UROGYNECOLOGY

Origin and insertion points involved in levator ani muscle defects

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OBJECTIVE: This project sought to identify and to describe the anatomical connections affected by levator ani defects involving the puboviscera portion of the muscle.

STUDY DESIGN: Fourteen magnetic resonance scans of women with unilateral levator defects were selected. The missing muscle mapping technique was used to characterize the absent muscle. Normal muscle was visualized and compared with the contralateral side. Using a three-dimensional slicer, the outline of the intact muscle was traced; models of this muscle and surrounding structures were generated.

RESULTS: The missing muscle originates from the posterior pubic bone and extends laterally over the obturator internus muscle; it inserts into the vaginal wall, perineal body, and the intersphincteric space. Architectural distortion, with an asymmetric lateral spilling of the vagina was present in 50% of women. The defect was right sided in 71% of patients.

CONCLUSION: The origin and insertion points of the damaged portion of the levator ani muscle were identified.

Key words: levator ani defects, magnetic resonance, missing muscle mapping, puboviscera

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Pelvic organ prolapse (POP) is a prevalent condition, with more than 200,000 inpatient procedures performed each year in the United States.[1] An association between abnormalities in the levator ani and prolapse has been established.[2] Recently it has become possible to visualize each component of the levator ani on magnetic resonance (MR) scans,[3] namely, the puboviscera (further subdivided into pubovaginal, puboperineal, and puboanal), the puborectal, and the iliococcygeal subdivisions and their separate attachments. Each portion of this complex muscle has a unique origin-insertion pair that establishes its unique mechanical line of action. Damage to a specific portion of a muscle would be expected to result in loss of that force vector.

Our objective in this project was to determine: (1) the specific subdivision of the levator ani muscle that is affected by damage and (2) to identify the origin and insertion points, as well as the medial and lateral points of attachment, of the damaged portion of the levator ani muscle.

Materials and Methods

Our existing database of 676 pelvic MR scans was queried to select for all subjects with unilateral levator defects. Women with unilateral defects were chosen to study and compare normal and abnormal muscles in the same individual. The database consists of scans made of women enrolled in ongoing institutional review board–approved studies of pelvic organ prolapse and urinary incontinence (IRBMED #1995-0477; #1999-0395; #2002-0636). This library contains scans of women with POP, stress urinary incontinence (SUI), both prolapse and incontinence, and asymptomatic controls. Between 1997 and 2006, these subjects were recruited from newspaper advertisements; posted fliers; and the Women's Health Registry, a local database of women interested in participating in women's health research.

All women underwent magnetic resonance imaging (MRI) of the pelvic floor using proton density T-2–weighted scans; 2-dimensional fast spin proton density MR scans were performed at 5 mm intervals in the axial, sagittal, and coronal planes in the supine position using a 1.5 Tesla superconducting magnet (Signa; General Electric Medical Systems, Milwaukee, WI). In addition, a pelvic examination to evaluate prolapse and a multichannel urodynamic evaluation were performed. The mean age of the subjects in our database was 46.4 years (SD 15.4), mean body mass index (BMI) was 26.3 kg/m^2 (SD 5.5), and mean parity was 1.9 (SD 1.7). Ninety-three percent were white, 4% were black, and 3% were classified as "other." Race

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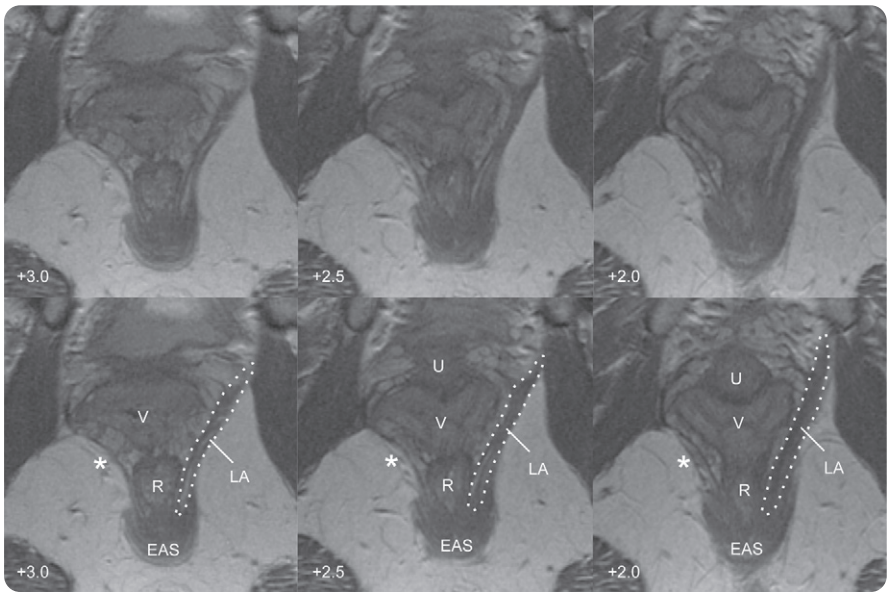
FIGURE 1
Exemplary axial MRI with unilateral defect



Axial MR scans of an exemplary 42-year-old female with a right-sided complete unilateral levator defect are shown. The intact levator ani muscle is traced (*dashed line,* labeled LA). The missing muscle is denoted (*asterisk*). Each panel is labeled with a number indicating the level of scan in centimeters relative to the arcuate pubic ligament; positive numbers indicate slices cephalad to the ligament. *EAS*, external anal sphincter; *R*, rectum; *U*, urethra; *V*, vagina. Printed with permission from John O. L. DeLancey, MD.

was determined by self-identification. In these ongoing studies, 155 women (23%) were recruited as cases of prolapse, and 154 (23%) were recruited as cases of stress urinary incontinence. The remaining 54% were asymptomatic controls.

Levator ani muscle defects in all MR scans had been scored by 2 independent investigators, using our previously described levator scoring system.[4] The left and right pubovisceral muscles are scored separately. Each muscle is assigned a score from 0 to 3. Normal muscle is given a score of 0. Injured muscle is given a score of 1 if less than half of the muscle is missing, a score of 2 if more than half the muscle is missing, and a score of 3 if complete muscle bulk is lost. The inclusion criterion for this study was a complete unilateral defect, defined as a levator score of 0 on 1 side and 3 on the opposite side. Our database was queried to select only patients with a complete unilateral defect; 14 subjects met this criterion.

To visualize the complex three-dimensional (3D) geometry of the levator muscle, axial, sagittal, and coronal MR images were imported into a 3D imaging program (3D Slicer, version 2.1b1, Brigham and Women's Hospital, Boston, MA) and aligned using anatomic landmarks. For all 14 subjects, 3D models of the following structures were created from axial scans: pubic bone, levator ani muscle, obturator internus muscle, urethra and bladder, vagina and uterus, perineal body, rectum and internal anal sphincter, and external anal sphincter. We then used the following technique of missing muscle mapping to characterize the absent muscle. In the axial plane, normal muscle was visualized on one side and compared with the contralateral missing muscle. We traced the outline of that portion of intact muscle whose corresponding mirror image was absent (Figure 1), and created a 3D model of the missing muscle (Figure 2).

These models were then evaluated alongside the original MR scans to detect lofting artifacts. Using the 3D models to evaluate the complex geometry of the intact and damaged muscle, we were able to determine the origin and insertion points of the missing muscle as well as relevant anatomic relationships and connections. In addition, we identified the specific levator subdivision[3] in which the defect was located and the side of the body on which the defect was seen.

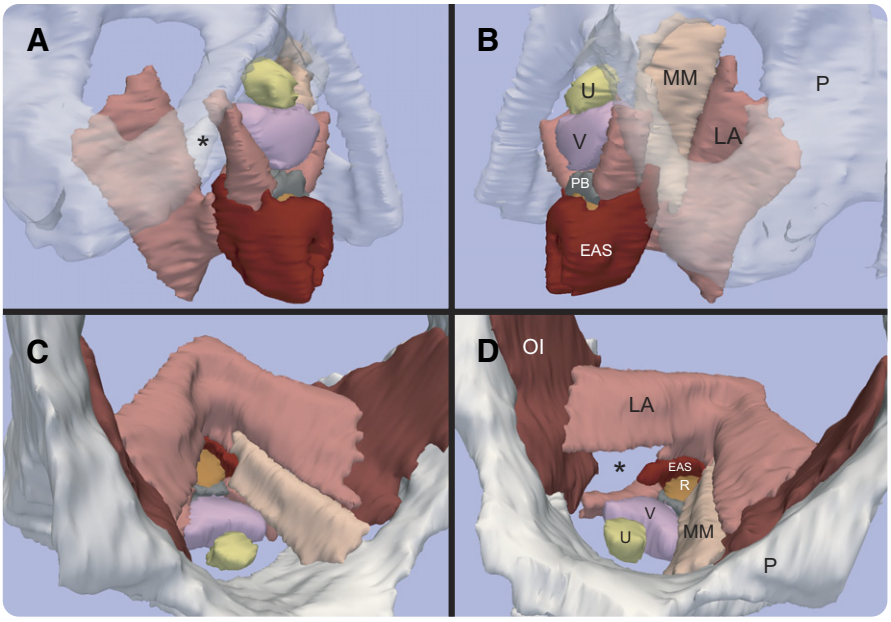
RESULTS

Within our study set, the mean age was 48.1 years (SD 12.6, range 19-65), mean BMI was 26.5 kg/m^2 (SD 4.6, range 19-36), and mean parity was 2.8 (SD 2.5, range 1-11). One hundred percent were white. Seven women (50%) had pelvic organ prolapse, with the leading point (any point on the vaginal wall or the cervix) at or beyond 1 cm outside the hymeneal ring; 8 (57%) had stress urinary incontinence that was demonstrated on physical exam (documented leakage with cough with a bladder filled to 300 mL). Five women (36%) had both POP and SUI, and 4 women (28%) had neither condition.

In all 14 cases of women with a complete unilateral levator defect, the damaged portion of the muscle was identified as part of the pubovisceral muscle. None had concurrent damage noted in the iliococcygeal or puborectal portions of the levator.

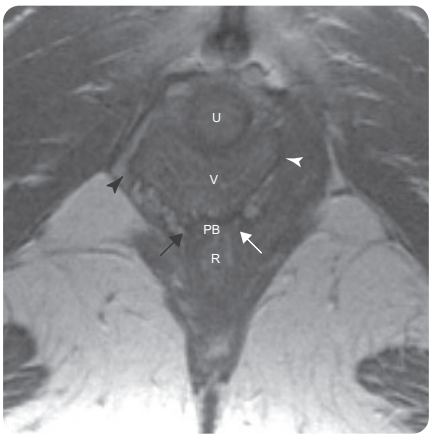
The overall direction of the missing muscle is diagonal from anterosuperior to posteroinferior (Figure 2). In all cases, the missing muscle originates from the posterior aspect of the pubic bone near the midline and extends laterally toward the iliococcygeal muscle. The fibers missing are those that arise from the pubis lateral to the pubic symphysis and over the anteromedial aspect of the obturator internus muscle (the arcus tendineus levator ani). This feature is best seen in Figure 2, **C** and **D**. The most anterior portion of the missing muscle is lateral to the space of Retzius and medial to the obturator internus muscle. The midportion of the muscle is lateral to the vagina, with attachment to the vaginal wall (Figure 3). The most posterior part of the muscle is lateral to the internal anal

FIGURE 2
Three-dimensional model of the pelvis



Three-dimensional model generated from the axial MR scans shown in Figure 1. **A,** and **B,** Oblique right and left inferolateral views, similar to the dorsal lithotomy position, are shown. In these panels the pubic bone is semitransparent and the obturator internus muscle is not shown. **C,** and **D,** Oblique right and left views peering over the pubic bone and down to the pelvic floor are shown. The urethra, vagina, and rectum have been truncated so as not to obscure the views of the levator muscles. *EAS*, external anal sphincter; *LA*, levator ani; *MM*, mirror image of the missing muscle; *P*, pubis; *PB*, perineal body; *U*, urethra; *V*, vagina. The missing muscle in **A** and **D** is denoted (*asterisk*). Printed with permission from John O. L. DeLancey, MD.

FIGURE 3
Axial MRI showing loss of attachment



Axial MR scan of a 46-year-old multiparous woman is shown. The intact attachment between the left pubovisceral muscle and the vaginal wall is indicated (white arrowhead), the loss of this attachment is shown on the right side (missing muscle; black arrowhead). The insertion of the intact pubovisceral muscle into the perineal body (PB) is shown (white arrow); the loss of this attachment on the contralateral side is also shown (black arrow). *R*, rectum; *U*, urethra; *V*, vagina. Printed with permission from John O. L. DeLancey, MD.

sphincter and rectum and medial to the ischiorectal fat.

The muscle inserts into the lateral margins of the perineal body (Figure 3). It also inserts into the intersphincteric space between the anal sphincters.

At a more cephalad level, fibers also pass into the space between the puborectal muscle and rectum crossing the midline in this location. Overall architectural distortion, with an asymmetric lateral spilling of the vagina toward the obturator internus muscle, filling the space left by the missing muscle, was present in 50% of women. The muscle defect was right sided in 71% of patients. There is considerable variation in the morphology of the muscles and their attachments (Figure 4).

COMMENT

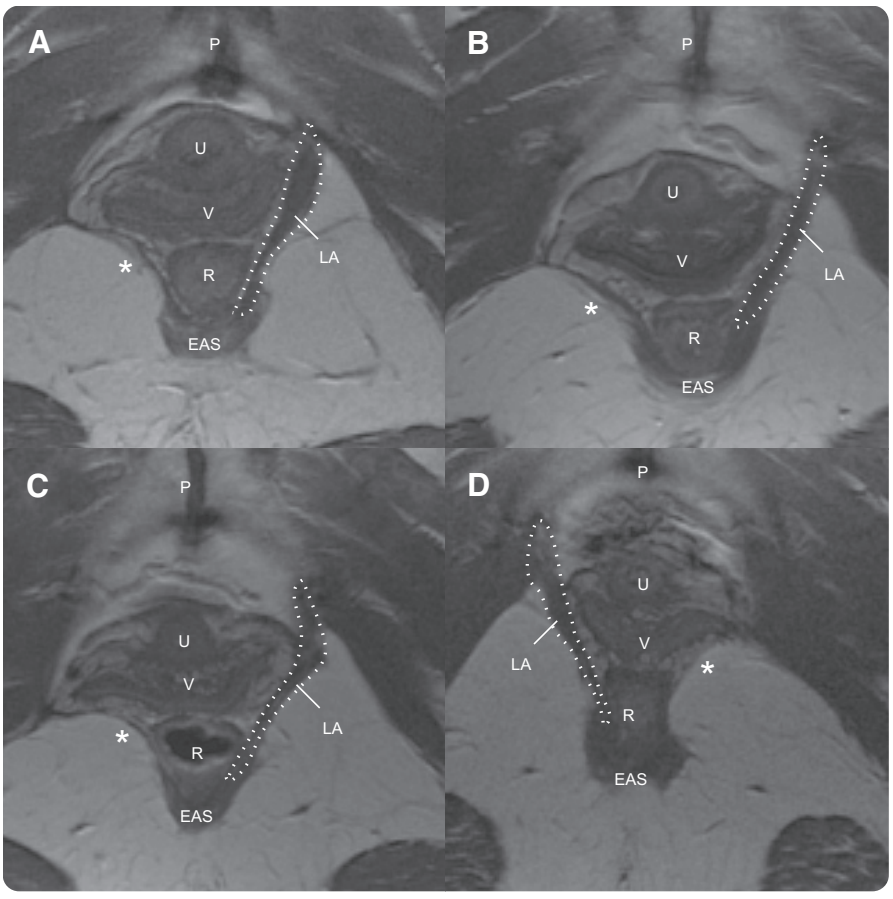
In this study we identified the origin (pubic bone) and insertion (vaginal wall, perineal body, and intersphincteric space) of the missing portion of the levator ani muscle in women with unilateral levator defects involving the pubovisceral portion of the levator. Pubovisceral defects are more commonly seen than iliococcygeal defects. In an earlier study of defects following a woman's first vaginal birth, pubovisceral defects were seen in 18% of 160 primiparous women, and iliococcygeal muscle defects were seen in only 2% of women.[5] In this group of women selected to have unilateral defects in the levator ani muscle, we found defects only in the pubovisceral portion of the muscle, with no concurrent defects of the puborectal or iliococcygeal portions. The pubovisceral muscle is complex, containing attachments to the vagina (pubovaginal), perineal body (puboperineal), and the intersphincteric space (puboanal).

Four attachments were affected by the missing muscle in these women, including the muscular origin at the posterior pubic bone and the insertions into the lateral vaginal wall, the perineal body, and the intersphincteric space.

This study expands on the work of other investigators who have described the general location of the damaged portion of the levator ani. Hoyte et al[2] has used 3D models created from MR images to study the morphology and volume of the levator muscles in women with and without pelvic floor dysfunction. Using MRI, DeLancey et al[5] identified levator defects in 20% of a primiparous population. Dietz and Lanzarone[6] used 3D translabial ultrasound to demonstrate avulsion of the inferomedial aspect of the levator ani muscles in postpartum women. For the first time, we have demonstrated the specific anatomic connections affected by damage to the levator.

We chose to analyze only women with a unilateral levator ani defect be-

FIGURE 4
Four examples of unilater levator defects



Axial MR scans of 4 different women with complete unilateral levator defects are shown. Note the variations in morphology. The intact levator ani muscle is traced (*dashed line*) and labeled LA. The missing muscle is denoted (*asterisk*). **A, B,** and **C,** the defect is shown on the right side and **D,** on the left side. *EAS*, external anal sphincter; *P*, pubis; *R*, rectum; *U*, urethra; *V*, vagina. Printed with permission from John O. L. DeLancey, MD.

cause of the unique opportunity to study normal and abnormal muscles in the same individual. Unilateral defects themselves are not associated with any particular clinical syndrome. However, by allowing us to study the specific anatomic connections affected by damaged levator ani muscles, which have been associated with pelvic floor dysfunction,[2] this subset of women can help to elucidate the anatomic abnormalities that may be associated with pelvic organ prolapse.

Clinicians have noticed the gaping introitus and enlarged hiatus present in women with prolapse[7] for many years. The origins and insertions of the missing muscle (pubis and perineal body, vagina, and anal sphincter) are directly relevant to holding the perineal body, vagina, and anus closer to the pubic bones. Loss of this muscle portion may help explain why women with prolapse, known to have a higher incidence of levator ani muscle loss,[2] have a larger genital hiatus.[8]

Why is this specific portion of the muscle most affected? In a computer simulation of vaginal birth, the medial portion of the pubovisceral muscle undergoes greater stretch than any other part of the levator ani muscle.[9] Our findings of missing muscle only in the pubovisceral portion of the levator are consistent with this model. The possible connection between muscle damage and stretch at the time of vaginal birth raises the question of mechanism of injury. Whether this stretching of the muscle leads to avulsion, with the muscle tearing away from its points of attachment, or neurologic injury caused by compression or ischemic damage to the muscle and/or nerves created by direct compression of the fetal head remains undetermined.

This study provides important information about the muscle portion affected in levator ani muscle defects. However, this type of investigation has some limitations. It is not always possible to see the direction of individual muscle fascicles on MR scans. 3D model reconstruction does, however, enable us to study the morphology and the anatomic connections affected by levator damage. Using our knowledge of muscle fiber direction gleaned from the literature, anatomical dissections, and MRI studies,[3,10,11] we are able to estimate fiber direction and thereby hypothesize about the lines of action of the different parts of the levator ani muscle.

Future research will focus on the development of biomechanical models to analyze these force vectors to better understand the specific functional deficits created by damage to a particular portion of the muscle.

Rapid advances in our understanding of the precise defects present in women after vaginal birth are adding to the body of scientific knowledge about pelvic floor dysfunction. A clearer understanding of the disease mechanisms of pelvic floor dysfunction will ultimately lead toward rational, evidence-based intervention, prevention, and treatment for these common and devastating conditions. ■

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