Hypothesis – a congenitally lax pubourethral ligament may be a contributing cause of vesicoureteral reflux

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KEY WORDS

vesicoureteral reflux

ABSTRACT

Introduction. The hypothesis derives from the field of female stress incontinence. Application of pressure on the anterior vaginal wall at midurethra with a hemostat restores the geometry of the vesicoureteral junction and continence.

Methods. We applied unilateral midurethral pressure during a radiological investigation of a 15-year-old female patient who had undergone 2 surgeries for ureteric reflux.

Results. On injection of the dye into the bladder, reflux was noted in the left ureter, and this disappeared within 2-3 seconds after pressure was applied on 2 successive occasions in the midurethral area of the vagina.

Conclusion. The hypothesis that a musculoelastic mechanism dependent on a competent pubourethral ligament may play a role in vesicoureteral valve closure appears to have been confirmed, at least in one case. Hopefully this observation will lead to further studies, and perhaps, new directions for therapy.

INTRODUCTION

There have been no new hypotheses for causation of vesicoureteral reflux for many years. The aim of this report is to present a new hypothesis, deriving from the field of female stress incontinence. In females with stress urinary incontinence, application of pressure on the anterior vaginal wall at midurethra with a hemostat restores the funneled geometry of the vesicoureteral junction to normal and continence [1]. The mechanism for this is based on a competent pubourethral ligament acting as a firm anchoring point for the three directional muscle forces that activate distal and proximal urethral closure (Fig. 1). Based on a report on improvement of vesicoureteral reflux in an adult female following a midurethral sling, it was hypothesized that a similar mechanism may act to prevent vesicoureteral reflux (Fig. 1) [2]. The ureters traverse the bladder wall to the trigone; the muscle forces (arrows) stretch the trigone backwards and downwards around a competent pubourethral ligament (PUL) to close off the proximal urethra, and ureterovesical junction.

We report on a serendipitous testing of this hypothesis.

PATIENT AND METHODS

A 15-year-old young woman presented with a long history of vesicoureteral reflux and chronic cystitis, treated with prophylactic antibiotic therapy. Symptoms during remission included, urgency abnormal bladder emptying, with residual urine volumes of up to 60 ml. A duplex system on the right side was corrected with an extravesical cystoneostomy (Gregoir-Lich). Because of continuing reflux, she had a 2nd operation of the right ureter duplex (Politano-Leadbetter).

The immediate reason for this admission was to exclude an upper renal calyceal bacterial focus for pyrexia not apparently due to bladder infection. Renal ultrasound indicated dilated right upper calyces, but no evidence of obstruction. Renal scintillography showed apparently decreased function in that area.

The management plan was to insert a ureteric catheter into the upper right renal calyx, and to take a sample of urine for bacterial culture and sensitivity. Radiopaque dye (250 ml) was injected into the bladder to guide the catheter. The test was applied as described previously (Fig. 1) [1].

RESULTS

There was no reflux observed into the right double system, but ureteric reflux was seen on the left side (Fig. 2). On cystoscopy, the urethra was normal, with no mechanical obstruction evident at the meatus, or anywhere along its length. Large complex trabeculae were seen in the bladder wall. The left orifice was "horseshoe" in shape, according to the classification of Lyon, and laterally displaced. When the forceps were unilaterally applied retropubically at midurethra Fig. 1, within 2-3 seconds the reflux had disappeared as documented fluoroscopically (Figs. 2 and 3) [3]. This was repeated on a 2nd occasion with the same results.

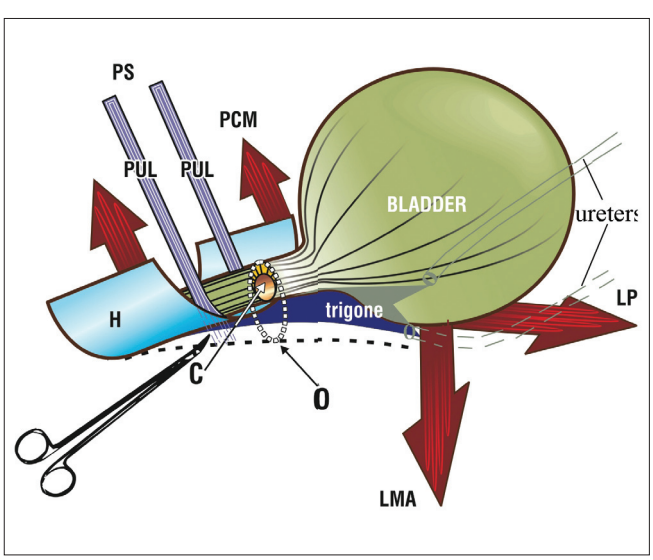

Fig. 1. A hypothesis for an adjunctive role of pelvic muscle forces in ureterovesical closure with three directional muscle forces (arrows): PCM (m. pubococcygeus), LP (levator plate), and LMA (longitudinal muscle of the anus). Vaginal hammock (H). Pubourethral ligament (PUL). Urethral diameter open position (O), closed position (C).

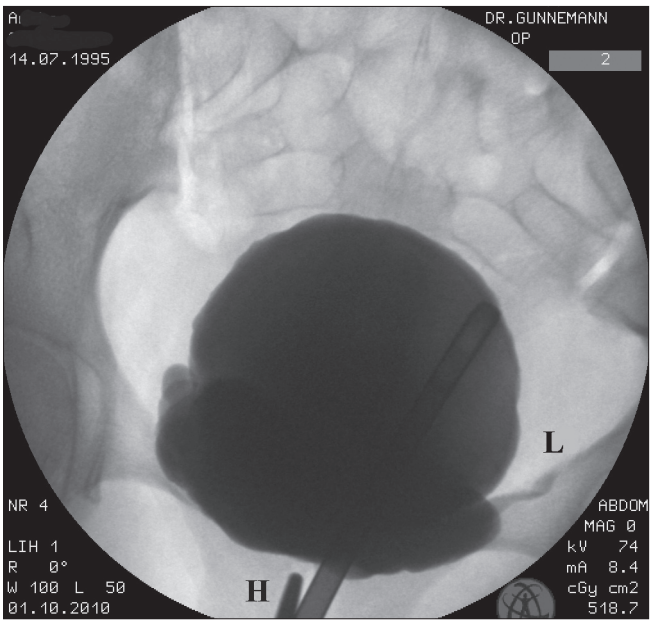

Fig. 2. Radiopaque dye injected into the bladder flows into the left ureter (L). The hemostat (H) is inserted, but no pressure has yet been exerted.

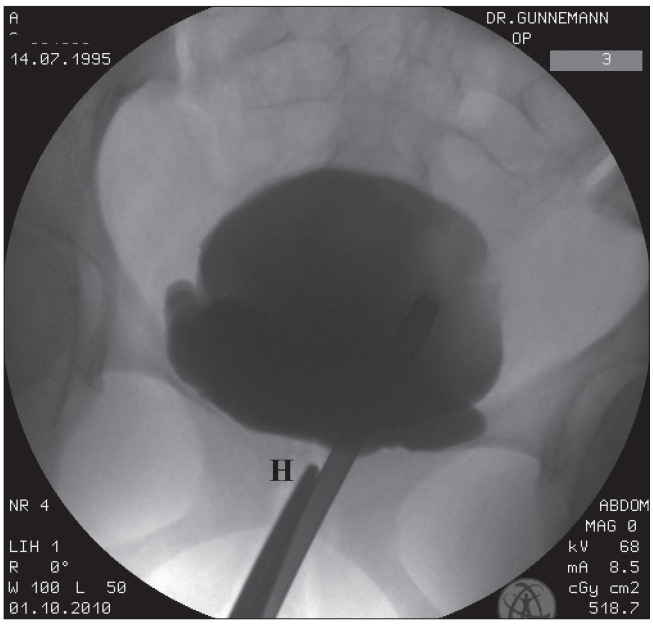

Fig. 3. Upward pressure on the anterior vaginal wall in the region of midurethra by a hemostat (H) prevents reflux.

DISCUSSION

According to a recent review, primary vesicoureteral reflux is the outcome of a congenital abnormality of the ureterovesical junction [4].

Our hypothesis (Fig. 1) is that a lax pubourethral ligament (PUL) may be the ultimate cause not only of reflux, but also of urge and stress symptoms in childhood.

The hypothesis for an adjunctive role of pelvic muscle forces in ureterovesical closure (Fig. 1).

The 3 directional muscle forces (arrows), PCM (m. pubococcygeus), LP (levator plate) and LMA (longitudinal muscle of the anus), stretch the hammock (H) forwards, and the trigone backwards/downwards to activate distal and proximal urethral closure [1]. It is hypothesized that this same action stretches the trigone and bladder base to assist closure of the ureterovesical junction. Pubourethral ligament (PUL) laxity inactivates these muscle forces, diminishing the backward stretching of the trigone, loosening the connective tissue/muscular junction sufficiently to cause vesicoureteral reflux. The forceps indicates point of upward pressure applied during the procedure, immediately behind symphysis pubis, at midurethra. In the stress incontinent patient, this action restores the urethral diameter from open (O) to closed (C).

We have seen many adult women and other family members with such childhood symptoms cured or improved at puberty. We attribute this to strengthening of the collagen component of the PUL by estrogen/testosterone. Those females who continue with problems into adulthood, respond well to a midurethral sling, which works by reinforcing the PUL [2, 5, 6]. Patients with ureterovesical reflux also improve at puberty. Based on our analysis of the biomechanics of all the structures in Figure 1, vagina, muscle forces, ureters, urethra, we concluded that the same musculoelastic mechanism that activates urethral closure, might also close the ureterovesical junction [1]. This closure mechanism (Fig. 1) relies entirely on a competent pubourethral ligament.

Clearly a midurethral sling is not appropriate for very young females. Excellent results have been achieved for urinary symptoms by encouraging squatting, and using a large rubber 'fitball' instead of a chair to strengthen the pelvic muscles and their ligamentous insertions [7].

CONCLUSION

The hypothesis that a musculoelastic mechanism dependent on a competent pubourethral ligament may play a role in vesicoureteral valve closure appears to have been confirmed at least in one case. Hopefully this observation will lead to further studies, and perhaps, new directions for therapy.

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Editorial comments to paper in this issue on pgs. 48-49

The article "Hypothesis – a congenitally lax pubourethral ligament may be a contributing cause of vesicoureteral reflux"

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The occurrence of vesicoureteral reflux (VUR) is the result of several pathologic factors. An important factor for normal function of the vesicoureteral junction (VUJ) is a proper anatomy of the intramural portion of the ureter, which is a functionally essential antirefluxing valve. The deficiency in this mechanism is often observed in cases of primary VUR, while other factors such as bladder and ureter functions are primarily normal. Secondary VUR is characterized by a normal anatomy of the VUJ, but often occurs in cases of abnormal function of the bladder or abnormal anatomy of the distal parts of the lower urinary tract. It is true that for many years there have been no new hypotheses formed for the causation of VUR.

The described case is a long treatment history of suspected primary VUR complicated by infections and abnormal bladder function (primary or secondary?). The nature of the occurrence of the left VUR in our opinion is mixed (primary and then secondary). The authors hypothesis that the cause of this VUR may be the congenital lax of the pubourethral ligament is interesting but controversial and not very well documented.

The value of the authors' paper lies in their conclusion that all mechanisms involved in the occurrence of VUR are not well known at this time and that there is value in future studies of musculoelastic parts of the bladder and urethra that may lead to new concepts of the VUR pathology and new concepts in therapy.

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