Are Midline and Paravaginal Defects the Cause of Bladder Prolapse?

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Abstract

Objective: To determine whether traditional defects such as midline and paravaginal defects are the real cause of bladder prolapse.

Methods: Biomechanical engineers and the author propose a new theory of the cause of bladder prolapse from vaginal birth. This theory suggests that tears in the pubocervical fascia from the pericervical ring during vaginal birth occur in a transverse direction and not in the midline or paravaginal (lateral) direction. These transverse defects were also discovered during cadaver dissections and in the operating room on 276 patients after MRI examination that did not reveal the bladder protruding through a midline or paravaginal defect, but from a transverse separation of the pubocervical fascia from the pericervical ring. IRB approval from Emory University was obtained.

Results: The bladder prolapse was only discovered herniating during surgery through a transverse defect in 276 patients with Stage 2 or greater prolapse and repaired via the vagina by reattaching the pubocervical fascia to the pericervical ring without synthetic mesh and with 95% success rates at 24 months.

Conclusion: The surgical correction of restoring the pubocervical fascia to the pericervical ring in our patient population appears to have long-term success rates in our patient population. However, multi-center studies and time will determine if this transverse defect repair will replace traditional midline or paravaginal repairs.

INTRODUCTION

The lifetime risk of having surgery for pelvic organ prolapse is at least 11.1%, and almost one out of every three women will need repair for recurrent prolapse [1]. Approximately 300,000-400,000 pelvic organ prolapse surgeries are performed each year in the U.S. with 30-40% for recurrences and 60% at the same site [2]. Why is anterior vaginal wall prolapse repair so difficult? For years I questioned whether midline defects and paravaginal defects were the real cause of anterior vaginal wall prolapse. If so, why the high failure rates of midline plication and paravaginal repairs?

Traditional anterior vaginal wall prolapse repair has a high failure/recurrent rate of 40 to 60% as the traditional techniques were based on the surgical knowledge of 100+ years ago. The goal of pelvic organ prolapse surgery at that time was to reduce the bulge, but not to correct the cause of prolapse.

It was finally time for me to critically evaluate what gynecologic surgeons have been taught for the past 100 years, from Howard Kelly’s concepts in 1913 to the beliefs of George White in 1909 and Culen Richardson in 1976. Our research centered on several questions that required further thought. First, is traditional anterior colporrhaphy (midline plication) the “gold standard” for repair of the prolapse anterior vaginal wall as some academics proposes? Second, is prolapse of the anterior vaginal wall a true hernia as suggested by Richardson and others [3]. Finally, how and where do vaginal births cause vaginal defects?

In 1976, Richardson described several defects that were accepted by most gynecologic surgeons [3]. He suggested a midline defect was “as an anterior-posterior separation of the fascia that occurs between the vagina and the overlying bladder and/or urethra. It results in a cystocele and urethrocele.” He further suggested “this defect is one of the most easily repaired, and excellent results can be expected with Kelly-Kennedy type procedures.” However, time has shown this repair to have unacceptable failure rates.

The lateral or paravaginal defect was described by Richardson as “a defect found in the fascia laterally at or near its attachments to the levator insertion in the lower margin of the superior pubic rami.” He further noted “that it usually results in a mild or moderate cystourethrocele, a loss of the urethrovesical angle, and significant stress urinary incontinence which could...
be unilateral or bilateral.” This defect occurs distal to the connection between the arcus tendentious fascia and the arcus rectovaginalis, and he believed it was the cause of stress urinary incontinence. Many researchers were not able to document the success of this procedure for correcting the incontinence issue. More importantly, surgeons somehow adopted a concept that a paravaginal defect extended from the pubis to the ischial spine that allowed the bladder to protrude through this space and was the cause of cystoceles. However, this was never described by Richardson.

Richardson also discussed a transverse defect that he identified as “a transverse separation that occurred in the pubocervical fascia from its insertion into the pericervical ring of connective tissue. He further stated, “It usually results in a large cystocele in which the bladder herniates beneath the mucosa of the anterior vaginal fornix. The urethra remains well supported with a good urethrovesical angle and there is rarely, if ever, stress urinary incontinence.”

Many gynecologic surgeons propose that reconstruction of anterior vaginal wall prolapse is really about hernia surgery. If so, the protrusion of the bladder through the tissues that are designed to contain them must be documented to be correctly considered a true hernia. The only hernia that Richardson identified was with a transverse defect. He did not describe hernias with midline or paravaginal defects, nor has our experience during surgery shown the bladder actually herniating through a midline or paravaginal defect. Therefore, unless the bladder can be identified protruding through a proposed defect, it is not a hernia so we questioned whether such repairs were appropriate.

Modern gynecologists displeased with the recurrence rates of Kelly, White, and Richardson procedures turned to mesh kits that ignored reconstruction of the defects and accepted a new concept from the medical industry to construct a mesh bridge for the centrally prolapsed bladder. The focus of mesh kits changed from reconstruction of long recognized and accepted theories of vaginal defects to simply replace reconstructive surgery with the formation of a mesh bridge promoted by the industry but basically untested in the pelvis. With these kits, there was supposedly no need to identify the fascial defects; instead, the surgeon just opened the vaginal epithelium, laid down the bolster, and then closed the epithelium. The concept that was promoted was that a permanent repair required a permanent biomaterial. When the complications from permanent mesh became more prevalent, the medical industry was forced by the FDA to remove them from the market, and gynecologic surgeons had no other choice but to return to traditional midline plication or paravaginal repairs regardless of their dismal recurrence rates to avoid erosions, pain, dyspareunia, and potential legal issues caused by vaginally inserted mesh.

Pelvic support defects are thought to occur, or at least be exacerbated, during the process of childbirth. Gynecologists have long believed that trauma to the supporting structures of the bladder leading to the vaginal prolapse were associated with vaginal birth. With clinical observation, there is little doubt that childbirth contributes to the likelihood that clinically symptomatic prolapse will occur. However, a major shortcoming of the profession is that the effects of labor and delivery to the female pelvis resulting in vaginal prolapse have never been fully studied or understood. Also little effort has been put forth to analyze the forces of childbirth, and also little proof in the past as to how and when, in the course of labor, the effects of childbirth occur as they relate to specific damage patterns found in the endopelvic supportive connective tissue [4]. We questioned whether the tears to the supportive tissues of the bladder during delivery really occur in a vertical (midline) or lateral (paravaginal) direction as most gynecologists have been led to believe.

**METHODS**

We thought the bony pelvic outlet and the pelvic supportive structures should be considered in mechanical terms. We considered these structures to be like a bridge; so when it fails, where does it fail and why does it fail required thoughtful analysis. We questioned do they really fail in the midline or is it laterally? After many years of studying this problem and with the assistance of biomechanical engineers, we theorized that during childbirth, descent of the fetal head to the level of the pericervical ring causes significant tensile and shear strain on the endopelvic fascia (pubocervical fascia) attached to the pericervical ring [5]. As the birth canal narrows at the level of the ischial spine, the narrowest diameter of the pelvis, stress and strain are significantly concentrated because the tissues the fetus passes through must undergo even greater deformation in order to accommodate the fetal head. Internal rotation of the fetal head occurs in order to present the optimal diameter of the fetal head to the bony pelvis, but we theorized that movement from the rotation of the fetal head induces transverse shearing forces onto the pubocervical fascia, already under high-loading strain caused by fetal descent. The strained and shearing forces can exceed the strength of the pubocervical fascia attached to the pericervical ring, resulting in soft tissue tears that affect the supportive tissues to the bladder. We theorized that the stress and strain to the pubocervical fascia occur in both a superior and inferior direction, and the internal rotation of the fetal head results in trauma separating the pubocervical fascia from the pericervical ring in a transverse direction with the bladder herniating in between (Figure 1).

This biomechanical modeling theory strongly suggests that tears to the pubocervical fascia during childbirth are more likely to occur as transverse tears to the pubocervical fascia from the pericervical ring rather than in the vertical direction in the midline or laterally (paravaginal tears). We concluded that transverse tears of the supporting tissues of the bladder were the most likely, and perhaps the only cause of anterior vaginal wall prolapse.

**RESULTS**

The validity of this theory was substantiated with cadaver dissections and in the operating theater. Patients who were clinically diagnosed with Stage 2 or greater prolapse with proposed midline and paravaginal defects preoperatively were studied with MRIs prior to surgery that did not document the bladder protruding through either a midline or paravaginal defect. This was also confirmed in the operating theater where the defect was documented and prolapsed found on each
occasion was a separation of the pubocervical fascia from the pericervical ring that was identified as causing the bladder to herniate between the separations.

The reasons for the poor result from midline and paravaginal repairs became obvious. Surgeons have been simply operating in the wrong place for many years. This theory represents a new, but anatomically correct, concept contrary to what gynecologic surgeons have been taught and believed for almost ten decades.

CONCLUSIONS

The results of the surgical correction using this concept of restoring the pubocervical fascia to the pericervical ring for anterior vaginal wall prolapse were surgically evaluated. Two hundred and seventy-six patients with Stage 2 or > prolapse underwent this new technique with follow-up of 24 months. Success rates of 95% demonstrate long-term success beyond midline and paravaginal repairs. Permanent synthetic mesh was not employed in any repairs. Multi-centered studies and time will tell if this new theory and operation to correct the transverse defect will replace traditional repairs and become a higher standard of care in the future. It is easy to recommend this theory and operation to correct the transverse defect as this repair returns the actual distorted anatomy to normal while providing sufficient apical support. Our results warrant further research to determine the etiology of bladder prolapse, and long-term treatment outcomes of these conditions.

REFERENCES