

## Spontaneous Perforation of Augmented Bladder after Exstrophy Repair

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**Key Words.** Rupture · Exstrophy · Bladder augmentation

**Abstract.** A patient with spontaneous perforation of an augmented bladder following exstrophy repair is presented. Apart from the clinical presentation the various causes for augmented bladder perforation are discussed and the literature reviewed.

### Case Report

In April 1990, a 13-year-old boy was admitted to the hospital because of severe abdominal pain of sudden onset. There was a history of surgical procedures for bladder exstrophy and epispadia. Following primary exstrophy closure in February 1977 in London, a Schrott bladder neck plasty and a penile plasty were performed in June 1981. Because of minimal bladder capacity and complete incontinence, an augmentation cystoplasty and implantation of an artificial genitourinary sphincter (Scott-AMS 800) were carried out in 1986. Finally the urethra, which had primarily turned into a hypospadias, was reconstructed with a Duckett procedure.

On physical examination the abdomen appeared tense and tender with diminished bowel sounds. Voluntary guarding and rebound tenderness were noted. Apart from a white cell count of 16,400/nl, the laboratory findings were normal. There was no fever. Ultrasonic examination revealed free fluid in Morison's pouch. A plain film of the abdomen in the upright position was consistent with partial ileus. Apparently bladder function and micturition were normal before admission, therefore cystography was not performed.

Laparotomy disclosed local peritonitis close to the sigmoid colon due to a 1 × 1-cm full-thickness perforation of the anterior wall of the intestinal segment used for ileocystoplasty. The perforation was excised and closed with a double layer of sutures as were two other atrophic lesions of 3 × 5 mm in the intestinal segment.

Postoperatively meclocillin, netilmicin-sulfate and metronidazole were begun and the patient fully recovered without sequelae.

### Discussion

Indication for augmentation cystoplasty is given for functional and organically reduced bladder capacity. Among the major causes are chronic recurrent urinary tract infection, interstitial cystitis [1], irradiation sequelae

[2], neurogenic bladder dysfunction [3] and bladder exstrophy [4].

Surgical management of bladder exstrophy has substantially changed in the last 80 years. Early attempts at reconstruction with unsatisfying results [5] subsequently led to primary urinary diversion into the sigmoid colon (ureterosigmoidostomy), which was favored for a long time as the therapy of choice [6].

Due to the poor long-term results concerning this technique [7-10], surgeons have reverted to primary exstrophy closure in the last 10 years [11, 12]. According to Gearhardt and Jeffs [13], it is sensible to perform augmentation cystoplasty 2-3 years following exstrophy closure in case the bladder capacity failed to increase adequately.

For augmentation enterocystoplasty part of the ileum, colon and ileocecal segment can be used. The first augmented ileocystoplasty was performed by Mikulicz [14] in 1899. In the last 30 years various surgical techniques for ileocystoplasty have been favored: the 'Camey bladder' [15], and the cup-patch ileocystoplasty or 'clam-ileocystoplasty' [16, 17]. The techniques differ in that a tubular ileal segment is applied in the first procedure and detubularized ileum used in the other.

Augmentation cystoplasty requires substantial surgical effort. After a 15-year experience with augmentation cystoplasty, Whitmore and Gittes [18] reported on the necessity for additional surgical intervention in 40% to achieve an acceptable result.

The most frequent complications following augmentation cystoplasty are electrolyte disorders, residual vol-

ume, incontinence, vesicoureteral reflux, infection and the occurrence of carcinoma [19]. Spontaneous perforation after augmentation cystoplasty is hardly ever reported [20].

The major risk factor for bladder perforation in these patients is undoubtedly intermittent self-catheterization. Thus traumatic perforation of the intestinal wall used for cystoplasty is conceivable and has to be taken into account in the diagnostic procedure. In our patient, who was supplied with an AMS-genitourinary sphincter, self-catheterization could be omitted.

A high intraluminal pressure of up to 100 cm H<sub>2</sub>O in the tubular intestinal segments represents a further risk factor for spontaneous perforation [21–24]. However, in this case a detubularized ileal segment was used for augmentation cystoplasty.

Furthermore, thinning of the denervated bladder wall and detubularized intestinal segment, respectively, can be discussed as a result of long-standing expansion caused by increased filling thereby leading to high intravesical pressure. Our patient reported to have emptied his bladder merely twice daily before admission. The idea of pressure-induced cystoplasty damage is further supported by the intraoperative findings of atrophic segments. However, a histological examination of the perforated segment was not performed.

Preoperatively a cystography to demonstrate the augmented bladder, as recommended by various authors [25], was not obtained. The indication for laparotomy was based solely on the patient's history, clinical examination, laboratory data, sonography and a plain film of the abdomen in the upright position.

The postoperative bladder capacity was 650 ml with detrusor contractions not exceeding 20–30 cm H<sub>2</sub>O. The patient was advised to void at regular intervals of 2–3 h in order to prevent a bladder volume of more than 300 ml.

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