Complications following augmentation cystoplasty; prevention and management

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ABSTRACT

The contemporary surgical management for both neurogenic and non-neurogenic urge incontinence is augmentation cystoplasty. Augmentation cystoplasty aims at increasing bladder capacity by incorporating either autologous (intestinal segments, ureter) or heterologous tissue (manufactured materials) as patches into the native bladder.

This review will address the issue of complications that are related to the use of different intestinal segments in bladder augmentation procedures. When possible, we will try to provide a time frame of the most important early and late complications as well as their prevention and managemen

Augmentation cystoplasty was fi rst described in 1888 when Tizzoni and Foggi augmented the bladder of a dog by connecting an ileal loop to the bladder neck [1]. Enterocystoplasty, fi rst performed in humans by von Mikulicz in 1899 was popularized by Couvelaire in the 50's originally for the treatment of small contracted tuberculous bladders [2, 3].

Bladder augmentation cystoplasty procedures were later introduced as surgical alternatives for cases of refractory detrusor overactivity and related urgency incontinence as well as for patients with neurogenic bladder dysfunction resulting from myelomeningocele, bladder exstrophy, spinal cord injury, multiple sclerosis, and myelodysplasia [4, 5]. The introduction of clean intermittent self catheterization was instrumental in broadening the applicability of bladder augmentation techniques for patients incapable of spontaneous voiding.

The rationale in augmentation cystoplasty is that by dividing a poorly compliant, small capacity bladder and interpositioning an intestinal segment it is possible to create a bladder with an increased functional capacity and a lower end fi lling pressure.

On the other hand there is no doubt that resolution to surgery that involves the transposition of intestinal segments into the urinary tract (eg. augmentation enterocystoplasty) is a procedure that is accompanied by signifi cant associated risks and complications.

Bladder augmentation is an invasive procedure that involves discontinuation of the intestinal tract and incorporation of a segment of bowel or ureter into the bladder. As a result complications inherent to this procedure are not limited to those that are common to any abdominal surgery but also those that result from both the disruption of the gastrointestinal tract, and the interposition of different types of bowel into the urinary tract.

This article aims to be a non-structured review on the complications following bladder augmentation procedures for the treatment of urgency urinary incontinence.

Towards that end we reviewed all available databases, including Pubmed and Medline, for publications in the English language. The search terms "bladder augmentation", "augmentation cystoplasty", "urgency incontinence", "enterocystoplasty", "complications", and "neurogenic bladder dysfunction" were tracked for in various combinations.

Complications that are related to the type of tissue used for bladder augmentation

Every portion of the gastrointestinal tract including the ureter has been used with varied success for bladder augmentation with the exception of the jejunum, which quickly fell into disrepute because of problems with water re-absorption [6].

This fact highly reflects the current absence of an ideal material for bladder augmentation as each intestinal segment is associated with unique properties and inherent risks for certain complications [7].

The Ileum has been the most widely used bowel segment for conduit urinary diversion and is considered the intestinal segment of choice for bladder augmentation [8]. A potential problem with the use of ileum is the persistence of peristaltic activity which cannot be completely abolished. In order to avoid this event the ileum segment is detubularized to interrupt the circular muscle fibers and prevent peristaltic contractions [9]. Moreover detubularized ileum is preferable to sigmoid colon because its tissue properties demonstrate better compliance and ensure the creation of a lower pressure reservoir [10].

A segment of ileum approximately 25-35 cm long, depending on the bladder defect, is isolated on its mesentery about 15 cm from the ileocecal valve, as this has been shown to result in the least metabolic disturbance.

The sigmoid colon is an equally efficient alternative to ileum in cases where ileum is not available due to prior small bowel surgery or adhesions. Sigmoid colon has the advantage of being able to create a large lumen and low pressure conduit with sigmoid cystoplasty resulting in significantly less postoperative bowel dysfunction compared to ileocystoplasty [11]. Disadvantages of sigmoid cystoplasty include excessive mucus production, a higher risk of UTIs (secondary to colonic bacteria), and a theoretically higher long-term risk of malignancy [4].

The cecum has also been used for augmentation cystoplasty usually in cases where subtrigonal cystectomy is considered. However and although cecum provides a larger reservoir, cecum cystoplasties have been related to higher incidences of diarrhea and

Table 1. Most common complications related to the GI segments used for bladder augmentation.

Intestinal segment	Risk of malignancy	Bowel dysfunction	Peristaltic activity/ urinary leakage	Metabolic disturbance	Mucus production	Calculi formation	Urinary tract infections	Other
Ileum	+	++	+	Metabolic acidosis +	++	++	+	Perforation ++
Sigmoid colon	++	+	++	Metabolic acidosis ++	+++	++	++	Perforation ++
Stomach	+		+	Metabolic alkalosis ++	+	+	+	Hematuria/ dysuria syndrome, peptic ulceration, perforation +

malabsorption due to the resection of the ileocecal valve while also producing more mucus than ileocystoplasties [4, 12].

The stomach has also been used as an alternative to enterocystoplasty with increasing popularity during the past decades. Stomach is theoretically ideal for bladder augmentation because it is easy to mobilize, well vascularized, produces less mucus, causes less postoperative bowel dysfunction and carries a lower risk for stone formation compared to ileum or colon [13, 14]. Furthermore, stomach secretes hydrochloric acid that has a bactericidal action (asymptomatic bacteriuria is seen in only 20-30% of patients after gastrocystoplasty) [15, 16].

Despite the impressive results regarding continence and bladder capacity reported in initial pediatric series , it was the increasing rate and the severity of complications encountered that led to a decline in the use of stomach for bladder augmentation. Stomach is now only used in situations where bowel is unavailable or unsuitable[17, 18, 19].

However, since most series of gastrocystoplasties are relatively small, the true rate of complications, including reoperation rates and metabolic derangements, is unknown.

Complications specific to gastrocystoplasty mainly include the "hematuria-dysuria" syndrome caused by gastric acid secretions. This syndrome consists of bladder and/or urethral pain, hematuria, and skin excoriation in the absence of infection. It is a serious complication, seen in up to 36% of patients after gastric augmentation [20].

Other reported complications following gastrocystoplasty include peptic ulceration of the bladder, stone formation, perforation of the gastric segment due to peptic ulcer, hypergastrinemia and hypochloremic metabolic alkalosis responsive only to omeprazole [21, 22, 23, 24, 25] .

Complications associated with partial gastrectomy, include the "dumping" syndrome and exacerbation of a pre-existing peptic ulcer or gastro-esophageal reflux, while there is also the risk of malignancy in the gastric patch [26]. The significant incidence of these complications has reduced the use of stomach alone for augmentation, except in cases of insensate bladders and in combination with other intestinal segments [4, 7]. Contemporary indications for gastrocystoplasty include children with renal insufficiency where the use of a bowel segment is contraindicated, and adults where bowel is unavailable or has been irradiated.

The realization that intestinal segments when used as bladder patches are not actually free of complications led the investigators to explore the possibility of using dilated ureter to enlarge the bladders capacity. The idea was initiated from the observation that ureteral dilatation in patients with massive reflux acts by both decompressing the bladder and preserving the upper urinary tract.

The use of ureter for bladder augmentation permits the creation of a low-pressure reservoir without interruption of the urothe-

lial lining; thus avoiding the side effects and complications encountered with the use of bowel like mucus production, malignant transformation, and metabolic disturbance. Studies have found ureterocystoplasty to be equally effective to enterocystoplasty in the pediatric population [27-29].

The theoretical advantages of ureterocystoplasty over the use of intestine for bladder augmentation are however counterbalanced by the currently limited applicability of the technique. It is the rare male child with unilateral ureteral dilation, hydronephrotic kidney with diminished renal function, and low bladder compliance resulting from posterior urethral obstruction that will make a suitable candidate for this technique [30]. Table 1 summarizes the specific complications related to the intestinal segment of choice for augmentation cystoplasty.

Early complications following enterocystoplasty

Augmentation cystoplasty is reported to have a 0-2.7% mortality rate, with higher mortality and complication rates reported in the earlier series where augmentation was part of urinary undiversion [31, 32].

Complications after augmentation enterocystoplasty can be significant, mounting up to a rate of 20-22% in some series [33, 34]. Early complications include the cardiovascular, thrombo-embolic, and respiratory complications that are associated with any major abdominal surgery. Early bowel-related complications include small bowel leak, ileus, or bowel-bladder anastomotic leak and fistula formation.

Voiding dysfunction

One of the major problems of patients undergoing enterocystoplasty is the high incidence of postoperative voiding dysfunction. A priori cystoplasty by dissecting the detrusor causes some degree of outflow impediment. In the majority of patients that have difficulty voiding postoperatively, the urethra sphincter mechanism fails to adjust to the creation of a low-pressure bowel substitution cystoplasty. As a result most patients have to strain to empty their bladder during the early postoperative period, and effective voiding by abdominal straining make take up to 2-3 months for some patients [35].

In cases of voiding dysfunction, a simple voiding cysto-urethrogram (VCUG) or an ultrasound voiding cysto-urethrogram is the investigation of choice for the evaluation of the reason for insufficient voiding. This examination can reliably locate the site of a possible outlet obstruction (bladder neck, urethra) and provide information for appropriate management.

In a cohort of patients estimated to be around 20% voiding will only be sufficient after a rebalancing procedure is undertaken

in order to reduce the urethral closure pressure while taking care not to create urethral incompetence [35]. Rebalancing is achieved in male patients by a bladder neck incision, an Ottis sphincterotomy or a combination of the two.

In cases where these rebalancing procedures fail to achieve effective voiding, then the patients will have to resort to self-catheterization. Clean intermittent self-catheterization (CISC) may be required for as briefly as a few weeks after the operation during the "balancing" stage. In general however, insufficient voiding and the need for CISC increases with time [36, 37].

Postoperative urine leakage

As was mentioned previously, despite detubularization and reconfiguration of the ileum some peristaltic activity may still persist. There are cases where persistent peristalsis of the ileal patch may lead to urine leakage during the early postoperative phase. It is usually the sigmoid colon and stomach that demonstrate increased peristaltic activity when used as patches into the native bladder.

Bladder hyperactivity might be another reason for leakage of urine after cystoplasty and can usually be effectively managed with anticholinergic medication. In case leakage persists postoperatively, the question should be raised regarding decreased outlet resistance. Once this is ruled out, re-augmentation of the bladder may be necessary using an additional patch of intestine. In a review of 323 patients following enterocystoplasty 5.9% were found to have high pressure bladder contractions postoperatively requiring some form of re-augmentation [38].

Late complications following enterocystoplasty

Late complications following ileocystoplasty can be broadly distinguished, with regard to etiology, to those related to the disruption of the gastrointestinal tract and to those that arise from the interposition of a segment of ileum into the native bladder. As any technique of bladder augmentation requires access to the peritoneal cavity, there is an increased risk of adhesions formation and bowel obstruction, as well as alteration in bowel habits.

Although the exact incidence of bowel obstruction from adhesions in the general population is not known, it was estimated to account for 10% of pediatric patients undergoing ileocystoplasty [39].

Bowel dysfunction is one of the most frequent major long-term complications following augmentation cystoplasty. Alteration in bowel habits, particularly frequency and diarrhea, constitute a debilitating and persistent long-term complication of ileocystoplasty, with significant impact on the patient's quality of life [11, 40]. Up to 42% of patients experience long-term problems of increased bowel frequency and troublesome diarrhea, while a quarter of patients with neurogenic bladder dysfunction present with fecal incontinence after enterocystoplasty [11, 41]. In a recently published 8-year follow up study of patients treated with clam enterocystoplasty, 59% of patients still reported troublesome diarrhea. High rates of fecal incontinence (47%), fecal urgency (41%), and nocturnal bowel movement (18%) with significant consequences in the patient's quality of social and sex life were also reported. The adverse impact of bowel dysfunction on everyday activities and quality of life was reported to be more severe after clam enterocystoplasty, with 24% of patients regretting undergoing the procedure [42]. The pathogenesis of post-augmentation diarrhea is not clear. A reduction in small bowel absorption may in part account for the increased frequency and diarrhea, although patients with an ileocystoplasty rarely have >30 cm of ileum resected [4].

Late complications associated with ileocystoplasty related to the interposition of an enteric segment into the bladder share a common etiology, namely the functional and metabolic mismatch between the bladder urothelium and the interpositioned bowel

mucosa which is anatomically presumed to act as bladder although physiologically remains a secreting endothelium. For instance, although the urothelium is impermeable to most electrolytes, the ileum is, on the contrary, designed to absorb electrolytes and secrete mucus. Therefore this "conflict of interest" is the original source for the majority of long-term complications that patients experience after enterocystoplasty.

Metabolic disturbance

The incorporation of a bowel segment into the bladder results in exposing the absorptive surface of ileum to urine metabolites. The consequence is a certain degree of reabsorption of water, sodium, hydrogen ions, ammonium, and chloride as well as an increased loss of potassium and bicarbonate into the urine [43, 44].

This chronic effect may lead to the disturbance of the acid-base balance and the development of hyperchloremic metabolic acidosis. However this complication almost always runs subclinical and only in rare symptomatic cases may require oral bicarbonate supplementation [6, 45]. Chronic metabolic acidosis causes the mobilization of calcium carbonate from bone and may lead to orthopedic problems or growth reduction in the pediatric population [46]. An advantage of ileum over sigmoid colon is that ileum can reabsorb urinary calcium and therefore ileocystoplasties are less prone to this complication.

Vitamin B12 deficiency may occur in the rare event that large segments of terminal ileum are used, in that case long-term follow-up for hematological sequelae is mandatory [47]. Metabolic changes like hypocitraturia, hypercalciuria, and mild hyperoxaluria may also accentuate the risk of forming oxalic or phosphate calcium stones [48].

Mucus formation

Mucus build-up after enterocystoplasty is a problem that also arises from the interposition of small bowel into the bladder and has been reported to occur to as many as 81% of patients [49]. The average daily mucus production from ileum when used as a cystoplasty patch is 35-40 g, and this amount does not reduce substantially over time, despite a time-related villous atrophy of the intestinal patch mucosa [4].

Preventive measures include proper patient instruction to perform weekly or even daily bladder wash out [50, 51]. According to a recent study oral ranitidine, aspirin, or the instillation of water soluble N-acetylcysteine into the bladder have not been proven efficacious in reducing the amount of mucus produced, despite previous encouraging results with the use of N-acetylcysteine [52, 53].

Recurrent urinary tract infections (UTIs)

The trouble of recurrent UTIs after augmentation cystoplasty has a critical impact on the outcome of the procedure, due to the added morbidity of recurrent infection, hospitalization, and long-term antibiotic treatment. Although recurrent bacteriuria can be found in more than 75% of patients after augmentation cystoplasty, the incidence of symptomatic UTI is lower [4, 40, 54]. Bacteriological data from patients who underwent clam ileocystoplasty revealed that 84% of patients on CISC had positive cultures, while bacteriuria was present in 60% of patients who voided spontaneously [55]. Predisposing factors to the development of UTIs, other than the high bacterial content of bowel, include large residual volumes, the presence of mucus, and the need for CISC. In the follow-up of a group of 48 patients after clam enterocystoplasty, 37% were found to suffer from recurrent UTIs more than 1 year postoperatively [49].

However, following enterocystoplasty and despite the continued need for CISC, there are fewer episodes of symptomatic UTIs

probably owing to the beneficial effect of lowering the intravesical pressure. Therefore, the actual incidence of significant UTIs after cystoplasty does not exceed that seen in patients with ileal conduits or indwelling urinary catheters [6].

Malignant transformation

Ileocystoplasty carries a low, although significant, risk of malignancy while cancers arising within augmented bladders are aggressive and associated with significant mortality [56, 57].

Tumors arising in ileocystoplasties are usually located at the enterovesical anastomosis or on the intestinal side of the bladder augmentation [58]. Since the intact ileum rarely undergoes malignant transformation *per se*, urine stasis within the augmented bladders together with the larger area of juxtaposition between transitional bladder and enteric epithelium are factors that may lead to the formation of carcinogens and trigger malignant transformation [57].

The majority of tumors arising in ileocystoplasties are adenocarcinomas of the bowel or bladder and squamous cell carcinomas, although cases of transitional cell carcinomas (TCCs) have also been reported [56, 59].

The etiology of malignant transformation in augmented bladders is probably multi-factorial. One of the predisposing factors for malignant transformation was postulated to be the elevated levels of urinary nitrosamines [60]. The basic idea is that nitrates, which are normally excreted in the urine, are degraded to nitrites by colonic bacteria. Nitrites then react with urinary secondary amines to form N-nitrosamines. These compounds have the capability of carcinogenic activity and, therefore may act as tumor initiators [57].

Surveillance for malignancy

There is controversy as to what should be the appropriate surveillance protocol for malignant transformation for patients undergoing bladder augmentation. However, with the time interval between cystoplasty and the occurrence of tumor being highly variable, lifelong surveillance is recommended [61-63].

Initial recommendations focused on elimination of urinary infection and suggested annual cystoscopy [35]. However, it seems unlikely that every patient with an enterocystoplasty is at high risk for developing a life-threatening tumor, and malignancy in augmented bladders is still a rare incidence [59, 64]. Moreover as the tumors described in the literature have presented within a latency period of 5 to 29 years (mean 18 years), some authors suggest that lifelong cystoscopic follow-up should start no earlier than 3 years after initial surgery [57, 65].

Perforation

Spontaneous perforation of the augmented bladder may occur in 5-13% of patients, and is associated with significant (>25%) mortality [66-68]. Perforation is more common in patients with NBD [69], recurrent UTIs or artificial urinary sphincter (AUS) implantation and in those under CISC, due to infrequent or traumatizing bladder catheterization. The perforation site is thought to be located at the anastomotic suture line between the bowel and the native bladder although this is not always the case [70-74].

Bladder over distention due to clot retention or obstruction from mucus and the resultant high intravesical pressures can cause transient localized microvascular occlusion and local ischemia [72, 73, 75]. This may lead to the creation of an ischemic scar tissue which can easily rupture after an abrupt increase in pressure.

However, other experimental studies support that it is the increased capacity of the augmented bladder and increased wall tension that lead to a reduction in bladder wall thickness and perforation [76]. Another possible reason is that repeated bladder infections cause chronic inflammatory changes, weakening of the bladder walls, and perforation.

Bladder perforation is a feared complication because in cases of delayed diagnosis the resultant peritonitis may lead to severe shock and death [66]. Abdominal pain is usually the first symptom and may be accompanied by fever. Shoulder pain due to irritation of the diaphragm from intraperitoneal urine may be the initial symptom [67].

Diagnostic delay is more of a problem in neurologically impaired patients, who due to altered sensation may develop peritonitis and intra-abdominal abscesses in the absence the signs and symptoms of acute abdomen [69, 77]. In cases of intra-abdominal perforation of an augmented bladder, signs and symptoms of acute abdomen prevail, necessitating abdominal exploration, regardless of the results of imaging studies.

Ultrasound and CT usually reveal urinary extravasation, either into the local pelvic tissues or the peritoneal cavity. Cystography may give false negative results in case of a suspected augmented bladder perforation and should not be relied upon for the establishment of diagnosis of perforation [77].

In cases where imaging shows no evidence of major extravasation, and there is absence of signs or symptoms of acute abdomen, conservative management with catheter drainage and antibiotics can lead to successful resolution in more than half the cases (6). However in patients on CISC whose urine is a priori infected, conservative management may prove inadequate and surgical exploration may be required [78].

Stone formation

Stones in the augmented bladder are common after cystoplasty and seem to occur with increasing frequency as the follow-up lengthens. The incidence of stones forming in enterocystoplasties is reported to range from 13-30% in contemporary series [4, 40, 54, 79].

Stones can cause bladder irritation and patients may present with incontinence, hematuria, or urinary tract infection, although they may be asymptomatic and diagnosed on routine follow-up ultrasound [6]. It has been noted that patients with an augmented bladder that is emptied by 'natural' voiding seldom (~2%) form a stone [4]. On the contrary, stones occur five times more often in patients with augmented bladders who are on CISC via an abdominal stoma, implicating stasis as an important factor for stone formation [80].

Other risk factors for stones include infection, immobilization, and the presence of intravesical foreign bodies such as staples or mesh. In patients with enterocystoplasty the risk of forming stones, especially triple phosphate stones, is mainly attributed to the high incidence of recurrent urinary tract infections usually from urea-splitting bacteria [79].

Regarding treatment, some form of endoscopic or open surgical procedure is necessary since stones in intestinal reservoirs are not amenable to medical treatment, may act as a nidus for infection and have a tendency to enlarge if left *in situ*. Any intervention contemplated must be tailored to the size of the stone and the type of bladder augmentation performed, however patients with large or multiple stones and those with no urethral access require open surgery for stone removal [80].

Renal function deterioration

Although augmentation cystoplasty aims at preserving the upper urinary tract and renal parenchyma from high pressures transmitted from the bladder, there is always the risk of deterioration of renal function following augmentation cystoplasty. This can be attributed to recurrent urinary tract infections or inadequate bladder emptying, with patients with elevated baseline creatinine levels being at a higher risk.

Table 2. Early and late complications following bladder augmentation.

Early complications	Late complications
Voiding dysfunction	Metabolic disturbances
Post-operative urine leakage	Mucus formation
Surgical complications	Recurrent UTI
	Malignant transformation
	Perforation
	Stone formation
	Renal function deterioration

Results from a 10-year follow up study after augmentation cystoplasty in 53 patients showed that 19% of patients demonstrated deterioration in renal function expressed by a decrease in glomerular filtration rate (GFR) of more than 20%. The most common reason for this decline in renal function was chronic retention or infection caused by inadequate catheterization in poorly compliant patients [81].

Table 2 summarizes the major early and late complications following augmentation cystoplasty.

CONCLUSIONS

Bladder augmentation is currently the surgical treatment of choice for patients with intractable non-neurogenic urgency incontinence unresponsive to minimally invasive treatments, as well as for the pediatric or adult patient with a high pressure, low compliance neurogenic bladder.

However, augmentation cystoplasty is a major abdominal intervention with significant and potentially severe complications and by no means is a "fit and forget" procedure. On the contrary it requires careful patient selection, lifetime patient commitment, and lifelong follow up and physician alert for the prevention and early identification of complications.

It is probably true that the variety and incidence of complications following augmentation cystoplasty is more a matter of material rather than of surgical technique. Latest advances in the field of tissue engineering will hopefully in the near future provide us with a material that will neither absorb electrolytes nor secrete mucus; will be compliant, distensible, endoscopically accessible and cheap. In other words, the perfect bladder patch.

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