## Bladder Necrosis Following Hydrodistention in Patients With Interstitial Cystitis

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**Purpose:** Bladder hydrodistention is used to diagnose and treat patients with interstitial cystitis. This procedure has been shown to have minimal morbidity and provide symptomatic relief in a subset of patients with interstitial cystitis. We report our experience with almost total bladder necrosis after hydrodistention at 2 institutions. To our knowledge this rare complication has not been previously reported in the literature. We also reviewed the literature regarding complications of hydrodistention and discuss their possible etiology.

**Materials and Methods:** We report 3 cases of bladder necrosis after therapeutic hydrodistention for interstitial cystitis at 2 institutions. All records were reviewed, and the clinical presentation, findings and treatments are discussed. A literature review was performed to evaluate the effectiveness and complications of hydrodistention for interstitial cystitis.

**Results:** There were 2 female and 1 male patient between ages 29 and 46. All patients had a previous diagnosis of interstitial cystitis and had been previously treated with hydrodistention. All patients presented with severe abdominal pain and had necrosis of the entire bladder wall with sparing of the trigone. Two patients were treated with supratrigonal cystectomy. A review of the literature revealed little data on the effectiveness of hydrodistention for interstitial cystitis.

**Conclusions:** Vesical necrosis is a rare but devastating complication of hydrodistention. It can occur in young patients in the absence of a contracted bladder and it usually presents as severe postoperative abdominal pain. At exploration bladder necrosis with sparing of the trigone was observed. All patients required enterocystoplasty.

Key Words: bladder; cystitis, interstitial; necrosis; complications

ainful bladder syndrome/IC is a debilitating disease defined by the International Continence Society as "suprapubic pain related to bladder filling, accompanied by other symptoms such as increased daytime and nighttime frequency in the absence of infection or other pathology." In 1987 the NIDDK established a set of diagnostic criteria to be applied to research subjects with IC.2 Although the NIDDK criteria were intended for research, hydrodistention has been generally accepted as a means of diagnosing this condition. Hydrodistention was used as a treatment modality long before it was used for diagnosis. Although few groups have evaluated its effectiveness and its mechanism of action is poorly understood, a subset of patients who undergo this treatment experience significant symptom relief for a variable period.3 The treatment is believed to be safe with minimal complications.

We evaluated 3 cases of almost total bladder necrosis after cystoscopy and hydrodistention using anesthesia at 2 institutions. To our knowledge this complication has not been previously reported in the literature. In addition, we reviewed the effectiveness, complications and mechanism of action of cystoscopy and hydrodistention.

#### **METHODS**

We retrospectively reviewed the medical records of 3 patients with a diagnosis of IC who had bladder necrosis after therapeutic hydrodistention. Clinical presentation, surgical and pathological findings, and treatments are described. In addition, we performed a literature review of the effectiveness, complications and mechanism of action of cystoscopy and hydrodistention as treatment for IC.

#### RESULTS

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There were 1 male and 2 female patients between ages 29 and 46. All patients had a long history of refractory IC symptoms and all had previously undergone hydrodistention with some therapeutic response.

All patients experienced total bladder necrosis sparing the trigone after the hydrodistention procedure. The table lists patient demographics, procedure details and final findings. All patients experienced severe abdominal pain post-operatively. Two patients complained of abdominal pain immediately after the procedure and the man had abdominal pain after the Foley catheter was removed a few days after the procedure. Initially they were all treated with Foley catheter drainage and pain medications. Abdominal pain was intractable, requiring narcotic analgesics. Cystogram in

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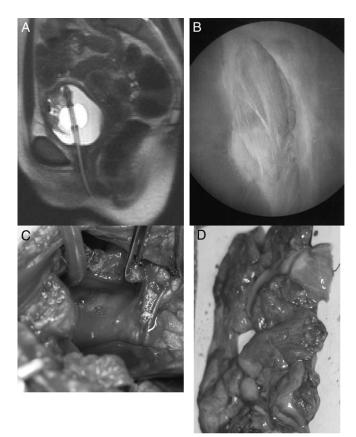
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Patient demographics and surgical findings			
	Pt 1	Pt 2	Pt 3
Age	46	29	44
Sex	$\mathbf{F}$	F	M
History (yrs)	IC (20)	IC (7)	Symptoms (12), IC (2)
Procedure	0.9% Saline hydrodistention bladder biopsies, 1,000 cc 0.4% chlorpactin in aliquots	0.9% Saline hydrodistention, bladder neck dilation	0.9% Saline hydrodistention $\times$ 2, 1,000 cc 0.4% chlorpactin in aliquots
Pressure (cm H <sub>2</sub> O)	80	100	80, 125
Duration (mins)	7		7, 5
Findings after hydrodistention	Petechial hemorrhage, terminal hematuria	0.5 cm Submucosal hemorrhage	Mucosal splitting extending into muscularis, terminal hematuria
Bladder capacity (cc)	475	500	650
Foley catheter	Yes	No	Yes
Final findings	Bladder necrosis sparing trigone	Bladder necrosis sparing trigone	Bladder necrosis on cystoscopy sparing trigone
Treatment	Supratrigonal cystectomy, enterocystoplasty	Supratrigonal cystectomy, enterocystoplasty	Refused
Outcome	Continued IC symptoms	Continued IC symptoms	Lost to followup

2 patients initially failed to demonstrate bladder extravasation. The 2 studies were suboptimal because only 75 and 100 cc contrast material, respectively, were instilled, likely due to patient discomfort. One of these patients had abnormal magnetic resonance imaging, which showed debris in the bladder (part A of figure).

These 2 patients underwent surgical exploration. They were found to have full-thickness necrosis of the vesical wall involving most of the bladder but sparing the bladder neck and trigone. The bladder wall, which was partly adherent to



Imaging at presentation. A, magnetic resonance imaging shows debris in bladder next to Foley catheter. B, cystoscopy demonstrates necrotic tissue. C, intraoperative photograph reveals bladder segment after débridement. D, surgically excised necrotic tissue.

the anterior abdominal wall, was gray, necrotic and friable without bleeding edges. All of this tissue was débrided until healthy, bleeding tissue was observed (parts C and D of figure). Each patient underwent augmentation cystoplasty. Pathological evaluation revealed transmural infarction with denudation of the mucosa and ischemic necrosis of the muscular wall, including necrosis of the perivesical fat, and acute and chronic inflammation. Unfortunately these 2 patients continued to have IC symptoms after cystoplasty.

In the third patient cystogram showed extravasation. He was originally treated with a Foley catheter. After 3 weeks of catheter drainage extravasation persisted. Cystoscopy showed a well demarcated, yellowish-gray, necrotic-appearing bladder wall, sparing the bladder neck and trigone (part *B* of figure). Although the patient was counseled regarding bladder reconstruction, he refused treatment and was lost to followup.

#### **DISCUSSION**

Although cystoscopy and hydrodistention have been used for almost a century to treat and diagnosis IC, their mechanism of action remains unknown. It is suggested that bladder distention leads to damage of the submucosal nerve plexus and stretch receptors, and decreases sympathetic nerve density, leading to decreased pain sensation.4 Another postulated theory is that hydrodistention may cause mast cell degranulation, at once resulting in symptom relief until new mast cells repopulate the bladder wall. In addition, in vitro stretch of bladder cells was shown to cause biochemical alterations in bladder physiology, such as increased production of nerve growth factor, parathyroid hormone related peptide, heparin binding epidermal growth factor-like growth factor and adenosine triphosphate. Furthermore, hydrodistention to 80 cm water was shown to increase urinary heparin binding epidermal growth factor-like growth factor and decrease antiproliferative factor activity, reversing factors implicated in the pathophysiology of IC.5,6

Hydrodistention was first described as a potential treatment modality in 1922 and it was later popularized after the publication of the National Institutes of Health, NIDDK diagnostic criteria in 1987.<sup>2</sup> Although the criteria were originally described as a means to better define research populations, they were then erroneously widely used to diagnose

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