytomegalic or Epstein-Barr viral infections heightened concern for malignancy should occur. Whether or not routine surveillance of this select patient population should be performed will require additional clinical investigations.

Screening studies

Due to the risk of malignancy and the advanced stage of the malignancy at the time of diagnosis, (note 12 out of 20 cases of case were stage T3–4) numerous authors have suggested routine surveillance with urine cytology, cystoscopy and radiologic evaluations of this patient population beginning 5–10 years following bladder augmentation [Husmann and Rathbun, 2008; Castellan *et al.* 2007; Soergel *et al.* 2004; Lane and Shah, 2000; Filmer and Spencer, 1990]. The extreme cost of this endeavor must be carefully evaluated with regard to the frequency of cancer development. In enteric augmentations during a 10-year time span >990 cystoscopies would be performed to find one cancer. Even if a cancer is found it is unknown if annual cystoscopy would result in the diagnosis of a low-stage tumor. Indeed similar protocols have been abandoned in the spinal cord population due to cost and the inability to diagnose low-stage tumors despite aggressive surveillance [Austin, 2008]. We would suggest selective guidelines be developed for the evaluation of these patients, personally we see all individuals at yearly intervals with medical history, urinalysis, serum B12 and electrolyte assessment, and renal and bladder ultrasound. If medical history, laboratory evaluations or radiographic studies reveal; >4 symptomatic UTI per year, a history of gross hematuria, chronic perineal or bladder pain, urinalysis reveals >50 RBC/HPF or an abnormal US finding we proceed with additional radiographic, endoscopic and cytologic evaluations. We do, however, routinely cystoscope all patients with a colocystoplasty at 50 years of age as per suggested surveillance for bowel cancer, in addition we cystoscope annually all patients on immunosuppressives or with a history of a systemic viral infection.

Conclusion

Patients augmented with ileal or colonic segment for a congenital bladder anomaly have a 7–8 fold

and gastric augments a 14–15 fold increased risk for the development of bladder cancer over standard norms. Published data is, however, unable to determine if gastrointestinal bladder augmentation is an independent risk factor for cancer over the inherent risk of cancer arising from a congenitally abnormal bladder.

Conflict of interest statement

None declared.

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