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New site-specific anatomical defects discovered within the female urogenital tract

Adam Ostrzenski

7001 Central Ave., St. Petersburg, FL, 33710, Institute of Gynecology, Inc., United States



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ABSTRACT

Objectives: Testing whether the site-specific anatomical defect(s) can occur within the urethral stabilizing mechanism; determining the anatomical structure compositions of the urethral stabilizing mechanism. *Study design:* In an international setting, fifteen human fresh female cadavers subjected to urogenital tract stratum-by-stratum macro- and micro-dissection and a case series study type utilized in this scientific research. The primary outcome measured occurrences of anatomical site-specific defects within the urethral stabilizing mechanism and the secondary outcome measured the anatomical structures of the urethral stabilizing mechanism.

Results: Site-specific defects occurred within the urethral stabilizing mechanism in four out of fifteen subjects (26.6%); defects most commonly identified within suburethral-paraurethral-periurethral areas. The ventral perineal membrane and distal condensations of the arcus tendineus levatoris ani defects were also present. The urethral stabilizing mechanism consists of multiple anatomical structures located in different anatomical regions.

Conclusions: Site-specific defects occur within the urethral stabilizing mechanism in women. The urethral stabilizing mechanism consists of multiple anatomical structures located in different anatomical regions, and they fuse with the urethral wall.

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Introduction

Scientific-clinical researchers did not report site-specific anatomical defects within the urethral stabilizing mechanism (USM) structures in women; although, they reported in fragments the urethral supporting and suspending structures. In 1951, Krantz described the gross anatomy and histology of suburethral support structures, and DeLancey verified this anatomical findings [1,2]. Krantz also established the presence of natural fusion between the bulbocavernosus muscle and the ischiocavernosus muscles and they cover the urethral wall partially [1]. Jeffcoat determined that the distal one-third of the urethra was immobile in her fluoroscopic study [3].

In 1983, Oelrich discovered the existence of the perineal membrane, which was confirmed by Stein and Delancey [4,5]. Kaufman and Faro documented the presence of posterior urethral imprint on the anterior-distal vaginal wall [6]. Intraoperatively, Ingelman-Sundberg demonstrated the presence of lateral vaginourethral ligaments, which connect the anterior-distal-lateral vaginal wall with the lateral urethral wall. These ligaments insert

nearby the urethral meatus. [7] The current author introduced the term of lateral vaginourethral ligaments (Terminologia Anatomica does not include this nomenclature [8]). El-Sayed et al. confirmed the existence of these anatomical structures by MRI study [9]. DeLancey determined that paraurethral anatomy consists of the paraurethral ligaments as well as the connective tissues, which play the role in connecting the urethral wall and the vaginal wall to the pubococcygeus muscle (the vaginolevator attachment) [10]. Tan et al. described the periurethral and paraurethral ligaments being located anteriorly to the vesicourethral junction and they traverse in the Retzius space [11]. These ligaments bilaterally extend to the lateral-medial aspect of the puborectalis muscle [12]. Researchers confirmed this finding was confirmed [9]. Langrader described the pubourethral ligaments (PULs) as suspensory urethral anatomical structures [13]. Other researchers confirmed these findings [13–17]. The existence of pubourethral ligaments has been debated for decades and resent scientific data presented by Pit et al. determined that the pubourethral ligaments are the distal condensations of the arcus tendineus fasciae pelvis (ATFP), which blends with the urethral wall via the endopelvic fascia [18].

The purpose of this study was to test potential occurrences of the site-specific anatomical defect(s) within the urethral stabilizing mechanism and to determine the USM anatomical

E-mail address: ao@baymedical.com (A. Ostrzenski).

compositions. The rationale for the study was to establish the potential occurrence of anatomical site-specific defect(s) within USM as a platform for a future clinical-scientific study to determine whether such a defect(s) can cause stress urinary incontinence (SUI) in women as well as to develop surgical therapy to reconstruct these anatomical defects.

Materials and methods

Study design

The author himself performed stratum-by-stratum dissections of the female urogenital tract in the deigned consecutive case series study. The local university Ethics Committee approved the study protocol (AKBE 146/12). The subjects in the dorsal supine position, knees maximally flexed, widely separated, and stabilized created the access to the female urogenital tract. The author used a magnifying surgical loop with 3.5–4 X during dissections in order to avoid causing anatomical defects and circumvent blunt dissections.

Study participants

The study included fifteen human fresh female multipara cadavers, and at the death, the age of subject ranged between 27-year-old and 88-year-old with the mean age of 57.5 ± 31 .

Exclusion and inclusion criteria

The medical record of any subject at the Department of Forensic Medicine was not available due to the type of medical services that the department provides. The implicit assumption of multiparity was made based on the uterine cervix characteristic appearance for multiparity, the vaginal introitus, and episiorrhaphy scars. Additionally, a notion that labor and delivery can cause potential site-specific defects within the urogenital tract, in some women.

Those subjects excluded, who presented with urogenital tract anatomical deformities or evidence of prior injury to the urogenital area, or evidence of prior surgery. Additionally, the subjects excluded who presented with a urogenital tract tumor, enlarged inguinal lymph nodes, and homicide associated with rapes or incest.

Anatomical dissection

Upon entering the Retzius space, the dissection was initiated by dividing the transversalis fascia. This maneuver provided access and exposure to the bladder neck, the proximal urethra, the distal condensations of the arcus tendineus fasciae pelvis (ATFP), retropubic peri- and para-urethral tissues, the arcus tendineus levator ani (ATLA), paravaginal and paraurethral sulci. Visualization of the bladder neck, the proximal urethra, and their suspension-support tissues improved by filling the urinary bladder cavity with 350 ml of water. At the end of these observations, emptying the urinary bladder cavity restores the bladder resting appearance. Introducing a metallic probe into the urethral lumen helps to move urethra upwards, downwards and laterally.

Making a transverse skin incision, 0.5 cm above the anterior external urethral meatus exposed the prepubic area. The natural fusion between the endopelvic fascia and the ventral perineal membrane was dissected using a surgical loop with 3.5–4.0-time magnifications. Upon identification of the ventral perineal membrane, the organs resting on this structure were sharply dissected and retracted. Identifying the ventral perineal membrane and its attachments to the ischiopubic rami, the pubic arch,

and the symphysis pubis helps to establish a potential site-specific defect(s).

To get access to peri- and para-urethral areas, a skin incision was made approximately 0.5 cm laterally from the distal urethral wall edge. A transverse incision was made on the anterior-distal vaginal wall approximately 1.0 cm below the posterior urethral meatus in order to access the suburethral area and the lateral vaginourethral ligaments.

Literature search

Both, a manual and an electronic literature searches have been carried out for scientific-clinical articles with regards to the urethral anatomy, urethral stabilizing mechanism, and the occurrence of site-specific defects within this mechanism. The searches were conducted from 1800 to September 2018 using Medical Subject Headings (MeSH), which were selected and used in a search on ISI Web of Science (including conferences proceedings), PubMed, ACOGNET, ProQuest, OVID, Cochrane Collection, the Lancet onLine Collection, MDConsultant, New England Journal of Medicine, American College of Physician onLine Resources, Highwire Journal, and Citation Index. The following keywords used: anatomical urethral stabilizing mechanism; defects of the urethral stabilizing mechanism; etiology of female stress urinary incontinence; female urethral anatomy; female SUI; female stress urinary incontinence; pathophysiology of female stress urinary incontinence; SUI; urethral anatomy, urethral stabilizing mechanism.

Results

Findings

Electronic and manual searches of the medical literature failed to identify any article with regards the occurrences of site-specific defects within USM. Many authors reported fragments of urethral suspensions or support anatomy, but the USM as one unit [1–8 11–18],

USM defects

Four out of fifteen subjects (26.6%) demonstrated the presence of site-specific defects within the anatomical urethral stabilizing mechanism. Stratum-by-stratum dissections determined that the ventral vestibule endopelvic fascia defects are identifiable, Fig. 1A, when compared with the intact ventral vestibule endopelvic fascia, Fig. 1B. Upon removing the ventral vestibule endopelvic fascia, the pars intermedia and vestibular bulbs rest on the ventral perineal membrane and, in some subjects, identifiable site-specific defects were present, Figs. 1C and 2. Multiple defects occurred within the ventral perineal membrane, Fig. 2, periurethral, and paraurethral defects were present Figs. 4 and 6, with long-lasting everted, thick, and fibrotic edges of the defects, Figs. 1C and 2. In one case, the ATFP distal condensation defect was present, Fig. 3. Another subject demonstrated para- and peri-urethral defects, Figs. 4 and 6. Additionally, one subject presented with the right vaginourethral ligament defect, Fig. 5.

The suburethral sportive anatomy

a. In each subject of this study, the posterior urethral wall imprint within the anterior-distal vaginal wall was present (the Terminologia Anatomica does not include this term [8]). The length of the anterior-distal vaginal imprint was between 1.5 cm and 3.1 cm (mean 2.5 ± 0.6 cm); at the upper part, the width measured 1.5 cm (on the average), and then the groove narrows to 1.0 cm for

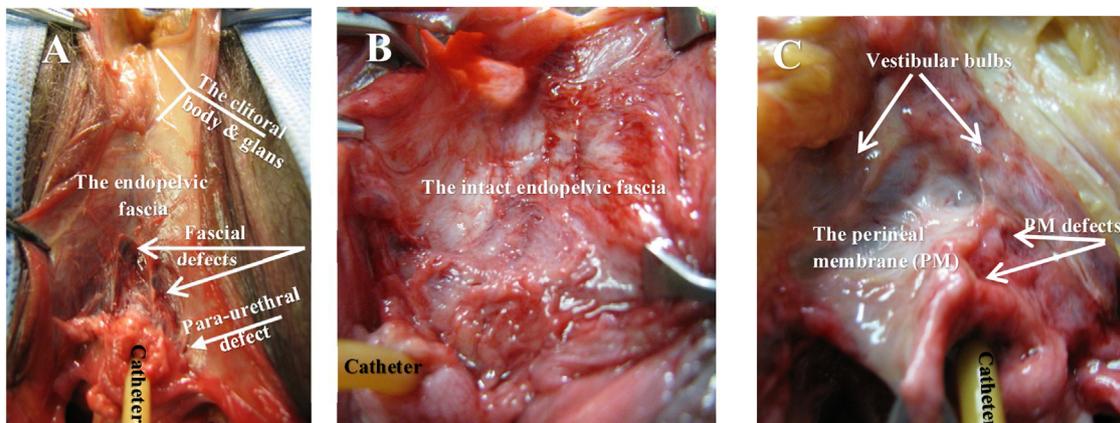


Fig. 1. Presenting the ventral vestibule endopelvic fascia (above the urethra) with the paraurethral, the periurethral, and the perineal membrane defects.
 A. Depicting multiple defects within the vestibule endopelvic facial defects (periurethral and paraurethral defects). The clitoral body, glans, and a Foley catheter serve as landmarks.
 B. The intact vestibule endopelvic fascia extends from the pubic arch to the suburethral region. The inferior surface of the vestibule endopelvic fascia naturally fuses with the superior perineal membrane (PM).
 C. Removing the endopelvic vestibule fascia unveiled the shiny perineal membrane with its defects, the pars intermedia, and the vestibular bulbs.

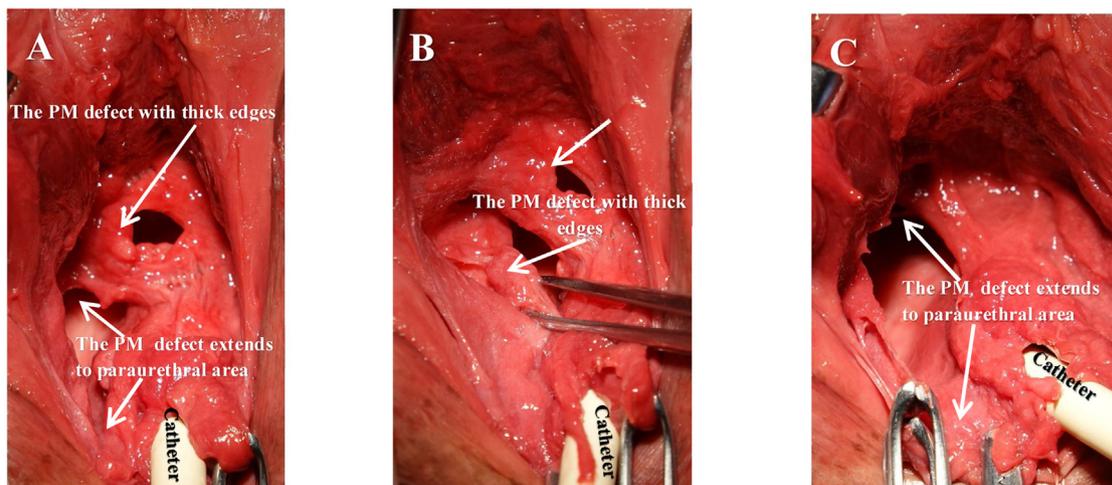


Fig. 2. Showing ventral perineal membrane and para-urethral defects.
 A. The more substantial lateral ventral perineal membrane defect extends to the paraurethral area with thick, everted, and healed defect's edges. There is also noticeable the smaller sub-symphysis ventral perineal defect with thick edges. Thick, everted edges in both defects document the presence of old lacerations.
 B. Separation of the perineal membrane from the ischiopubic ramus with thick, everted and fibrotic edges are visible. The sub-symphysis defect is the smaller defect with everted, thick, and fibrotic edges, which indicates long lasting lacerations.
 C. The lateral perineal membrane defect extends to the paraurethral area.

the remaining length. The distal posterior urethra rests within this shallow groove (imprint) in the midline of the anterior-distal vaginal wall.

b. The dorsal perineal membrane merges with the lateral vaginal wall and the perineal body. The superior surface of the perineal membrane interlocks with the vaginal vestibular bulbs, parts intermedia of the vaginal vestibular bulbs, and clitoral crus. The perineal membrane also fuses with the superior fascia of the levator ani muscle. The inferior surface of the perineal membrane naturally fuses with the urethral compressor muscle, the urethrovaginal sphincter muscle (both of these muscles creates the external urethral rhabdosphincter muscle), Fig. 1.

c. The distal urethral wall receives support from the lateral vaginourethral ligaments (the Terminologia Anatomica does not include this medical term [8]). These ligaments insert to 1) the anterolateral distal vaginal wall; 2) the lateral edge of the urethral

wall; 3) the clitoral body; 4) retropubic space. Grossly, the ligament appears to have a sharp edge and covered by white, thick tissue, Fig. 5A and B. Bilaterally, these ligaments run from the lateral vaginal wall towards the lateral side of the urethral wall where they insert into the urethral wall, approximately 4 to 5 mm from the external urethral meatus wall; then they run-up and attach to the clitoral body and run under the pubic bones to the Retzius space.

d. The vaginal wall supports the posterior distal urethra and the bladder neck. The suburethral part of the vaginal wall is connected indirectly with the superior surface of the ATLA, ATFP, and the paravaginal endopelvic fascia. The entire anterior-distal vaginal wall enhanced the anatomical stability of the urethra.

e. The pubourethral septum term applies to the part of urethropelvic ligament located under the urinary bladder (the Terminologia Anatomica does not include this term [8]). The distal

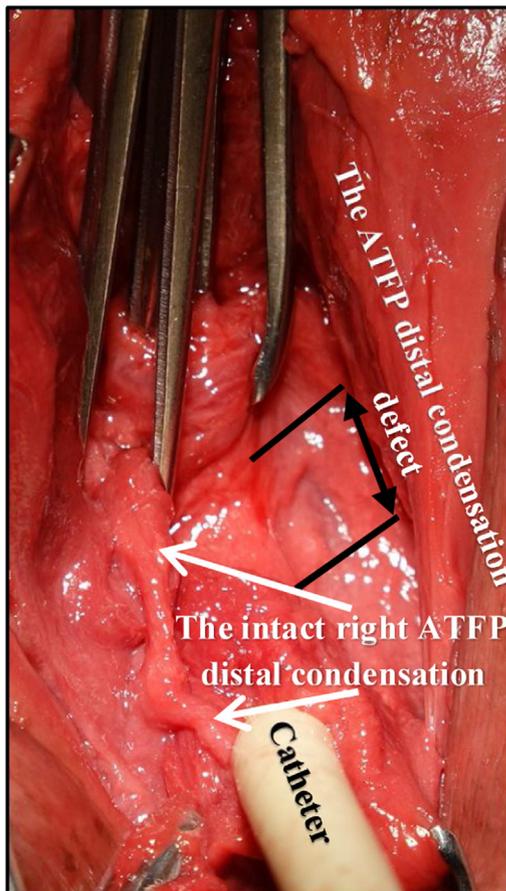


Fig. 3. Depicting the defect of the distal condensation of the arcus tendineus fasciae pelvis (ATFP). The right side of the distal condensation of ATFP is intact and runs parallel to the left side of the defective distal condensation of ATFP (the black bracket embraces the defect). The cord-like structure of the ATFP distal condensation is ruptured. The well-organized reparative tissues are visible (more intense pink color).

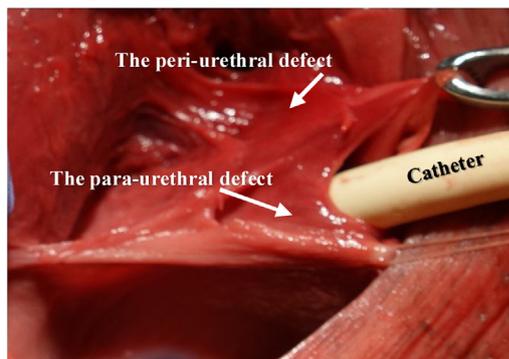


Fig. 4. Presenting the peri- and para-urethral defects. The thick edges of the defects indicate the old lacerations.

urethra rests within a loop of urethropelvic ligament, which creates two arms; the upper and lower arm fuses with each other in the midline of the superior and inferior surfaces of the urethral wall, and then they run in the form the cord-like structure, which attaches this ligament to the ATFP and the pubovaginalis portion of the pubococcygeus muscles. The upper arm of the urethropelvic ligament also fuses with the internal obturator muscle fascia, the ATFP distal condensation, and then intimately fuses with the anterior aspect of the symphysis pubis. The lower arm of

the urethropelvic ligament fuses with underlining vaginal wall. The urethrovaginal sphincter muscle blends posteriorly with the perineal body.

The anatomy of urethral suspensory anatomical structures consists of: a. The ATFP distal condensation extends from the retropubic space to the prepubic space in the form of a cord-like structure (the average length of 2.5 cm). The distal tissue condensation of ATFP suspends the urethra wall to the lower-inner part of the pubic bone arch, Fig. 3.

b. The periurethral ligaments located anteriorly to the vesicourethral junction and traverse in the Retzius space in a transverse orientation, and they extend to the lateral-medial aspect of the puborectalis muscle. Therefore, the proximal part of the anterior periurethral ligaments attaches the anterior urethral wall to the levator ani muscles.

c. The paraurethral ligaments also located in the Retzius space and they run in an oblique orientation. The proximal part of the ligaments inserts into the lateral urethral wall, and the distal part fuses with the periurethral ligaments. This study verified that paraurethral anatomy comprises not only from paraurethral ligaments but also consist of the connective tissues, which plays a role in stabilizing the urethra by connecting the urethra wall to the vaginal wall and the pubococcygeus muscle (the vaginolevator attachment). d. The retropubic space houses not only mentioned above ligaments but also the levator ani muscles, paravaginal fascia, the ATFP, and ATLA. These structures are also a part of the anatomical urethral stabilizing mechanism.

e. The ischiocavernosus and bulbocavernosus muscles are fusing in the vicinity of the distal urethra and cover the urethral wall partially. It is functionally unclear what a role these muscles play in the urethral stabilizing mechanism. An electromyographic study in combination with urodynamic study can clarify this uncertainty.

f. The urethrovaginal sphincter muscle, Fig. 7, plays dual anatomical roles as a) the part of the external urethral sphincter, which is composed of urethrovaginal sphincter muscle and the urethral compressor muscle (rhabdosphincter); b) the vaginal orifice sphincter, which prevents the vaginal introitus from gaping in harmony with the bulbocavernosus muscle.

Comment

Main finding

The urethral stabilizing mechanism is not a specific, a single anatomical structure but it is a composition of multiple anatomical structures located in the different regions such as 1) prepubic, 2) retropubic, 3) lateral, and 4) posterior. The current study established that site-specific defects within the USM occur in 26.6%. Furthermore, this investigation verified that the pubourethral ligaments are the distal condensation tissue of ATFP [18].

Strengths and limitations

The strength of this investigation rests in establishing occurrences of site-specific defects within the USM and determining the anatomical composition of the USM. The anatomical urethral stabilizing mechanism consists of interlocking anatomical supportive, suspense, and connective tissues. Currently, surgical interventions do not have the sophistication to reconstruct site-specific anatomical defects within USM. The number of the study's participants was sufficient for determining the occurrences of site-specific defects within the USM and documenting the existence of the anatomical urethral stabilizing mechanism.

A weakness of this study is related to the fact that one researcher interpreted the anatomical findings; although, always

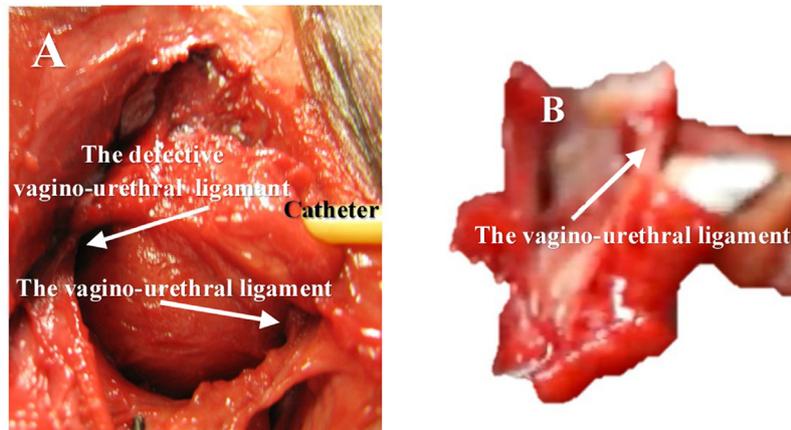


Fig. 5. The lateral vaginourethral ligament is displayed.
 A. Demonstrating the intact left lateral vaginourethral ligament within the anterior-distal vaginal wall and the defective right lateral vaginourethral ligament.
 B. The background from the original image of the vaginourethral ligament was removed to bring the ligament itself into the view.

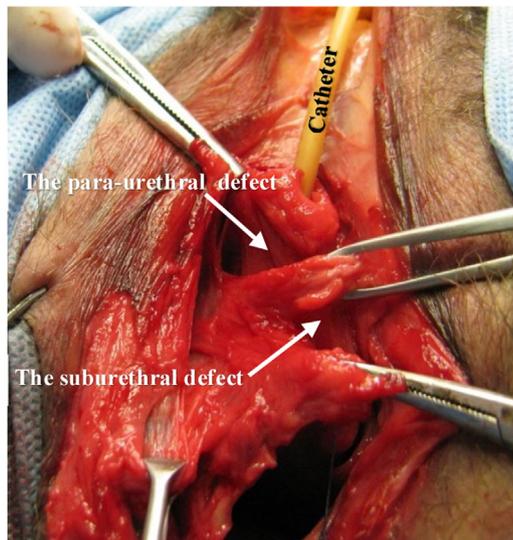


Fig. 6. Presenting the dorsal perineal membrane defect (the suburethral defect) and the paraurethral defect in the same subject. The tick everted edges of the defect are present.

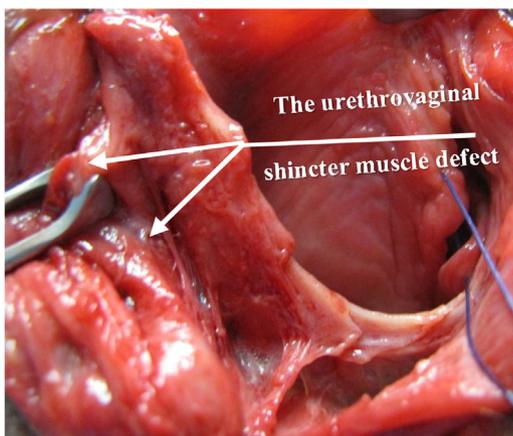


Fig. 7. Grasping the edge of the urethrovaginal sphincter muscle defect between the jaws of the instrument. Such a defect not only negatively affects the external urethral sphincter mechanism but also the vaginal introitus mechanism.

anatomists and gynecologists were present to discuss the findings. New terminology and the subjects' age variations could also influence the anatomical evaluation. Additionally, the absence of the urinary stress incontinence history of the subjects can be considered a weakness.

Interpretations

Four out of fifteen subjects (26.6%) demonstrated the presence of site-specific defects within the anatomical urethral stabilizing mechanism. However, the current study was not able to establish how the USM site-specific defects correlate to clinical symptoms of stress urinary incontinence in women, and new clinical-scientific research is needed to determine whether such a correlation exists.

Existing two-separate defects within the ventral perineal membrane occurred in one subject, (the lateral detachment of the ventral perineal membrane from the ischiopubic rami and the sub-symphysis pubic area), Fig. 2. In the current study, everted, thick, and fibrotic edges of the defects documented long-lasting duration of the defects, Fig. 2; therefore, anatomical dissections did not create these defects. In one case, the ATRP distal condensation defect was present, Fig. 3. Also, in one subject, the para- and peri-urethral defects were recognized, Figs. 4 and 6. The right vaginourethral ligament defect occurred in one subject, Fig. 5.

Denervation and collagen deficiency can also cause urinary incontinence in women [2,19,20]. Leading theories of female stress urinary incontinence do not include site-specific defects as the pathophysiology of urinary incontinence. Utilizing the current study findings may help to develop new surgical interventions to treat female stress urinary incontinence without using surgical slings, surgical meshes or permanent sutures. Eliminating surgical slings, meshes, and permanent sutures from the therapy of female stress urinary incontinence can eradicate low, but severe complications associated with using these materials.

There are noticeable differences between the current study's findings and previous scientific research results. Prior scientific-clinical studies did not evaluate overall anatomical structures responsible for creating the anatomical urethral stabilizing mechanism but did the particular anatomical structure(s) [1,2,5,14]. The current anatomical investigation focused on determining the anatomical structures of the urethral stabilizing mechanism and examining the USM for site-specific defects. The results of the current study are auspicious in providing bases to

establish a correlation between site-specific defects and symptoms associated with female stress urinary incontinence.

The discovery of site-specific anatomical defect occurrences within the urethral stabilizing mechanism in women adds new information with regards to the urogenital tract. This finding constitutes a base for forming a hypothesis that those defects may play a role in female stress urinary incontinence (a separate clinical-scientific study should test this supposition). Additionally, the new anatomical discovery of the urethral stabilizing mechanism expands our knowledge about the urogenital tract anatomy.

Conclusions

Site-specific defects occur within the urethral stabilizing mechanism in women. The urethral stabilizing mechanism consists of multiple anatomical structures located in different anatomical regions, and they fuse with the urethral wall.

Contribution to authorship

The author solo work.

Funding

None.

Details of ethics approval

The Local Ethics Committee approved the study protocol (AKBE 146/12).

Disclosure of interest

None.

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