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Bilateral Ureteral Obstruction After Appendectomy in Children

By C.A.C. Hugen, P.F.A. Mulders, L.A.H. Monnens, R.H.M. Dijkman-Neerinxc, and J.D.M. de Vries

Nijmegen, The Netherlands and Arnhem, The Netherlands

Five cases of bilateral ureteral obstruction after appendectomy are presented. All five patients were boys (age range, 9 to 15 years). All of them had had severe appendicitis. Based on the urothrocystoscopy findings, edema of the posterior bladder wall appeared to be the cause of obstruction of both distal ureters. This is confirmed by the immediate recovery of renal function after installation of bilateral uretercatheters. It is known that contamination of the peritoneal cavity can occur by organisms leaking from a gangrenous or perforated appendix. This can cause localized inflammatory edema of the posterior bladder wall. It is remarkable that through ultrasound investigation, only mild to moderate dilatation of the urinary tract was observed. An explanation can be obtained from animal models, wherein acute obstruction of the ureter leads only to a transient increase in ureteral pressure, followed by a decline toward the preobstruction level. It is important to be aware that this complication can occur after appendectomy; bilateral uretercatheters can be installed, and irreversible renal damage can be avoided.

INDEX WORDS: Anuria, renal failure, bilateral ureteral obstruction, appendicitis.

COMPLETE ANURIA after appendicectomy for acute appendicitis is an exceptional and alarming complication. Possible causes include septic shock, severe dehydration, or antibiotic-induced nephropathy. We describe five cases in which the complication arose from bilateral distal ureteric obstruction, seemingly caused by edema of the bladder wall.

CASE REPORTS

The clinical features of our cases are summarized in Table 1. Case 2 is described in more detail as being representative of the series. This 11-year-old boy presented with a 24-hour history of vomiting and right iliac fossa pain. He was moderately toxic, febrile (38.6°C), and normotensive. A perforated appendix was excised on the day of admission. He received antibiotics preoperatively and for 3 days subsequently (gentamycin 60 mg and metronidazole 250 mg, both three times daily). Recovery was uneventful, with normal urinary output (1,600 mL/d) until the sixth postoperative day, when sudden, severe, generalized abdominal pain with vomiting occurred, and urinary output decreased to 216 mL/d, followed by complete anuria on the seventh postoperative day. He was afebrile and did not have significant abdominal signs. Urinalysis showed more than 200 red blood cells per high-power field. The result of a plain abdominal radiograph was normal, and an ultrasound examination showed only slight renal pelvic dilatation bilaterally. The anuria persisted for another 24 hours, by which time his serum creatinine level had increased to 326 μmol/L. In light of possible obstruction at the level of the distal urinary tract, urothrocystoscopy was performed. During inspection of the bladder, edema was seen at the trigone, extending to both sides of the bladder. Uretercatheters were inserted bilaterally. We used 3F uretercatheters fixed on a transurethral balloon catheter. The patient became polyuric. The uretercatheters were kept in situ during the polyuric phase until urine production normalized; this took 2 days in our cases. During ultrasound investigation a few days later, no dilatation of the pyelum was noted. His serum creatinine level normalized and he went home in good health.

Case 1 was referred from another hospital for hemodialysis after 3 days of anuria; because the anuria resolved spontaneously and soon after the transfer, intervention was not necessary.

DISCUSSION

Although right-sided hydroureteronephrosis (and occasionally bilateral dilatation) has been recognized in patients with appendicular abscess formation, this has not been further complicated by anuria. The first case of obstructive anuria after appendicectomy for acute appendicitis was described in 1974. Another case was reported in 1978. Recently, an article in Lancet described three boys with this complication. Features common to all five of our cases were male gender, severe appendicitis, anuria that occurred between the fifth and eighth postoperative days, and microscopic hematuria. The prompt response to therapeutic intervention is proof of an obstructive etiology, which appears to have been related to edema of the posterior bladder wall, the cause of which is uncertain in the apparent absence of ongoing pelvic sepsis. The comparatively late onset of the anuria is suggestive of slow progression of the edematous process, and the hematuria presumably occurred from inflammation of the urinary tract mucosa. It is possible that the edematous process itself was triggered by a localized peritoneal reaction to intraoperative bacterial contamination. Perhaps boys are more susceptible to the complication because their appendix is situated closer to the bladder; in girls, the internal genitals (especially the mesosalphinx and the ligamentum latum uteri) are located between the appendix and the bladder.

Because acute appendicitis is not common in young children (the highest incidence is in the 10- to 19-year age group; peak incidence for boys is at 10 to 14 years of age), the age distribution of our group appears to

From the Departments of Paediatrics, and Urology, Academic Hospital Nijmegen, and the Department of Paediatrics, Rijnstate Hospital Arnhem, The Netherlands. Address reprint requests to P.F.A. Mulders, Department of Urology, Academic Hospital Nijmegen, Geer Grooteplein 16, PO Box 9101, 6500 HB Nijmegen, The Netherlands. Copyright © 1995 by W.B. Saunders Company 0022-3468/95/3012-0008$03.00/0
be logical. Another explanation for the male predominance may lie in epidemiologic features. Most series show that 1.2 to 2.3 times more boys than girls suffer from acute appendicitis. 

That the anuria was accompanied by only slight renal pelvic dilatation is noteworthy. One would expect ongoing dilatation of the renal pelvis after obstruction of both ureters. Experimental research showed that acute obstruction of both ureters in animal models leads only to a transient increase in pressure in the proximal ureter, followed by a gradual decline toward the preobstruction level. This fact appears to explain our ultrasound findings. In clinical practice, despite the fact that only slight abnormalities are noted through ultrasound examination, one should include postrenal obstruction in the differential diagnosis of anuria after appendectomy and take action accordingly.

Installation of bilateral uretercatheters is the appropriate treatment in these cases. If the edema of the bladder wall is very severe, it is possible that one might not find the ureter-ostia, and a percutaneous nephrostomy may be necessary. This will be followed by a polyuric phase with natriuresis and increased excretion of magnesium. Although there was spontaneous resolution in case 1, it is not advisable (and is even dangerous) to watch the levels of serum urea, creatinine, and potassium increase significantly.

In the dog, serial glomerular filtration rate (GFR) studies have been made after 1 week of complete unilateral ureteral obstruction; in no case was there complete functional recovery of the obstructed kidney. In experiments with rats, a release of ureteral obstruction after 30 hours resulted in complete recovery of the GFR. However, if ureteral obstruction had been present for more than 72 hours, a permanent decrease in GFR occurred. In humans, the length of time before irreversible kidney damage occurs is not known; thus, how long one can wait to install the bilateral uretercatheters also is uncertain.

REFERENCES