INTRODUCTION

Vaginal laxity is a symptom of pelvic floor dysfunction, with vaginal looseness being the most common complaint. The leading cause of vaginal looseness is unknown, but it is assumed that it is caused by pregnancy and the vaginal delivery process. Another symptom that may arise is reduced vaginal sensation. One therapeutic option for vaginal laxity is fractional erbium laser management in vaginal laxity. This review aimed to explain the usage of erbium laser management in vaginal laxity.

VAGINAL LAXITY

Vaginal laxity is rarely discussed in the medical literature. The medical literature mainly discusses urinary incontinence symptoms, decreased vaginal sensation during sexual intercourse, and loss of sexual satisfaction. Vaginal laxity is also often considered genital organ prolapse; thus, there is a need for a common nomenclature for vaginal laxity. Specific questioning is required during the clinical examination to differentiate it from genital organ prolapse.

Vaginal laxity differs from vaginal prolapse. In vaginal laxity, the vaginal tissue becomes loose or relaxed. The frequent complaint of vaginal laxity is reduced density. In contrast, vaginal prolapse is the displacement of pelvic organs, such as the bladder, rectum, urethra, or small intestine, to push on the vaginal wall, leaving the normal position. The clinical symptoms are usually a "drop or fall" sensation and discomfort or pain during sexual intercourse.

Vaginal laxity is considered to be the result of stretching of the introitus of the vagina during childbirth. The most common effect is reduced physical sensation and satisfaction with sexual intercourse. The exact prevalence of vaginal laxity remains unknown. The number of women affected is estimated to be about 25%–63% of sexually active adults, and this was obtained from a survey of patients attending an urogynecology clinic. In a prospective study with validated questionnaire data, which included 2,621 women, vaginal laxity was reported in 38% of the women. It was significantly associated with parity, prolapse symptoms, stress urinary incontinence, overactive bladder, reduced vaginal sensation during intercourse, and a more unsatisfactory sexual life in general.

Research conducted by members of the International Urogynecological Association assessed how doctors understand and manage vaginal laxity. Using the internet-based survey method, which consisted of questions designed to determine practical attitudes regarding
vaginal laxity, the results were obtained for 563 out of 2,235 surveys sent (response rate 25%), most respondents were male (65%) and only minority were female (35% female). The geographical distribution of respondents included Europe (39%), North America (23%), Asia (15%), South America (14%), Australia (6%), and Africa (3%), and vaginal laxity was reported by 83% of the patients. The majority (95%) considered vaginal laxity a disruptive condition that affected relationship happiness and sexual function: lack of self-confidence, inability to please their partner, loss of sensation, and decreased satisfaction. Loose introitus was the most common cause of these symptoms. While only 54% of the patients were offered surgical treatment, surgery was more effective than Kegel exercises or physical therapy. North Americans are more likely to choose and perform surgical treatments for this problem. This study concluded that vaginal laxity is common and can affect sexual function and quality of life.12

In a recent study of plastic surgery patients, vaginal laxity was reported in one in six women. The authors concluded that delivery causes vaginal laxity and affects sexual pressure.13 In a retrospective observational study, 337 patients completed a questionnaire and underwent four-dimensional ultrasound to examine the pelvic floor anatomy. Vaginal laxity was reported in 24% of the patients. This symptom was related to younger age, vaginal parity, prolapse symptoms, and an objective ultrasound result that showed the distance of the levator ani.2

ANATOMY AND HISTOLOGY OF THE VAGINAL WALL

The morphology and physiology of the vulva and vagina are known to change with age. Visible changes occur when a woman reaches puberty, the menstrual cycle starts, and pregnancy and menopause occur.9 The vaginal wall consists of four layers: (i) stratified, nonkeratinized, stratified squamous epithelium; (ii) lamina propria, a dense connective tissue layer consisting mainly of fibrillar and elastin collagen, which is occupied by fibroblasts; (iii) muscle layer consisting of longitudinal inner and outer circular smooth muscle fibers; and (iv) adventitia, a layer of tissue rich in fiber, collagen, and elasticity, which supports the vaginal wall. The lamina propria and muscle layer are two essential layers that provide strength to the vaginal wall.14,15

The function and behavior of the vaginal mucosal epithelium depend on the estrogen effect. Hormonal fluctuations may occur during a woman’s lifetime and the menstrual cycle. The epithelium is rich in glycogen, which is fermented by lactobacilli, which lowers the pH of the vagina. The lamina propria mainly consists of collagen fibers, elastin, small blood vessel plexuses, lymphatic vessels, and nerves. The lamina propria papillae are rarely present on the anterior vaginal wall and more prominently on the posterior wall.14

The biomechanical properties of the vaginal tissue are controlled by collagen and elastin. Collagen fibers are stiff and not easily distorted, and while elastin fibers produce tissue elasticity, collagen fibers are a determinant of the strength of the vaginal wall. The primary collagen subtype is subtype I, which forms large and robust fibers. Meanwhile, subtype III takes the form of small and low tensile fibers and contributes to tissue elasticity. Subtype V, which forms small fibers with low tensile strength, is usually located in the core of the fibrils.16

PATHOPHYSIOLOGY OF VAGINAL LAXITY

The pathophysiology of vaginal laxity is not fully understood, but it has been agreed that there is a relationship between vaginal laxity and pregnancy and childbirth. In labor, there is excessive stretching of the vaginal wall and introitus, associated with increased dimensions of the levator and trauma to the levator ani muscle (excessive avulsion or distension).17–19

Pregnancy causes several biomechanical and behavioral changes in humans and animals. It causes ligament weakness and increased vaginal distensibility. Pelvic floor muscles in mice also exhibit certain behaviors during pregnancy. Increased stiffness and sarcomere numbers are protective processes against perineal trauma during childbirth.20

High stretch occurs in the puborectalis muscle during vaginal delivery. The length that occurs varies (25–250%). Micro and macroscopic injuries occur when muscle fibers are stretched. Moreover, 10–35% of women develop levator avulsion (damage to the pubococcygeus muscle at bone insertion). Levator avulsion causes a Hiatal enlargement of 20–30%, and the pelvic floor muscles do not contract easily.13

In one study, 153 nulliparous women at 35–39 weeks’ gestation completed a questionnaire about urinary incontinence symptoms. The study also included three- or four-dimensional ultrasound examinations, repeated 3–6 months postpartum. The majority of the women (54.9%) came back to the clinic at 3-6 months after deliveries, 71% of deliveries were vaginal, and 29% were cesarean. There was a significant increase in bladder neck decline, pelvic organ decline, and Hiatal levator distension. A levator avulsion was seen in 15% of vaginal deliveries. Vaginal laxity was the most common symptom (60.7%). Thus, it can be concluded that there are significant changes in the pelvic organs and levator Hiatal distension after normal vaginal delivery.21

The vaginal and pelvic support muscles are formed at the cellular level from the connective tissue integrity and adhesions between the vagina, pelvic sidewalls, and levator ani muscles. The connective tissue of the vagina and surrounding structures contains collagen, elastin, glycoproteins, hyaluronan, and proteoglycans. It is actively restructured throughout a woman’s lifetime, especially during hormonal changes, such as pregnancy and menopause. For example, during the hypoestrogenic state of menopause, modifications to the underlying architecture can affect overall elasticity.7

Fibroblasts are critical regulators in repairing and rebuilding soft tissues throughout the body, including the vagina. They maintain the integrity of the vaginal wall tissue and avoid any prolapse. The microenvironment of the vaginal tissue in prolapsed patients is more rigid and has a different extracellular matrix composition than healthy vaginal tissue. Rigidity is associated with the production of a-smooth muscle actin (α-SMA). The vaginal extracellular matrix induces myofibroblasts due to the increased
expression of α-SMA and collagen genes. It suggests that the rigidity and content of the extracellular matrix regulate vaginal myofibroblast differentiation. Vaginal fibroblasts appear to recognize the prolapsed extracellular matrix as scar tissue that requires remodeling.25

ER: YAG FRACTIONAL LASER

The Er: YAG laser (2,940 nm) was introduced in the 2000s and targeted superficial skin surface resurfacing. Er: YAG lasers have a higher water absorption coefficient than CO2 lasers, are approximately 10 times more efficient, and erode the tissue with much less thermal damage (5–10 μm). The tissue reaction to a laser depends mainly on the wavelength because the laser light wavelength determines the absorption mechanism and depth of penetration. Visible laser light is absorbed by a specific pigment or chromophore, depending on the laser light’s color (or wavelength).27

Infrared lasers such as Er: YAG lasers and CO2 lasers only interact with water, heating the tissue for evaporation or ablation. The ablative laser causes thermal damage to the epidermis and dermis. The damage may stimulate neocolagenesis, tissue rejuvenation, and skin tightening.24

In the 1980s and the 1990s, ablative lasers were very popular for treating aging facial skin due to sun rays and skin sagging. The results are excellent and dramatic, but the side effects, such as pigmentation and scar development, are significant, and the recovery time is also long. Because of the many side effects that occur, the results are not satisfactory. In response to this dilemma, the concept of fractional photothermolysis has emerged. In fractional photothermolysis, thermal micro-injury appears in spatially distributed columns in the therapeutic area called microscopic treatment zones (MTZs), resulting from focused laser irradiation. In each MTZ column, there is sufficient energy to induce thermal heating and ablation without spreading to adjacent tissues. Fractional irradiation allows deeper penetration into the dermis, which causes denaturation, eliminates debris, collagen remodeling, maintains epidermal integrity, shortens healing time, and reduces side effects.24,25

The Er: YAG laser is a near-infrared ablative laser emitting light at a wavelength of 2,940 nm, with a water absorption coefficient that is much higher (12,800 cm-1) than that of the CO2 laser (800 cm-1), thus producing laser energy. Er: YAG is 12–18 times more efficiently absorbed by water-containing tissues than CO2 laser energy (Mcllwee and Alster, 2019). The Er: YAG laser penetration depth is limited to approximately 1–3 μm of tissue per J/cm², compared to 20–30 μm for CO2 lasers. Thus, the Er: YAG laser allows for more precise skin ablation with minimal thermal damage to the surrounding tissue. The approximate resonant tunneling structure is 10–40 μm. Using the Er: YAG laser at an energy density of 5 J/cm² vaporizes the epidermis after four phases, whereas energy densities in the range of 8–12 J/cm² require only two phases. The Er: YAG variable-pulsed laser, with pulse durations ranging from 10–50 ms, induces direct tissue contraction and a rate of healing that is intermediate between short Er: YAG pulses of pulse duration 250–350 μs and the CO2 laser.14,25

The Er laser: YAG is associated with reduced post-laser discomfort, erythema, and edema and a faster overall cure time compared to the CO2 laser. In contrast, the CO2 laser treatment is bloodless because of its ability to photogulate blood vessels with a diameter smaller than 0.5 mm, whereas bleeding increases with repeated phases of the Er: YAG laser.14

Histologic examination of the Er: YAG laser was described by Orringer et al. in evaluating human arm skincare. Ten patients were treated with a 2,940-nm single-phase laser (SmoothPeel, Candela Corp, Wayland, MA, USA) using a 5-mm spot size and an energy of 750 mJ. Punch biopsies were performed at baseline 1, 3, 7, and 14 days after the procedure. After this superficial treatment, minimal epidermal damage occurred via loosening keratinocyte cell adhesion and sparse granularlayermicro-vesicles. The basement membrane was intact, as confirmed by laminin-2 immunohistochemical staining. Molecular studies have revealed increased epidermal keratin 16 expressions, a marker of epidermal injury. Interestingly, reports suggest that HSP-70, which participates in procollagen synthesis during normal wound healing, does not increase. Furthermore, there is no evidence of thermal damage to the dermis with this superficial treatment. However, epidermal damage induces a molecular cascade including increased interleukin (IL)-1β and IL-8 and upregulation of extracellular matrix degradation proteinases, followed by a significant increase in type I and III procollagen. This study showed that superficial Er: YAG laser treatment could induce skin neocolagenesis.27

Orringer et al. measured a full-field CO2 laser (Ultrapulse; Lumenis Ltd., Yokneam, Israel) by analyzing the mRNA transcripts of collagen I and collagen III. Production of procollagen type I and procollagen type III mRNA peaked 21 days post-therapy and increased for at least six months. Changes in collagen levels were accompanied by increases in several cytokines (IL-1β, tumor necrosis factor [TNF]-α, and transforming growth factor-β1). Significant increases in the mRNA levels of matrix metalloproteinase (MMP)-1, -3, -9, and -13 were also noted. Increased expression of some MMPs preceded an increase in the production of procollagen I and procollagen III. Based on these findings, the following scheme was proposed: proinflammatory cytokines (IL-1β and TNF-α) induce the expression of multiple MMPs. MMP, including collagenase (MMP-1 and MMP-3) and gelatinase (MMP-9), break down collagen. The level of MMP-9 continues to increase during the post-treatment period, as evidenced by the continuous removal of collagen. Photodamaged collagen degradation facilitates replacement with new, well-organized collagen bundles.25

ER: YAG FRACTIONAL LASER RESEARCH ON VAGINAL LAXITY

A preliminary study was performed on 21 patients who received vaginal tightening laser therapy with an Er: YAG fractional laser, comprising two sessions at 15–30-day intervals, at 90 J in the inner vaginal area and 10 J in the vaginal introitus. The Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire 12 is a standard assessment tool for pelvic organ prolapse, urinary incontinence, and sexual satisfaction. The results obtained were as follows: 95% reported
a significant improvement in vaginal tightness, and 85% of partners reported significant vaginal tightening. Nearly all patients (95%) reported better sex after laser treatment, except for one patient who did not. Three patients with urinary incontinence reported improvement after laser treatment, two reported improvements, and one reported she was cured, with no side effects and minimal discomfort.⑥

In an Er: YAG fractional laser therapy study conducted in Korea on 30 postpartum women with VRS and vaginal atrophy with a mean age of 41.7 years, patients were randomly divided into Group A and Group B, who received treatment at 1–2 weekly intervals, using 90- and 360-degree probes, respectively. Group A received the first two sessions with 360° and the last two sessions with 90° using the multi-micropulse mode, 1.7 J energy per shot, three multiple shots, and three passes per session. Group B received several modes, with 90° across four sessions (same parameters as Group A). During the last two sessions, two additional sessions were given with 360° in extended pulse mode and 3.7 J per shot. Perineometer assessment and histological specimen biopsy were performed at baseline, and sexual satisfaction was assessed. After two months of laser therapy, the perineometer results showed a significant increase in vaginal wall relaxation in all patients. In addition, 70% of the patients reported increased sexual satisfaction with vaginal tightening, and 70.6% of the couples found that the vagina was firmer. Histology showed a thicker epithelium and denser lamina propria than before laser therapy.⑦

One study compared the efficacy of Er: YAG laser therapy versus hormonal replacement therapy using estril in 50 patients with vaginal atrophy. The study was divided into two groups: Group A was treated with long-term estril (0.5 mg/day) three times a week for 2 weeks, followed by the same estril dose twice a week for four weeks. Group B received short-term, 2-week estril therapy, followed by three Er: YAG laser sessions at 3-week intervals. Both groups showed reduced vaginal atrophy, and laser therapy showed a better and longer-lasting effect.⑧

CONCLUSION

In conclusion, the Er: YAG fractional laser shows promising results as a treatment for vaginal laxity; namely, there are improvements in vaginal tightness and increased satisfaction with therapy results, with no side effects or minimal side effects. Further research is necessary to explain all clinical aspects of Erbium laser in vaginal laxity management.

DISCLOSURE

Author Contribution

The author contributed to all processes publishing this review.

Conflict of Interest

The author stated no conflict of interest for publishing this review.

Funding

The author stated that no grant or third party was involved in funding.

Ethics Consideration

Not applicable in this review.

REFERENCES


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⑨ Link between histological and biomechanical properties of vaginal tissue in women with pelvic organ prolapse? Int Urogynecol J. 2016;27(4):629-35.


