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Levator Ani Abnormality Six Weeks After Delivery Persists At Six Months

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Abstract

Objective: Assess post partum changes in the levator ani muscle using magnetic resonance (MR) imaging and relate these changes to obstetrical events and risk factors associated with pelvic floor dysfunction.

Study Design: A board certified radiologist specializing in abdominal imaging evaluated 146 pelvic MR studies from 57 primiparas 6 weeks and 6 months after first obstetrical delivery and 32 nulliparas. A yes/no determination of muscle body and insertion integrity, muscle thinning, and measurement of muscle thickness in mm was made for each of four muscle sites: right and left puborectalis, right and left ileococcygeous.

Incidence of muscle abnormality and mean muscle thickness were tested in pairs between 1) nulliparas and 6 week primiparas, 2) 6 week and 6 month primipara pairs, and 3) three age/race groups using test of two proportions and one-way anova.

Results: Initial review indicated only 3 subjects not of African-American (AA) or Caucasian (W) race, and only 1 AA primipara of age ≥30yrs, therefore statistical analysis was limited to 45 primiparas and 25 nulliparas. Incidence of any abnormality at any of the 4 sites was considered abnormal. Frequency of abnormal muscle by race and age is shown in the table.

		Caucasian, <30yrs	Caucasian, ≥30yrs	African-American, <30yrs
paras				
	Normal	9 (82%)	6 (100%)	7 (88%)
	Injured	2 (18%)	0 (0%)	1 (12%)
paras				
	Normal	7 (44%)	12 (52%)	4 (66%)
	Injury; persistent	2 (12%)	5 (22%)	1 (17%)
	Injury; recovered	7 (44%)	6 (26%)	1 (17%)

In those subjects recovering to normal MR by 6 months, an average of nearly 60% increase in R puborectalis muscle thickness compared to that seen at 6 weeks indicated the extent of the injury. Subjects with injury to both the puborectalis and ileococcygeous at 6 weeks did not recover to normal at 6 months, whereas those with injury only to the puborectalis tended to have normal MR at 6 months.

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Conclusions: Nulliparity did not guarantee a normal assessment of levator ani anatomy by our blinded reader, and frequency of injury in this series is somewhat greater than that previously reported for primiparas. Younger Caucasian primiparas had a better recovery at 6 months than older Caucasians. Subjects experiencing more global injury, in particular to the ileococcygeous, tended not to recover muscle bulk.

Keywords

Levator ani injury; Pelvic floor dysfunction; Magnetic Resonance Imaging

Introduction

Pelvic floor dysfunction is a major women's health problem with significant negative economic and quality of life impact.[1] While the etiology of urinary and fecal incontinence and pelvic organ prolapse is almost certainly multifactorial, vaginal parity is the most consistently associated risk factor. [2,3] The marvelous complexity of obstetrical labor and delivery has made it difficult to determine the exact nature of the injury. Various investigators have identified connective tissue disruption, muscle fiber hypoxia, nerve compression injury, among others.[4,5] Direct damage to pelvic floor musculature has been suggested by computer models of parturition, which simulate the fetal head causing stretching of the levator ani complex to potentially injurious lengths. This stretching could result in a tearing of the muscle body, detachment from its bony insertions, and neuropathic injury. [6]

Pelvic magnetic resonance imaging (MRI) has emerged as a noninvasive means of evaluating muscle morphology, both normal and pathologic. Abnormal muscle can be identified by a change in the thickness or topography of the muscle. However, normal pelvic floor morphology can vary significantly in women of different race [7] and age [8]. The present study evaluated changes in levator ani abnormalities on pelvic MRI 6 weeks and 6 months after a first obstetrical delivery and correlated these findings with demographic data and mode of delivery.

Material and Methods

Magnetic resonance images of 32 nulliparas and 57 primiparas were obtained as part of an institutional review board-approved prospective cohort study of pelvic floor injury during first obstetrical delivery. Demographic data and mode of delivery of the groups are shown in Table I. Exclusion criteria included a history of pelvic surgery, pelvic floor symptoms, known diabetes or neuromuscular disorder, or operative or cesarean delivery planned at enrollment. Primiparous subjects were recruited while still pregnant, and ultimately underwent pelvic MRI 6 weeks and 6 months after term delivery, while the nulliparous subjects underwent a single study upon enrollment. MRI of the pelvis was performed on a 1.5 Tesla MRI scanner (1.5T, Signa, GE medical systems, Milwaukee). Sequences used were as follows: axial and coronal fast spin echo, FSE (TR 3000, TE 104, 2/0 thickness, FOV 16cm, matrix 256 x 160, sagittal single shot fast spin echo, SSFSE (TR max, TE 100, 90 degree flip, 5/5 thickness, FOV 20 cm, matrix 256 x 256, NEX 0.5). Subjects were positioned comfortably supine, with knees supported by a standard small triangular pad, and a scout film confirmed that the subject was centered appropriately in the scanner.

Labor and delivery in the primiparous subjects was managed utilizing principals of active management of labor. [9] We recorded time of labor benchmarks, minutes spent pushing, and mode of delivery. We defined four modes of delivery: spontaneous vaginal delivery (SVD), operative vaginal delivery (OVD) involving vaginal delivery after placement of forceps and/or vacuum, cesarean in labor as cesarean performed after the onset of labor, and elective cesarean (EC) prior to the onset of labor.

The 32 nulliparas underwent screening pelvic organ prolapse quantitation (POPQ) exam[10] at enrollment, and the 57 primiparas underwent the exam at each visit: antepartum, 6 weeks post partum, and 6 months post partum. Each subject also completed the pelvic function quality of life questionnaire (long form) at each visit. [11]

A board-certified radiologist specializing in abdominal imaging (JT) who was blinded to demographic information and parity, evaluated all of the digital MR images individually, in random order, using the Efilm Lite software program. A minority of studies (n=29) were read on standard film as digital images were not available. Assessment of the left and right puborectalis muscles were made in the axial plane over 2-3 adjacent images, just caudal to the lowest portion of the urinary bladder. (Figure 1) Assessment of the left and right ileococcygeus muscles were made in the coronal plane, at the level that contained the most complete coronal section of the anal canal. (Figure 2) A yes or no determination of muscle injury was made for each site, as well as a measurement of the thickest part of the midpoint of the muscle. Criteria for injury included muscle belly thinning, disruption of the continuity of the muscle, and images consistent with detachment of the muscle from its insertion. An additional criterion for injury to the puborectalis muscle was the presence of protrusion of the vagina into the paravaginal space, implying disruption of the normal paravaginal architecture. (Figures 3 and 4)

Incidence, type, and location of muscle injury were identified in all the subjects. Primiparous subjects were categorized into three groups according to observed patterns of injury and recovery: normal on both MRI scans, injury at both 6 weeks and 6 months (persistent injury), and injury at 6 weeks but recovered at 6 months (recovered injury). Subjects were further grouped by both age and race, creating four groups: 27 Caucasians aged <30 yrs, 29 Caucasians ≥30 yrs, 14 African-Americans <30 yrs, and 8 African-Americans ≥30 yrs. There was only 1 primiparous subject in the last group, therefore these 8 subjects were excluded from those comparisons. Injury rates in the three race-age groups were compared using a two-way Chi-square test. In the primiparas, patterns of injury and recovery and association of injury with mode of delivery were compared using a two-way Chi-square test. For each subject who had a levator muscle injury, a checklist of location and whether the injury was due to thinning or loss of muscle insertion for each of the four muscle sites was compiled. A linear model for right puborectalis muscle thickness with two sources of variation, three injury groups and three age-race groups was computed. Estimated marginal means with 95% confidence intervals for all injury × age-race groups were estimated.

Results

Table I indicates the initial subject demographics including all four of the age/race groups. We were forced to exclude 11 more primiparas because of inadequate quality or incomplete MRI studies precluding full assessment. The remaining 25 nulliparas and 45 primiparas were the subjects of the full analysis. Table II compares injury rates in these subjects by the three age/race groups. Nulliparity did not guarantee a normal assessment of levator ani anatomy, though all nulliparas had Stage 0 prolapse on POPQ exam. Rates of injury were similar across the age/race groups, with a trend toward increased persistence of injury at 6 months post partum in Caucasians ≥30yrs. Most primiparas had Stage I POPQ support at 6 months post partum, with a minority meeting criteria for Stage II. Post partum PFQOL scores were not clinically significantly increased. We were unable to establish a statistical relationship between POPQ measurements, QOL scores, and MR findings (data not presented).

In Table III, the type and location of injury detected at 6 weeks postpartum in the 23 injured primiparas are graphically displayed. Each row represents a single subject. The muscle site and type (thinning vs. loss of integrity or insertion) are indicated for each abnormality identified. The top portion of the table shows the subjects with persistent MR abnormalities at

6 months postpartum, while the bottom portion are those subjects who recovered, and had a normal MR read at 6 months. As the table demonstrates, those subjects with persistent abnormal pelvic MR at 6 months post partum tended to have more global muscle damage, with both thinning and loss of integrity or insertion noted in the same muscle and more frequent involvement of the ileococcygeus muscle. Notably, subjects with recovery of normal muscle at 6 months had more focal puborectalis injury. However, we observed an average 59% increase in muscle thickness on the right and 58% increase in thickness on the left puborectalis in these subjects at 6 months when compared to the 6 weeks post partum MR, suggesting a fairly extensive local injury with subsequent recovery of muscle bulk.

Table IV compares injury rates to mode of delivery. Although the numbers are small, it is apparent that in our study elective cesarean did not protect against the identification of MR abnormalities by our blinded radiologist, with 2 of 4 subjects demonstrating muscle injury at both postpartum visits. Although there was no statistical significance, there was a trend toward less injury in subjects having SVD and a second stage of labor <30 minutes (p=0.09).

Table V compares muscle thickness of the right puborectalis, the most common injury site, for normal nulliparas, normal primiparas (those with no abnormality called by the reader), and primiparas with injury (those with focal injury on MR called by the reader). These groups are further subdivided into groups by age and race. Younger Caucasian primiparas with levator injury on MR had significantly thinner puborectalis muscle than primiparas with normal MR, as evident by confidence interval. Primiparas with injury tended to have thinner puborectalis muscles, whereas those without injury were not generally different than that of the nulliparas. It is important to note, however, that the “normal” Caucasian primiparas ≥30 yrs had mean muscle thickness that does not appear different from subjects in that age/race group who were called “injured” on MR. This could only have occurred if the reader determined that the muscle appeared symmetrical and intact bilaterally, but then made an actual measure of muscle thickness in mm that ultimately produced a low average thickness for the group.

Comment

This study used a systematic approach to evaluation of the levator ani that incorporated methods previously reported for the puborectalis [12,13] in order to more globally assess the entire levator complex. Important unique elements of our dataset were that it included both obstetrical information and serial images in subjects during the post partum period, allowing us to observe changes in the muscle over time. We used this approach to monitor any recovery from acute injury during the postpartum period. DeLancey and others have shown that acute or sub acute muscle disruption can be identified on MRI by muscle thinning and loss of either muscle integrity or normal attachments, and have theorized that these changes are consistent with muscle avulsion. [12] This study does not definitively disprove or prove any specific mechanism of injury to pelvic floor muscles during delivery. However, we hypothesize that our observed recovery of muscle thickness in a substantial portion of subjects with initial injury at 6 weeks is more consistent with a neuropathic mechanism of injury and subsequent recovery of muscle bulk, rather than the more permanent muscular tear or avulsion. Our finding that ileococcygeal injury on MR is almost always associated with failure to recover from the injury is also consistent with a neuropathic mechanism, if it means that reinnervation and recovery is unlikely once a certain threshold of severity of the injury is reached. One explanation for our findings of abnormal muscle insertion/integrity in subjects who apparently recovered from their initial injury is that the muscle was so thinned that insufficient bulk was visible at the insertion point for our reader to definitively say that the insertion was intact. Ongoing studies testing the reliability of multiple readers as well as functional electromyographic studies of these subjects may help clarify these issues.

While the size of our dataset was limited, our data suggest that Caucasians may be more likely to suffer a muscular injury after delivery, and that older subjects may be less likely to recover from this injury. Both these findings are consistent with larger trends of prevalence of pelvic floor dysfunction in the female population. [1] While vaginal parity is clearly a risk factor for pelvic floor dysfunction, we were unable to demonstrate clear relationships of frequency of injury on MR to obstetrical events.

In a recent series of primiparas examined with pelvic MRI 9-12 months after delivery, 20% had a visible levator defect, the majority (18%) involved the puborectalis portion of the levator. [12] The 6 week post partum injury rates identified in our study were higher than those seen in DeLancey's series, but our 6 month post partum rates of injury were similar, suggesting concordance. It is possible that our more acute assessments at 6 weeks post partum allowed initial detection of injuries that that recovered to some degree by 6-12 months post partum.

We did have the unexpected finding of a low, but present, rate of identified injury in the nulliparous group. This finding raises concerns about the accuracy of identifying levator ani injury on MRI, or the assumption that nulliparas are free of pelvic floor damage, or both. This finding highlights one methodologic difficulty with this study, which is that determination of “normal” or “abnormal” in a left and right assessment of and image inevitably presents the difficulty of being forced to compare sides without an assurance of which, if any, side is actually normal. We intend to assess this in further analysis, with the hypothesis that normal variations in the thickness of these muscles contributes to difficulty of assessment, particularly when there is not a baseline image available for comparison. Three dimensional reconstruction of MR data such as that in this study also holds promise for elucidating and confirming observations made in two dimensions. We also intend to pursue confirmation of the validity of assessment of pelvic floor MR by a single experienced reader, which has received only limited attention in published literature. [14]

Our understanding of the relationship between vaginal birth and pelvic floor damage is currently incomplete, and this study demonstrates some of the difficulties inherent in imaging this anatomy. However, if pelvic MR proves to be a suitable diagnostic technique for identifyingv pelvic floor injury, it may aid in understanding the mechanisms of injury and thereby allow us to target preventative and reparative strategies to those at risk.

Acknowledgements

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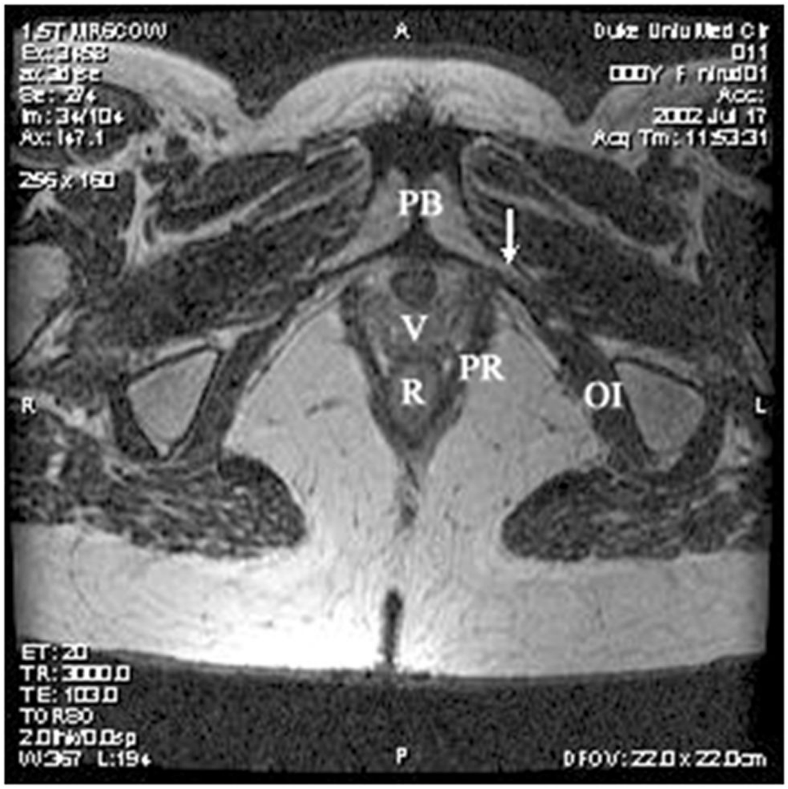

Figure 1.

Axial image of the pelvis showing the puborectalis portion of the levator ani muscle in a normal subject, This image is typical of the anatomic level at which the muscle was assessed in this protocol. The broad, symmetric insertion of the muscle into the pubic rami is apparent, as is the uniform thickness of the muscle and its symmetry. (PB=pubic bone, white arrow= pubic ramus, OI= obturator internus m., PR=puborectalis m., V=vagina, R=rectum)

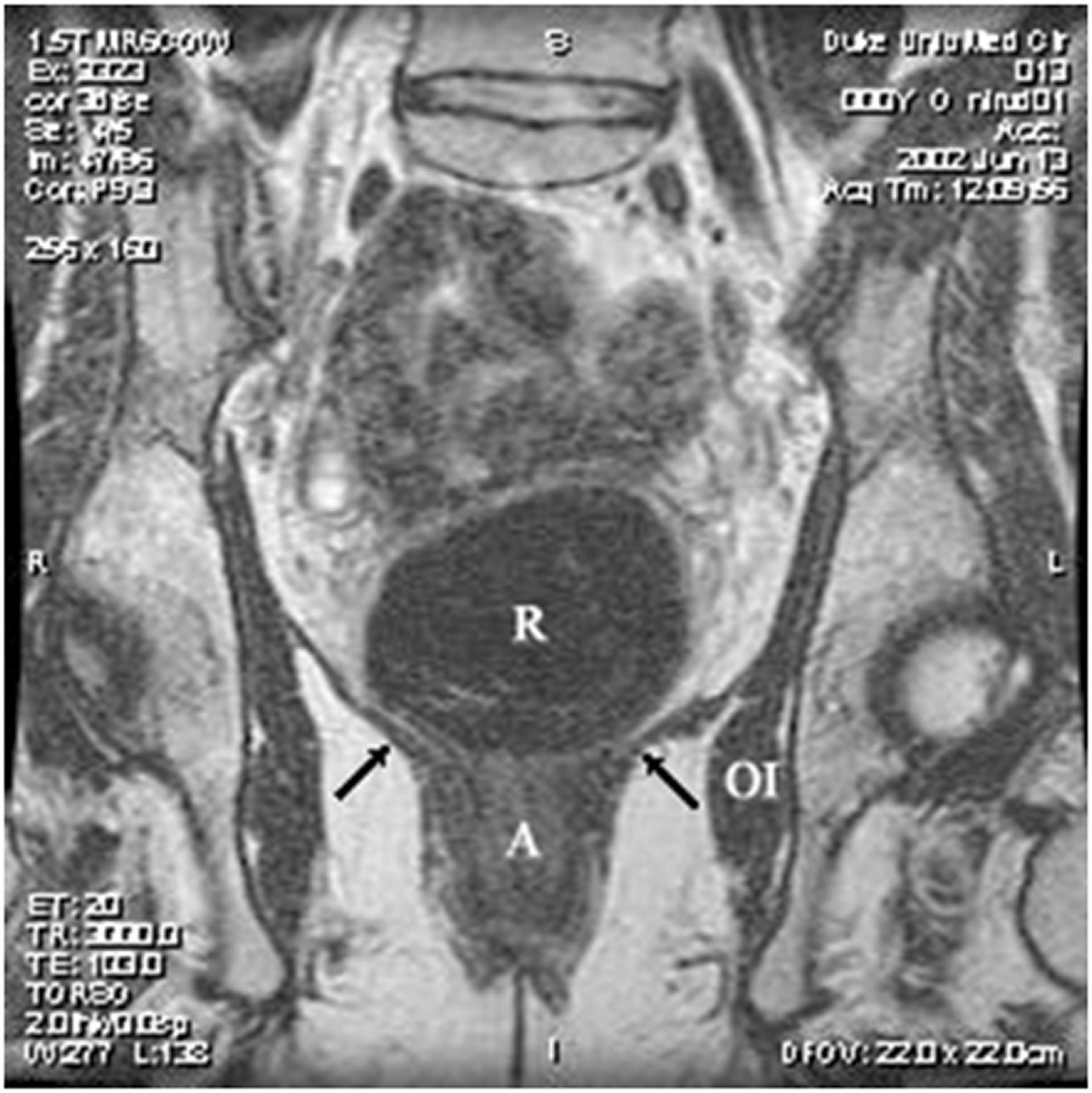

Figure 2.

Coronal image of a nulliparous pelvis showing the ileococcygeous muscle, typical of the anatomic level assessed in this protocol. Note that the entire anal canal (A) passes in the coronal plane. (R=rectum, black arrows indicate the ileococcygeous m., OI=obturator internus m.)

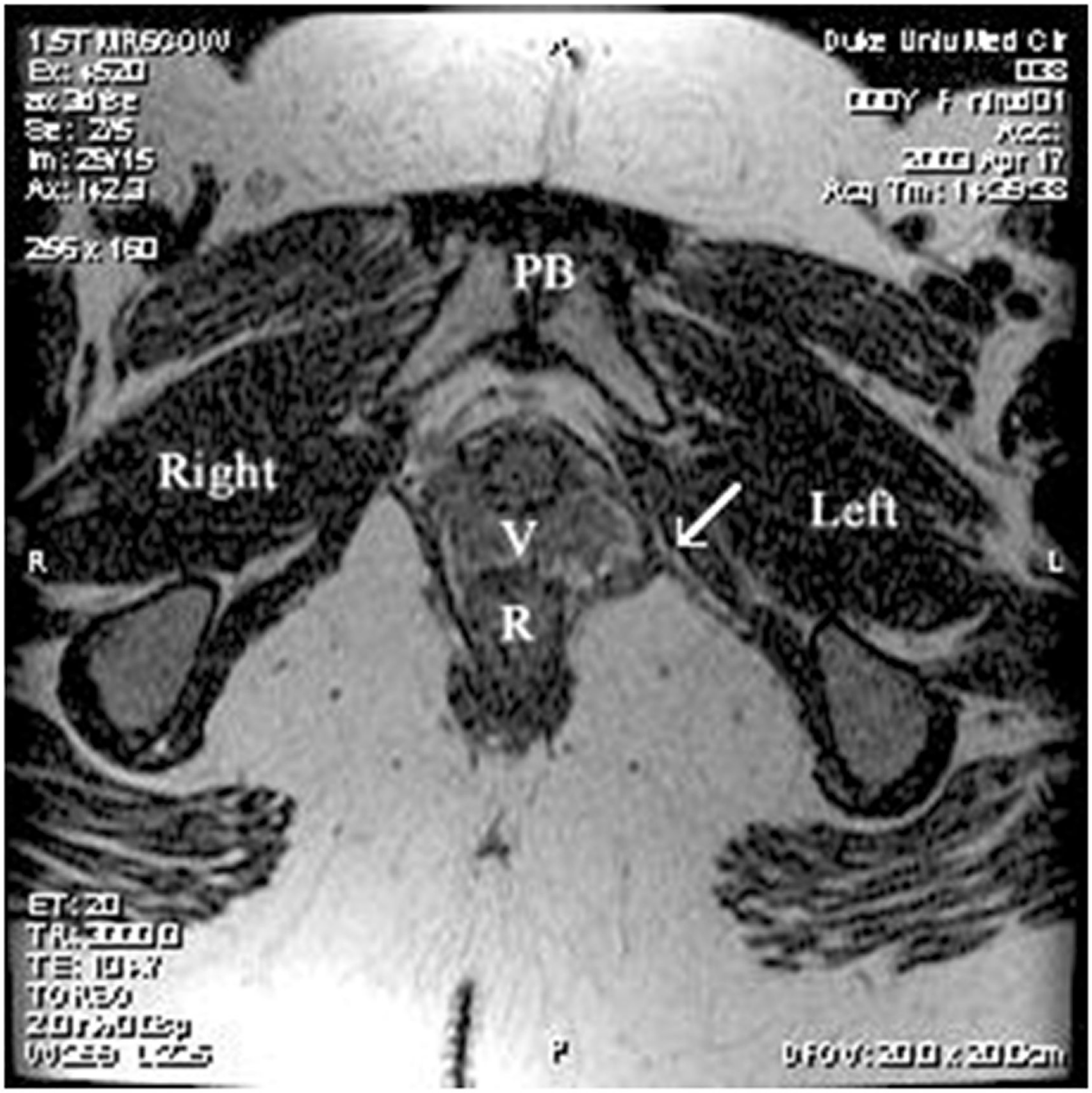

Figure 3.

Axial image demonstrating left puborectalis muscle abnormality noted by white arrow. The insertion into the pubic ramus is not intact. Note the protrusion of the vagina into the paravaginal space. (V=vagina, PB=public bone, R=rectum)

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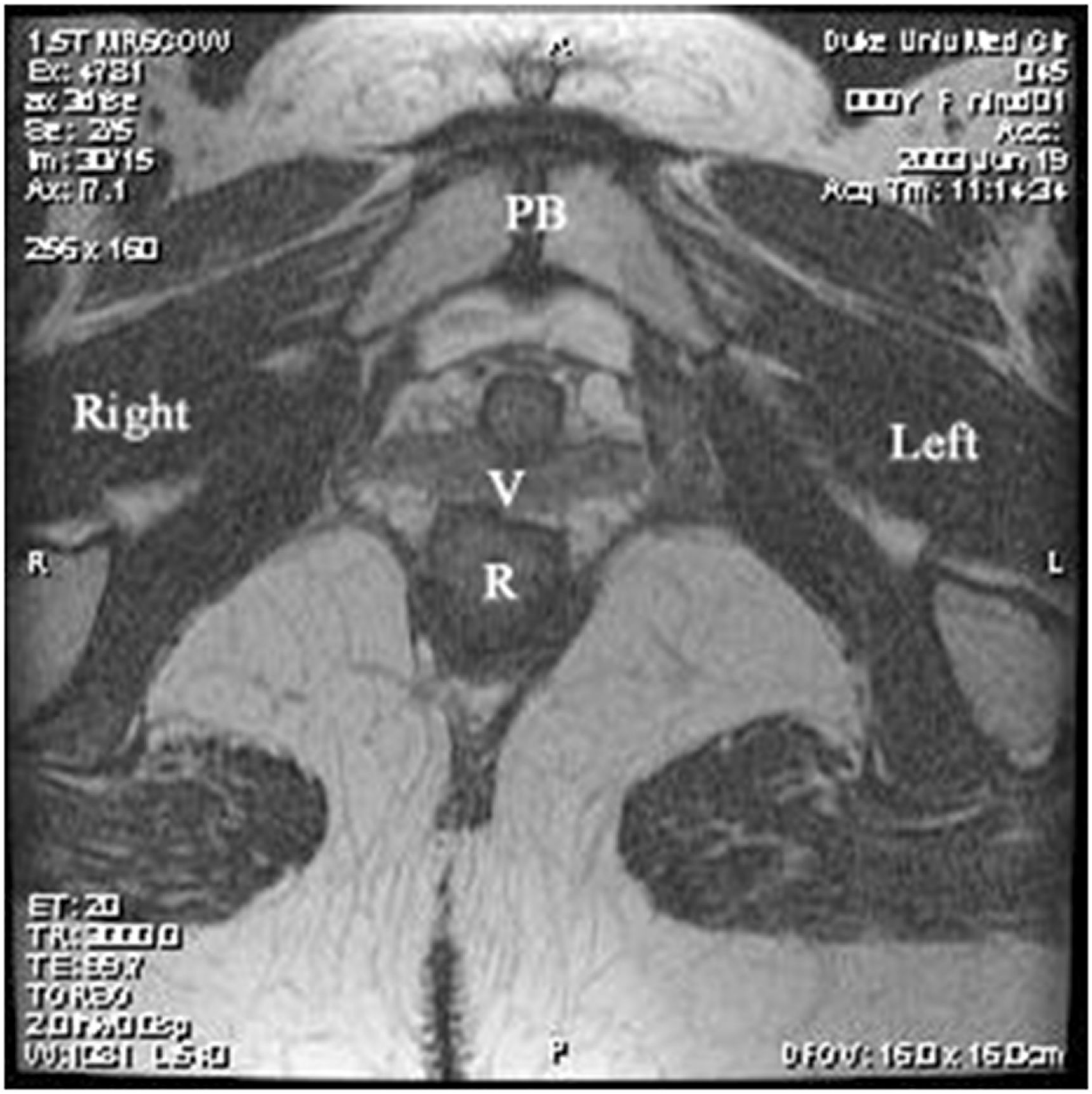

Figure 4.

Axial image demonstrating bilateral puborectalis muscle abnormality. Muscle thinning and loss of insertion is noted. (V=vagina, PB=public bone, R=rectum)

Table I

Subject demographic data and mode of delivery. (SVD=spontaneous vaginal delivery, OVD=operative vaginal delivery via forceps or vacuum, C/S in labor= cesarean performed after the onset of labor, Elective C/S=cesarean performed prior to labor)

Group	N	Age in yrs	Race		Mode of Delivery	
Nulliparas	32	Range: 19-48 18-25: 12 26-34: 14 ≥ 35: 6	Caucasian African-American	18 (56%) 14 (44%)	N/A	
Primiparas	57	Range: 18-39 18-25: 9 26-34: 39 ≥35: 9	Caucasian African-American	47 (82%) 10 (18%)	SVD OVD C/S in labor Elective C/S	34 (60%) 8 (14%) 11 (19%) 4 (7%)

Table II

Comparison of incidence of levator injury for subjects, by age and race.

	Caucasian, <30yrs	Caucasian, ≥30yrs	African-American, <30yrs
Nulliparas			
Normal	9 (82%)	6 (100%)	7 (88%)
Injured	2 (18%)	0 (0%)	1 (12%)
Primiparas			
Normal	7 (44%)	12 (52%)	4 (66%)
Injury; persistent	2 (12%)	5 (22%)	1 (17%)
Injury; recovered	7 (44%)	6 (26%)	1 (17%)

Table III

Type and location of injury identified at 6 weeks post partum in the 22 injured primiparas. This table shows raw data for all primiparous subjects and demonstrates the association of ileococcygeus injury with persistent levator injury.

	R PR Thickness	R PR Insertion/ Integrity	L PR Thickness	L PR Insertion/ Integrity	R IL Thickness	R IL Insertion/ Integrity	L IL Thickness	L IL Insertion/ Integrity
Persistent Injury	•	•	•	•		•		•
	•		•		•		•	
	•	•	•	•			•	
					•	•	•	
	•	•						
	•	•	•			•		
					•	•	•	•
		•						
Recovered From Injury					•			
	•		•					
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	•		•					
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	•		•					

(R PR= right puborectalis, L PR= left puborectalis, R IL=right ileococcygeus, L IL=left ileococcygeus)

Table IV

Relationship of mode of delivery to frequency and persistence of levator injury.

Delivery Type	N	No injury	Injured-persistent at 6 months	Injured-recovered by 6 months
Vaginal; push <30 min	6	4 (67%)	0 (0%)	2 (33%)
Vaginal; push ≥ 30min	29	15 (52%)	4 (14%)	10 (35%)
C/S in labor	6	2 (33%)	2 (33%)	2 (33%)
Elective C/S	4	2 (50%)	2 (50%)	0 (0%)

Table V

Muscle thickness (mean and 95% confidence intervals in mm) for right puborectalis for normal nulliparas, normal primiparas, and primiparas with injury and by age and race. Any muscle abnormality noted on MRI is characterized as injury.

		White <30 yrs			White ≥30yrs			Black <30 yrs	
	n	**Mean**	**95% CI**	**n**	**Mean**	**95% CI**	**n**	**Mean**	**95% CI**
Normal Nulliparas	9	3.4	(2.4, 4.5)	6	5.5	(4.2, 6.8)	7	4.3	(3.1, 5.5)
Normal Primiparas	7	3.0	(1.8, 4.2)	12	2.6*	(1.7, 3.5)	4	4.0	(2.4, 5.6)
Primiparas with Injury	9	2.3	(1.3, 3.4)	11	2.0*	(1.1, 3.0)	2	2.0	(−0.3, 4.3)

* Significant difference at α=.05 from normal nulliparas for white ≥30yrs.