Research Article

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The pelvic floor muscle in man and its disfunctions : role played in functional bladder outlet obstruction and Reverse of fibrosis in Chronic Bacterial-Induced Prostatic Inflammation

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Abstract

Aim of this work is to analyse come condition that can affect the urinary flux form the bladder neck and the uretra with inflamatory or not inflamatory pathogenetic movens.All this condition are reponsible of an inefficient urinary flux with all it is involved.

The role payed by pelvic floor muscle is analyzed as well as the infective -inflamatory role played by pr ostatic chronic infection.

Are also reported literature about the reverse action played by some antibiotic and antinflamatory therapy to reverse the deposition of collagen and fibrotic process in the interconnection bladder neck and urethra.

This work can have relevance both in benign patology but also in oncological setting.

Keywords: pelvic floor muscles , striated urethral sfincthere ,bulbocavernosus, puborectal muscles, Non-Relaxing Pelvic Floor Dysfunction, functional Bladder Outlet Obstruction, physiology , pathology, oncology physiotherapy

Introduction

Before to start this work it is interesting to observe some references related the pelvic floor muscle and the physiology of urination:

From https://ashwinsridharurology.com/how-to-improve-urine-flow/

How to Improve Urine Flow: 14 Safe, Doctor-Approved Tips Ashwin Sridhar.

"How stronger pelvic muscles PM improve flow Firm yet

supple pelvic fibres prop up the bladder neck and keep the urethra aligned. When they contract and relax on cue, the channel opens fully, letting urine exit quickly and reducing post-void dribble."

The Male Pelvic Floor by Cathy Watson

"The male pelvic floor muscles PFM help: in control bladder and bowel function (helps control the opening/closing of the urethra and the anus),give support to your back, hip and pelvis gives support to the organs (bladder, rectum, prostate , seminal vesicles) aid in the sexual function"

Cleveland clinics : Pelvic Floor Muscles Medically Reviewed.06/27/2025.
"Squeezing your pelvic muscles PM narrows your urethra and anus so waste material can't escape. Relaxing these muscles widens these passages so you can pee, poop or fart."

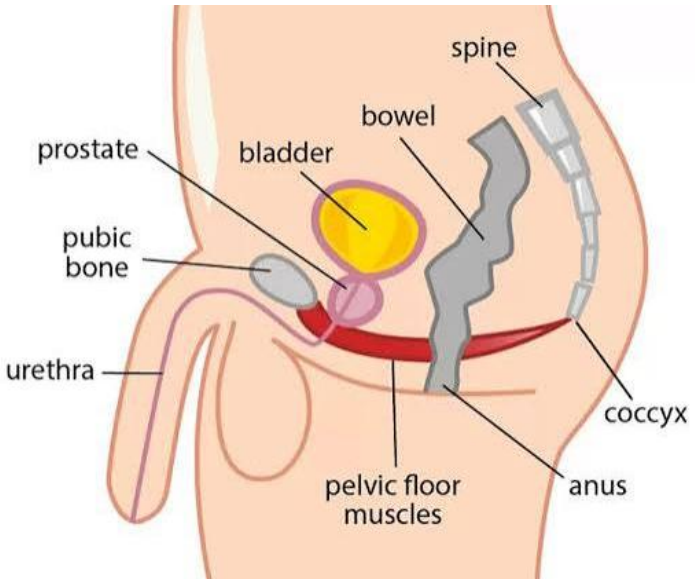

Figure n. 1 The pelvic floor muscles

And in Urology & Continence Care Today | July 2026 by Rachel Leaver
"the bladder muscle BM and sphincters need to coordinate when they open and close with muscle contraction and relaxation."

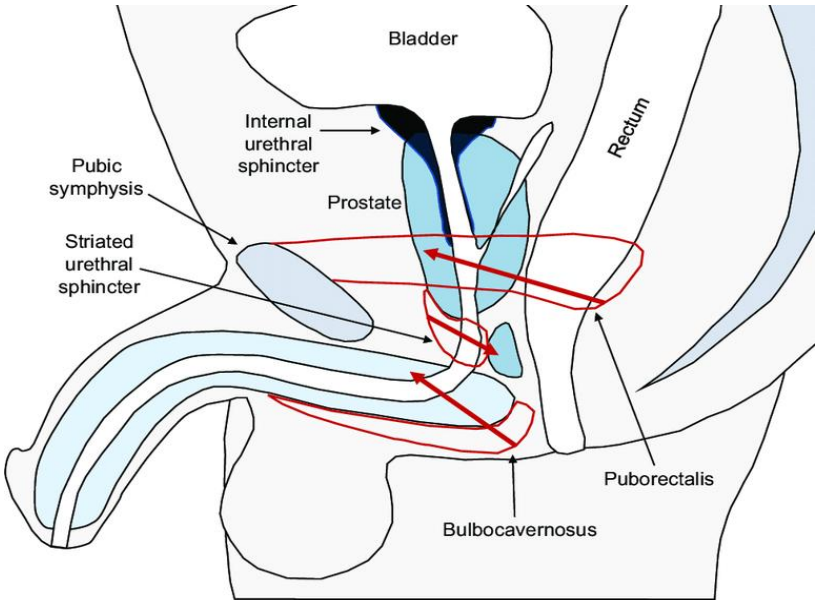

Figure n 2 from Physiotherapy management of incontinence in men 2021 J. of Physiotherapy DOI:10.1016/j.jphys.2021.02.010 The male urogenital system and pelvic floor muscles PFM . Thick arrows indicate the direction of action of horseshoe-shaped muscles to pull the urethra closed. Modified from Hodges et al

Physiotherapy management of incontinence in men
Irmina Nahon j. of Phisiotherapy 67- 2021
"The striated urethral sphincter (SUS) lies below the prostate and around the anterior and lateral sides of the urethra, attaching to the central tendon of the perineum and perineal fascia, forming an omega shape.

On contraction it is the strongest of the striated muscles SM , pulling on the urethra posteriorly against the fascia and perineal body between the urethra and the anus. This is countered by the contractions of the puborectalis and bulbocavernosus BC , which pull the urethra and the perineal body anteriorly.

The puborectalis PR is part of the levator ani. Together with the pubovisceralis PV and the iliococcygeus, these muscles sandwich the SUS. The bulbocavernosus BC , which extends to the perineal body superiorly and cups the penile bulb inferiorly, compresses the bulb of the penis, also constricting the distal urethra DU opposing the SUS.

Emerging research works evidence indicates that these muscles inside the pelvis contribute to the control of urethral pressure UP by differentially activating in a coordinated manner.

During the storage phase of the micturition, the detrusor of the bladder is inhibited. The SUS has tonic activation at rest and phasic activity observed before and during in creases in intra-abdominal pressure IAP . Stafford et al have also shown that both bulbocavernosus BC and puborectalis PR contract in a distal-to-proximal sequence during activities that increase intra abdominal pressure, to increase the urethral pressure UP.

Voiding is not simply the reversal of storage. A complex series of coordinated peripheral and central pathways, mainly under para sympathetic PS control, relax the urinary sphincters while activating the detrusor muscle DM . The central mechanism of micturition CMM is poorly understood. Fullness is a stimulus for voiding, and urge is an primary motivator. If it is conceptualised that urgency is the Bladder's distress or 'pain', it can be understood how bladder dysfunctions BD might respond well to the bio-psychosocial approaches used to manage pain. This approach extends the influences onpain beyond the biological and includes the social and psychological. Pain is perceived to be worse when there are elements that increase the danger that the body is in. In this paradigm, 'danger' is an social embarrassment when the incontinence occurs."

The SUS—often referred to as the external urethral sphincter EUS —is a vital muscle in the pelvic floor complex PFC . Its primary function is to compress the urethra, inhibit involuntary bladder reflexes IBR, and maintain urinary continence, particularly during sudden increases in abdominal pressure from coughing or sneezing.

The bulbocavernosus reflex
By Olushola Odusanya, Vaibhav Modgil and Ian Pearce 2023
Urology news
"The BCR bulbocavernosus reflex involves contraction of the bulbo- and ischiocavernosus pelvic floor muscles ICPFM, often referred to as the 'bulbocavernosus BC muscle', in response to stimulation of the glans penis.

In males, the BC is located at the base of the penis . The BCR is an oligosynaptic reflex arc composed of pudendal nerve fibres PNF and is associated with sacral segments S2-4 in the spinal cord SC . Stimulation of the BC results in compression and ventral motion of the bulb of the penis . Contraction of these pelvic floor muscles PFM also occurs during the ejaculation ."

Functional urinary obstruction FUO related to the puborectalis muscle is often a manifestation of Non-Relaxing Pelvic Floor Dysfunction (NR-PFD) or Pelvic Dyssynergia PD . When the puborectalis muscle PRM fails to relax during urination, it causes functional Bladder Outlet Obstruction FBOO, characterized by urinary hesitancy, a slow or intermittent stream, whit a feeling of incomplete emptying.

From https://urology.ucsf.edu
"The lower urinary tract LUT includes the bladder and urethra, which allows for storage and timely expulsion of urine. Voiding dysfunction VD is a broad term, used to describe the condition where there is poor coordination between the bladder muscle and the urethra. This results in incomplete relaxation or over-activity of the pelvic floor muscles PFM during voiding. Voiding symptoms VS represent a continuum of what is referred to as LUTS."

A misalignment or lack of coordination between the male urethra MU and pelvic floor muscles PFM often indicates pelvic floor dysfunction or voiding dysfunction VD . This causes the muscles to fail to relax properly during urination, leading to a weak stream, hesitancy, and incomplete bladder emptying.If you are experiencing this, several contributing factors and diagnostic paths can help address the issue.

Potential Causes Pelvic Floor Hypertonia: Muscles are too tight or in spasm, pulling the urethral sphincter out of alignment.

Urethral Stricture US : Scarring in the urethra alters the flow path and creates resistance.

Neurological Conditions NC : Nerve issues disrupt the natural coordination between brain, bladder, and pelvic muscles. Prior Trauma or Surgery: Scarring from pelvic injuries or prostate procedures can affect muscular alignment.

Pelvic Floor Dysfunction in Men: What It Is and How to Treat It
K. Beckham 2025

"Pelvic floor dysfunction PFD occurs when these muscles are too tight, too weak, or poorly coordinated. Instead of

relaxing properly to allow urination or bowel movements, the muscles stay tense, leading to a wide range of symptoms that may affect everyday life. What Causes Male PFD?

The causes PFD can vary. Often, multiple contributing factors are at play. Some of the most common include:

Post-surgical trauma PST (prostatectomy or vasectomy),Orthopedic injuries (spine, hip, pelvic injuries)

Excessive exercise or poor lifting mechanics,Chronic constipation or habitual straining ,Long hours of sitting or sedentary lifestyle,Psychological stress PS , anxiety, trauma .

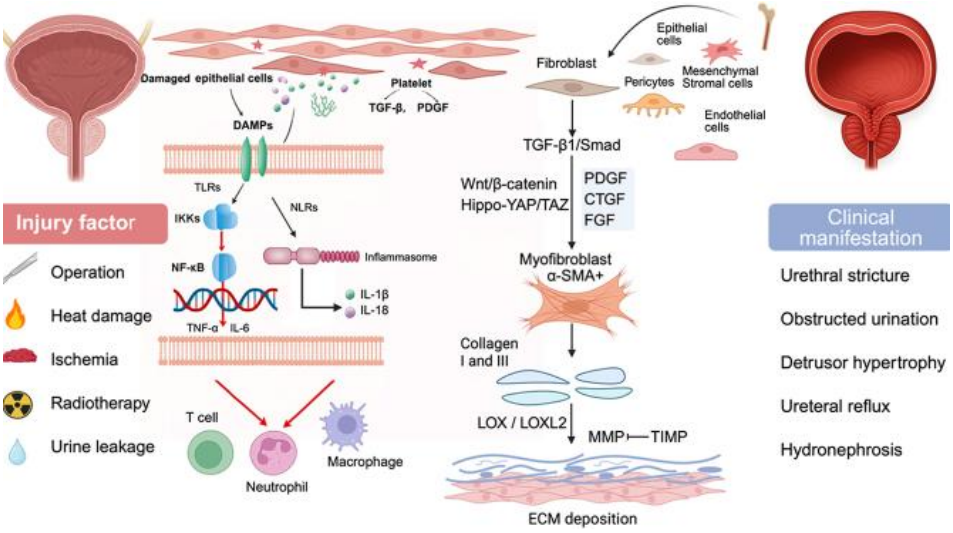

Figure n 3 from Y. REN et al 2025 Pathophysiological progression of bladder neck fibrosis.

Fromhttps://pnnmedical.com/patients/bladder-neck-stricture/
"Bladder neck stricture BNS is a condition in which this opening becomes narrowed, restricting the flow of urine from the bladder. This narrowing can interfere with normal bladder emptying and may lead to urinary symptoms US that require medical intervention to avoid patient complications. Symptoms of Bladder Neck Stricture BNS :Difficulty starting urination,Weak or interrupted urine stream

A feeling of incomplete bladder emptying,Increased need to urinate, especially at night Straining during urination"

Bladder Neck Dysfunction - Symptoms, Complications, and Treatment

Bladder neck dysfunction BND is a condition in which the bladder neck fails to open while urinating. It is commonly seen in men.

Dr. Porwal Himanshi Avadheshkumar

" Constipation is when the stools become hard and cannot pass through the body. These hard stools put pressure on the bladder neck BN , making it difficult for the muscles to work and drain the urine to the urethra."

Material and methods

Whit an observational point of view for the scope of this work various relevant literature involved are reproted and then analyzed.
All literature comes from biomedic database like Pubmed and other.

Figure (1- 4) helps in the general meaning
An experimental experience (case report) is submiteed
After all this an general comclusion is provided

Results

From literature :
According Junyang Jung et al

"The urethral sphincter US can be used to control micturition. Voiding urine begins with voluntary relaxation of the external sphincter ES muscle of the bladder. Parasympathetic impulses induce contractions of the bladder and relaxation of the internal sphincter . Voluntary control of urination is possible only if neural innervation of the bladder and urethra is intact . Occasionally, injury and malfunctioning to any of the supplying nerves causes involuntary emptying of the bladder . This involuntary urination is called incontinence. I n males, during ejaculation, the closure of the urethral sphincter US prevents mixing between urine and semen and backward flow of semen into the bladder ."(1)

A. S. Afyouni et al
"In individuals with NR-PFD Non-relaxing pelvic floor dysfunction NRPFD, this coordinated relaxation fails to occur and the pelvic floor muscles remain contracted during voiding, effectively resulting in a functional bladder outlet obstruction.

Unlike detrusor-sphincter dyssynergia , which results from suprasacral spinal lesions, NR-PFD occurs in neurologically intact individuals and lacks structural or neurogenic abnormalities. It is also distinct from other non-neurogenic, non-structural causes of dyssynergic voiding DV, such

as dysfunctional voiding (DV) and poor relaxation of the external sphincter . While DV typically involves both the external sphincter ES and pelvic floor involvement, NR-PFD selectively affects only the pelvic floor muscles . Pelvic floor nonrelaxation and hypertonicity may also develop in response to chronic visceral pain syndromes. Conditions such as interstitial cystitis IC /bladder pain syndrome, irritable bowel syndrome IBS , endometriosis, atrophic vaginitis, and vulvodynia have all been linked to pelvic floor overactivity PFO via peripheral and central sensitization, nociceptive hypersensitivity, and referred myofascial pain . Dyspareunia may reinforce involuntary muscle contraction and promote a chronic state of non-relaxation, especially when intercourse is continued despite discomfort. A history of childhood or adult sexual trauma has been associated with higher rates of chronic pelvic pain CPP and pelvic floor dysfunction PFD . In many cases, no single causative event is identified. NR-PFD likely results from a constellation of behavioral, anatomic, neurologic, and psychosocial contributors. Once established, the condition may be further perpetuated by chronic pain–related factors, including sleep disturbance, anxiety, and depression, complicating both the diagnosis and its management."

"At rest, tonic activity in the pelvic floor and external urethral sphincter EUS maintains urethral closure pressure above bladder pressure to preserve continence. Effective voiding, by contrast, requires: (a) sustained detrusor contraction, (b) complete and sustained relaxation of the urethral sphincters, (c) coordinated pelvic floor muscle relaxation, and (d) absence of mechanical obstruction (urethral stricture, diverticulum, pelvic trauma). The pelvic floor muscles PFM, in particular, must fully relax and act in synchrony to permit descent of the bladder neck and opening of the urethra, allowing for efficient urine expulsion . In individuals with NR-PFD, this coordinated relaxation fails to occur and the pelvic floor muscles PFM remain contracted during voiding, effectively resulting in a functional BOO.

Pelvic floor physical therapy (PFPT) is the first-line treatment for NR-PFD and serves as the foundation of conservative management for urologic symptoms related to pelvic floor hypertonicity PFH and/or impaired relaxation .

Biofeedback BFB can enhance PFPT by helping patients identify and relax overactive pelvic muscles, especially when symptoms are thought to stem from learned behaviors .

Muscle relaxants, such as oral baclofen, tizanidine, cyclobenzaprine, or diazepam, may reduce pelvic floor spasm PFS and provide symptom relief. Diazepam suppositories are the most commonly prescribed vaginal formulation and are preferred over oral routes due to lower systemic absorption and longer local activity, resulting in fewer side effects.

"Video urodynamics VUD and surface EMG, alongside focused physical examination, are key tools for diagnosing NR-PFD. Pelvic floor physical therapy remains the first-line treatment, with strong evidence supporting its efficacy across sexes. Adjunctive options, including biofeedback, trigger point injections, botulinum toxinBT , and sacral neuromodulation, can benefit patients with refractory symptoms. Cognitive behavioral therapy and integrative modalities are also increasingly utilized."(2)

Hannes Cash et al written
"PBNO Primary bladder neck obstruction there is a persistence of the cranial part of the skeletal muscle cells of the urethral sphincter, which may interfere in the complex micturition process, and that may explain why those patients did not respond to alpha-blockers, typically working on smooth muscle cells.

The structural changes at the BN causing PBNO have been described as "fibrous narrowing and hyperplasia" "fibrous stroma and subepithelial scar, often with inflammatory change", "abnormal morphologic arrangement of the bladder musculature BM (detrusor/trigonal)" a structurally healthy but dysfunctional BN have also been described. Many theories included altered mechanisms of BN opening such as neurological dysfunction of the sympathetic nervous system with a detrusor-sphincter-dyssynergia DSD, abnormal BN striated muscle components of the urethral sphincter US , increase in the density of Neuropeptide Y immunoreactive nerves (supporting the hypothesis of the PBNO as a more functional illness linked to pelvic floor dyssynergia) . PBNO is a dysfunction of the bladder neck BN in which the collum vesicae is narrow or fails to open adequately during voiding, resulting in bladder outlet obstruction .

Hinata described a correlation between an increased prostate volume due to BPH and an increase of collagen fibers and degeneration of muscle bundles in the BN. By progression of prostatic hyperplasia PH , the BN muscles were progressively affected by fibrosis with fragmentation of the smooth muscle sphincter vesicae .

Non-inflammatory pathways
The data on the non-inflammatory possible causes of PBNO, are scarse and weak. Like Yalla , Billis demonstrated a significantly amount of skeletal striated muscle fibers SSMF in transurethral resection of the prostate resection fragments of the BN in PBNO patients than in patients with typical symptomatic BPH. In PBNO patients the fibers were thick, prominent, hypertrophied, and frequently in a parallel distribution; whereas BPH patients showed discrete, thin, and transversally or longitudinally cut fibers. They assumed that in PBNO there is a persistence of the cranial part of the skeletal muscle SM cells of the urethral sphincter US , which may interfere in the complex micturition process, and that may explain why those patients did not respond to the alpha-blockers, typically working on smooth muscle cells . According to Yalla et al hypothized, in a case series, a possible role of unbalanced biomechanics of the pelvis on the urethral and vesical sphincter VS activity by an unknown postural orthopedic condition with pelvic torsion that causes hypercontraction on the pelvic floor PF and interfering with the normal micturition.

Camerota found out that PBNO patients have a high prevalence (76%) of myofascial or articular, mostly nociceptive pain across different regions as a relevant component . They postulated that an interplay of peripheral inflammation, postural imbalance and chronic pain CP could induce nociceptor activation and sympathetic nervous system SNS hyperactivation which in turn leads to a bladder sphincter dysfunction . Hruz described in 60% of patients with diagnosis of category IIIb chronic pelvic pain syndrome a BN hypertrophy as the primary cause of their symptomatology.

Bolton proved that BN shows a physiological variability around its circumference with a uniform response to noradrenaline of whole circumference, significantly stronger response to alpha-adrenergic agonists compared with the cholinergic agonists in the posterior part, whereas the anterior part had no significant differential response to these .Alpha-1-blocker just only have a variable success of 30–60% in PBNO. PBNO-affected patients present symptoms which can be confounded with prostatitis, potentially leading to chronic pelvic pain supporting the idea that PBNO can be also a consequence of previous prostatic inflammations. There is still a debate on the reversibility of subclinical prostatic inflammation. Wong examined the stability of the newly synthesized collagen in an bacterial-induced prostatic inflammation and the reversibility of fibrosis and collagen content after resolution of infection and inflammation. Generating inflammation by injecting E.coli into prostates of mice the authors found the half-life of newly synthesized collagen to be significantly shorter in infected/inflamed prostates than in controls. The authors found antibiotic treatment to reverse the collagenic deposition, supporting that fibrosis linked to infectious disease is a reversible process . Kim et al. demonstrated that profound epithelial mesenchymal transition EMT is observed in lipopolysaccharide induced prostatitis and that the natural HIF-1α inhibitors ascorbate and curcumin were capable to attenuate prostate enlargement both in vivo and in vitro ."(3).

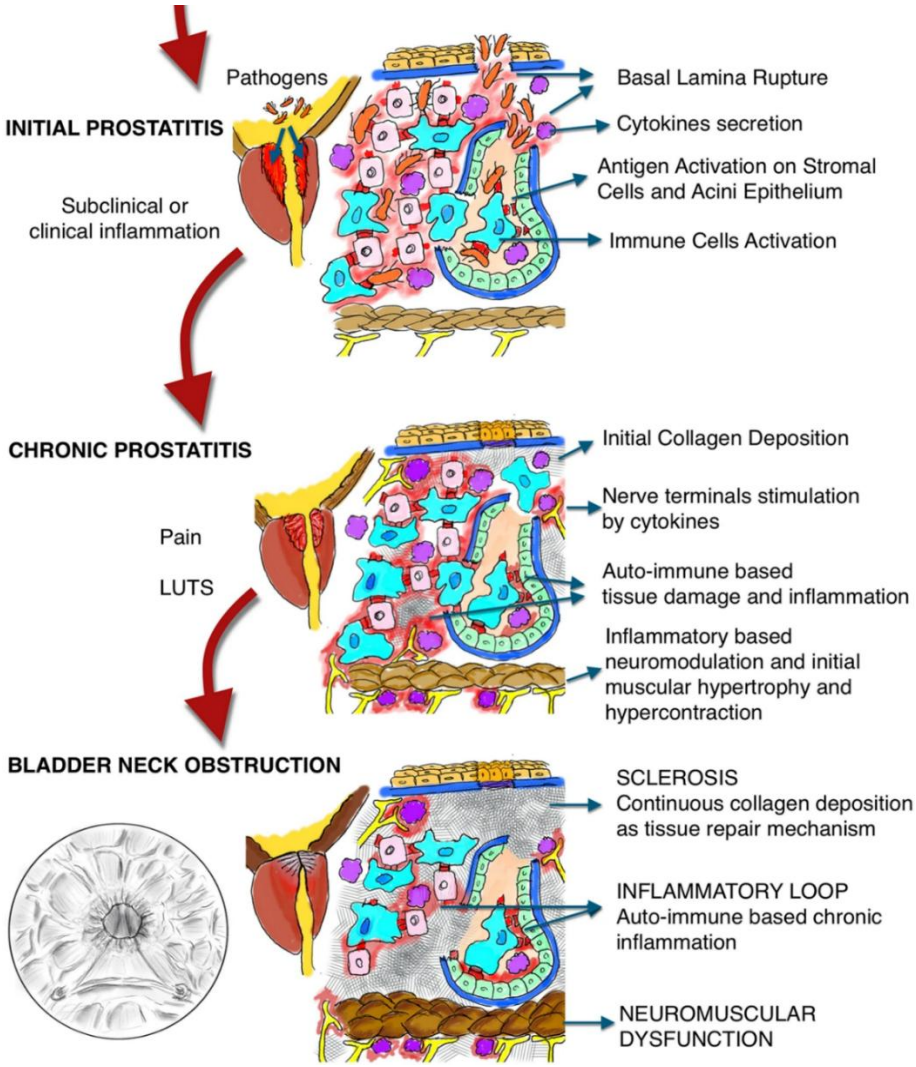

Figure n 4 From h CASH et al The hypotesized relationship between infection, inflammation and development of primary bladder neck obstruction PBNO is shown by the changes from the normal bladder neck, the initial inflammatory changes during prostatitis and the development of a chronic prostatitis. The continuous collagen deposition and underlying chronic inflammation CI lead to a bladder neck obstruction BNO.

T.C. Camerota :
"Urinary continence and micturition strongly depend on the integration of 2 different functional units of the lower urinary tract LUT : the bladder and the urethral sphincter complex (bladder neck and rabdosphincter). Coordination within the system is guaranteed by neural control at 3 levels: peripheral ganglia, spinal cord and brain.

This normal physiology of the system is extensively reviewed in the thesis. A crucial step in micturition is represented by the relaxation of the urethral sphincters US , which depends both on the disappearance of adrenergic and somatic excitatory inputs and on the activation of a parasympathetic reflex. This neuromuscular NM integrated system involves not only bladder neck and the urethral sphincters, but also the pelvic diaphragm, which is composed by a complex set of structures: the levator ani muscle, the coccygeal muscle CM and the surrounding connective tissue and fascias ."(4)

Letitia Wong et al

" Uropathogenic E. coli 1677 was instilled transurethrally into adult C3H/HeOuJ male mice to induce chronic prostatic inflammation CPI . Collagen was labeled by 3 H-proline administration for 28 days post-inoculation and 3 H-hydroxyproline incorporation measured to determine stability of the newly synthesized collagen. Inflammation score was graded using a previously established system and total collagen content TCC was measured by picrosirius red staining quantitation and hydroxyproline content. Resolution of inflammation and reversal of collagen deposition CD was assessed after treatment with antibiotic enrofloxacin for 2 weeks on day 28 post-inoculation followed by an 8-week recovery period.

Decay analysis of incorporated 3H-hydroxyproline revealed the half-life of newly synthesized collagen to be significantly shorter in infected/inflamed prostates than in controls.

Treatment with antibiotic enrofloxacin completely eradicated the bacterial infection and allowed resolution of inflammation.

This was followed by marked attenuation of collagen content and correlation analysis verified a positive association between the resolution of inflammation and the reversal of collagen deposition. Our findings show that inflammation-induced prostatic fibrosis IIPF is at least partly reversible and suggest that fibrosis of the human prostate may be addressed therapeutically by removing the cause of inflammation or suppressing the inflammatory response."(5)

A.M . Bujor et al
"In this study work , we demonstrated that the ciprofloxacin has dual antifibrotic effects on SSc dermal and lung fibroblasts by upregulating MMP1 and downregulating CCN2 and collagen type I levels."(6)

Scott A. LeMaire et al 2020
"ciprofloxacin treatment in WT Fbn1þ/þ mice reduced the level of LOX , a protein critical for cross-linking and stabilizing elastin and collagen."(7)

Enríquez-Casillas Rubén et al
"Scleroderma is an autoimmune connective AC tissue disorder that is characterized by microvascular injury, excessive fibrosis of the skin, and distinctive visceral changes that can involve the lungs, heart, kidneys and gastrointestinal tract GI . Our results suggest that oral administration OS of ciprofloxacin for 6 months reduces the severity of symptoms affecting the skin of patients with systemic scleroderma SS , and does so without important secondary effects."(8)

A. m Bujor et al

"Tendinopathies, including tendon ruptures, rare but severe complications of ciprofloxacin treatment, with the main risk factors RF being old age, systemic steroid therapy, strenuous physical activity. Although the pathologic mechanisms are poorly understood, various mechanisms have been proposed, like the upregulation of matrix metalloproteinases (MMPs) followed by type I collagen degradation, inhibition of cell proliferation by the downregulation of cyclin B and cyclin-dependent kinase 1 (Cdk1) and the inhibition of the tenocyte migration by the downregulation of focal adhesion kinase phosphorylation.

Ciprofloxacin-induced collagen downregulation DR in tendon cells is the result of increased ECM degradation due to the upregulation UR of various MMPs. Fibrosis in SSc is the result of the uncontrolled deposition of ECM that is presumably due to increased synthesis and to decreased degradation of matricellular components MC. Ciprofloxacin may affect SSc fibrosis by regulating the aberrant expression of MMPs, leading to increased collagen turnover. MMP1 is the only enzyme capable of initiating the breakdown of interstitial collagens IC , including collagen type I, and published data indicates that this enzyme is downregulated DR in SSc cells (33). Our study work demonstrates that, in addition to the effects on MMP1, ciprofloxacin treatment may directly block collagen synthesis in SSc fibroblasts, but not in healthy controls, by upregulating Fli1 levels."(9)

Experimental experience - case report
Patient of 60 year , decades of recidives in chronic prostatitis , uretral prostatic restriction , Psa negative
Diabete type II controlled , reduced urinary flux.

Many reacutization during the year .
Efficay therapy with ciprofloxacin 10 days (very effective to resolve the acute prostatitis for more then 6 months) and claritromicin. Three days of ciprofloxacin 500 m was not so effectile like 10 days to get a prolonged efficacy . (efficacy repeated in all reacutization)

Sovrainfection of candida : responce with spiromicin 7 days .

Used in the acute phase : steropids suppository for 10 days
Lower efficacy of oral fans to resolve the acute episode .
The attack start often with an episode of constipation , added with sedentariety .
Alchool or spicies agravate .

Discussion

It is clear the role played by the pelvic floor muscle in man : the SUS, the bulbocavernosus and the puborectal.

During the voiding bladder phases are crucial ther right alignment and coordination and fucntionality to make possible the full bladder emptyng.

Effective voiding, by contrast, requires: (a) sustained detrusor contraction, (b) complete and sustained relaxation of the urethral sphincters, (c) coordinated pelvic floor muscle relaxation, and (d) absence of mechanical obstruction

Voiding dysfunction VD is a broad term, used to describe the condition where there is poor coordination between the bladder muscle BM and the urethra. This results in incomplete relaxation or over-activity of the pelvic floor muscles during voiding

A misalignment or lack of coordination between the male urethra and pelvic floor muscles often indicates pelvic floor dysfunction or voiding dysfunction VD.

This causes the muscles to fail to relax properly during the urination, leading to a weak stream, hesitancy, and incomplete bladder emptying.

Potential Causes Pelvic Floor Hypertonia PFH : Muscles are too tight or in spasm, pulling the urethral sphincter out of alignment.Urethral Stricture: Scarring in the urethra alters the flow path and creates resistance.Neurological Conditions NC : Nerve issues disrupt the natural coordination between the brain, bladder, and pelvic muscles PM .Prior Trauma or Surgery: Scarring from pelvic injuries or prostate procedures can affect muscular alignment.

Pelvic floor dysfunction PFD occurs when these muscles are too tight, too weak, or poorly coordinated.
The pelvic floor muscles PFM , in particular, must fully relax and act in synchrony to permit descent of the bladder neck BN and opening of the urethra, allowing for efficient urine expulsion . In individuals with NR-PFD, this coordinated relaxation fails to occur and the pelvic floor muscles PFM remain contracted during voiding, effectively resulting in a functional bladder outlet obstruction (BOO)

The causes of pelvic floor dysfunction can vary. Often, multiple contributing factors are at play. Some of the most common include:
Post-surgical trauma PST (such as prostatectomy or vasectomy),Orthopedic injuries (spine, hip, or pelvic injuries) Excessive exercise or poor lifting mechanics,Chronic constipation CC or habitual straining

Long hours of sitting or a sedentary lifestyle,Psychological stress, anxiety, or trauma

Practices like jelqing or attempts at genital enhancement"

Bladder neck stricture BNS is a condition in which this opening becomes narrowed, restricting the flow of urine from the bladder. This narrowing can interfere with normal bladder emptying and may lead to urinary symptoms that require medical intervention to avoid patient complications. " Constipation is when the stools become hard and cannot pass through the body. These hard stools put pressure on the bladder neck BN , making it difficult for the muscles to work and drain the urine to the urethra."

Primary bladder neck obstruction (PBNO) is a dysfunction of the bladder neck (BN) in which the collum vesicae is narrow or fails to open adequately during voiding, resulting in bladder outlet obstruction.

This condition can be or of inflamatory origin or non inflamatory.

The structural changes at the BN causing PBNO have been described as "fibrous narrowing and hyperplasia" "fibrous stroma and subepithelial scar, often with inflammatory change" (3)

Abnormal morphologic arrangement of the bladder musculature (detrusor/trigonal) (3)

A structurally healthy but dysfunctional BN have also been described. (3)

Several theories included altered mechanisms of BN opening such as neurological dysfunction of the sympathetic nervous system SNS with a detrusor-sphincter-dyssynergia , abnormal BN striated muscle components of the urethral sphincter , increase in the density of Neuropeptide Y immunoreactive nerves (supporting the hypothesis of the PBNO as a more functional illness linked to pelvic floor dyssynergia) (3)

Hinata described a correlation between increased prostate volume due to BPH and an increase of collagen fibers and degeneration of muscle bundles in the BN. By progression of prostatic hyperplasia, the BN muscles were progressively affected by fibrosis with fragmentation of the smooth muscle sphincter vesicae

Also it is reported in literature the possibility of the induced fibrosys by prostatic chronic bacteria infection CBI and the flogotic reaction involved.

PBNO can be also a consequence of previous prostatic inflammations.

The same the reverse of this process is proved with the resolution of the infection and relted inflmatory state (5) Instead the data on the non-inflammatory possible causes of PBNO, are scarse and weak.

Conclusion

Pelvic floor physical therapy PFPT , biofeed back muscle relaxant can be efficacy in this situation.

Adjunctive options, including trigger point injections, botulinum toxin, and sacral neuromodulation, can benefit patients with refractory symptoms. Cognitive behavioral therapy and integrative modalities are also increasingly utilized." (3)

PBNO there is a persistence of the cranial part of the skeletal muscle cells of the urethral sphincter, which may interfere in the complex micturition process, and that may explain why those patients did not respond to alpha-blockers, typically working on smooth muscle cells.

The natural HIF-1α inhibitors ascorbate and curcumin were capable to attenuate prostate enlargement both in vivo and in vitro.

In every way the case report submiteed confirm the study of WONG about the efficacy of specific antibiotic therapy for about 10 days to reverse the acute attack promoting a better urinary flux.

About the Inflammation-induced conditions : prostatic fibrosis is at least partly reversible and suggest that fibrosis of the human prostate may be addressed therapeutically by removing the cause of inflammation or suppressing the inflammatory response.

A crucial step in micturition is represented by the relaxation of the urethral sphincters US , which depends both on the disappearance of adrenergic and somatic excitatory inputs and on the activation of a parasympathetic reflex PR . This neuromuscular NM integrated system involves not only bladder neck and the urethral sphincters, but also the pelvic diaphragm, which is composed by a complex set of structures: the levator ani muscle, the coccygeal muscle and the surrounding connective tissue and fascias ."(4)

Aa a final consideration_ the vicious circle in this condition can aggarvate the situation: it is really interesting fo the authors the Research reported by Wong et al about the possibility to reverse the connective tissue deposition and the fibrotic process even if proved in an experimental settings.

Conflict of interest : no

References

1. Int Neurourol J. 2012 Sep 30;16(3):102–106. doi: 10.5213/inj.2012.16.3.102 Clinical and Functional Anatomy of the Urethral Sphincter Junyang Jung , Hyo Kwang Ahn , Youngbuhm Huh
2. Curr Urol Rep. 2025 Oct 16;26(1):66. doi: 10.1007/s11934-025-01290-4 Urologic Manifestations of Nonrelaxing Pelvic Floor Dysfunction: Insights on Clinical Workup and Management Andrew S Afyouni , Narmina Khanmammadova , Alireza Bozorgi , Akhil K Das , Joel Gelman , Zhina Sadeghi
3. Perspective Clinical Research Open access 08 July 2023 Primary bladder neck obstruction in men—new perspectives in physiopathology Hannes Cash, Johann Jakob Wendler, Antonio Minore, Ioannis Kartalas Goumas & Luca Cindolo Prostate Cancer and Prostatic Diseases volume 27, pages 54–57 (2024)
4. primary bladder neck obstruction: a new etiopathogenesis. T.C. Camerota 2018 IRIS univ. Studi Milano The Prostate (2015) Resolution of Chronic Bacterial-Induced Prostatic Inflammation Reverses Established Fibrosis Letitia Wong, Paul R. Hutson, and Wade Bushman
5. Int J Mol Med. 2012 Oct 5;30(6):1473–1480. doi: 10.3892/ijmm.2012.1150 Ciprofloxacin has antifibrotic effects in scleroderma fibroblasts via downregulation of Dnmt1 and upregulation of Fli1 Andreea M Bujor 1, Paul Haines , Cristina Padilla , Romy B C hristmann , Monica Junie, Percival d Sampaio-Barros , Robert Lafyatis , Maria Trojanowska
6. The Journal of Thoracic and Cardiovascular Surgery c Volume 163, Number 3 Ciprofloxacin accelerates aortic enlargement and promotes dissection and rupture in Marfan mice Scott A. LeMaire, Lin Zhang, MS, Nicholas S. Zhang,a Wei Luo, James P. Barrish, Qianzi Zhang,e Joseph S. Coselli, and Ying H. Shen, 2020
7. J Dermatol. 2010 Apr;37(4):323-9. Ciprofloxacin utility as antifibrotic in the skin of patients with scleroderma Enríquez-Casillas Rubén , Vázquez-Rodríguez Manuel, Ochoa-Ramírez Agustín, Miguel Huerta, Fraga-Mouret Antonio, Delgado-Enciso Iván DOI: 10.1111/j.1346-8138.2010.00826.x
8. Int J Mol Med. 2012 Oct 5;30(6):1473–1480. doi: 10.3892/ijmm.2012.1150 Ciprofloxacin has antifibrotic effects in scleroderma fibroblasts via downregulation of Dnmt1 and upregulation of Fli1 Andreea m Bujor , Paul haines , Cristina Padilla , Romy b Christmann , Monica junie , Percival d Sampaio-barros , Robert lafyatis , Maria Trojanowska