

Sexual abuse evaluation in urological practice Beck, J.J.H.

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PART IV:

CONCLUSIONS

Chapter 8:

Sexual abuse and over active bladder: adding the pelvic floor pathway to the sexual abuse - overactive bladder - model

Based on:

Beck JJH, Nicolai MP, Voorham- Zalm P, Pelger RCM, Elzevier HW *Sexual abuse and over active bladder: adding the pelvic floor pathway to the sexual abuse - over active bladder - model.* Submitted

Abstract

Introduction: We review evidence linking pelvic floor dysfunction (PFD) to the current concept of sexual abuse (SA), overactive bladder (OAB) and corticotrophin releasing factor (CRF).

Methods: We review the literature and add the pelvic floor pathway to the current Klausner-Steers model for emotional influence on the bladder.

Results: CRF is expressed in areas of the central nervous system that response to stress and is increased during anxiety and after SA. CRF is expressed in areas of the central nerve system that control voiding and response to stress. Epidemiological and case control studies reveal an association between SA and PFD. PFD is related to long-lasting bladder outlet obstruction (BOO), which can lead to OAB.

Conclusions: PFD after SA is another link between the relation of SA and OAB. Besides CRF and OAB as a therapeutic target, maybe pelvic floor physiotherapy can improve OAB after SA. We add the pelvic floor pathway to the current Klausner-Steers model for emotional influence on the bladder.

Introduction

The Pelvic Floor and Sexuality Research Group at the department of urology at Leiden University Medical Center, the Netherlands, investigates pelvic floor function and dysfunction in relation to urological, sexual and pelvic floor related complaints. We review evidence linking pelvic floor dysfunction (PFD) to the current model of sexual abuse (SA), overactive bladder (OAB) and corticotrophin releasing factor (CRF). Sexual abuse (SA) is defined by International Society for the Prevention of Child Abuse and Neglect as "a social and medical problem in which a child under the age of consent is involved in an act resulting in sexual satisfaction of an adult or connivance of such an act''^1 . The frequency with which children are exposed to sexual advances from adults varies according to the definition of abuse, the age range studied, and the methods of ascertainment. The prevalence of SA is estimated to be 12% to 25% for females and 8% to 10% males². A metaanalysis shows that a history of SA is associated with lifetime diagnosis of multiple disorders, like seizures, gastrointestinal problems and non-specific chronic pelvic pain³. Another recent metaanalysis demonstrates that SA is associated with multiple psychiatric problems, including lifetime diagnosis of anxiety disorders, depression, eating disorders, post-traumatic stress disorder (PTSD), sleep disorders, and attempted suicide⁴. The reported prevalence of SA-history in the urological population varies from 2% for males to 13% for females⁵. Selection bias and recall bias are mentioned; therefore more studies are necessary to measure the exact prevalence of SA in urologic patient populations. In 2004 a biological model explaining the emotional influence on the bladder through the CRF-OAB-pathway was presented by Klausner and Steers⁶. Their model is shown in Figure 1. It was until 2007 that SA was causally related to OAB, using the Hill-criteria^{7,8}. We add the role of PFD to the current available Klausner-Steers model (Figure 2). OAB is defined as: urinary urgency, usually accompanied by frequency and nocturia, with or without urgency urinary incontinence, in the absence of urinary tract infection (UTI) or other obvious pathology 9 .

Klausner-Steers model⁶

Stress and anxiety are intimately associated with bladder function. The extensive neural networking, multilevel inputs and reciprocal innervation involved in the control of bladder function suggest a multifactorial etiology⁶. CRF, a peptide synthesized in neurons of the paraventricular nucleus (PVN) of the hypothalamus, has been found to be a key regulator of the endocrine, behavioral, autonomic and immune responses to stress⁶. After its release from the hypothalamus, CRF triggers the release of adrenocorticotropic hormone (ACTH) in the pituitary. ACTH, in turn, stimulates the release of the stress steroid, cortisol, from the adrenal cortex. In this manner CRF acts as a hormone, triggering the peripheral stress response also known as the "hypothalamic-pituitary-adrenal (HPA) axis". CRF also acts directly in the central nervous system as a neurotransmitter. Neurons in the brain expressing CRF project to critical areas that control the central stress response. These areas include the locus ceruleus - an area containing noradrenergic neurons and important in the regulation of the

flight or fight response, the dorsal raphe nucleus - an area rich in serotonin and potentially important in the pathogenesis of major depression, the central nucleus of the amygdale - a relay center for emotional stress and visceral pain, and the hippocampus - a region in memory processing⁶. In mammalian species CRF binds to 2 cyclic adenosine monophosphate coupled receptors, CRF-R1 and CRF-R2. CRF-R1 is prominent in the limbic regions, and CRF-R2 is more widely distributed in the brain and spinal cord. CRF appears to be the endogenous ligand for CRF-R1 as it has 10-fold greater binding affinity for this receptor than for CRF-R2. Acute and chronic stress produces significant increases in CRF mRNA in Barrington's nucleus, the region that controls micturition. Moreover, electrical stimulation of these areas of the brain excites or inhibits bladder activity. CRF also is important in the control of micturition at the level of the spinal cord and may be important in sensory processing of painful bladder stimuli. Patients with interstitial cystitis (IC), an idiopathic disorder characterized by urinary frequency, urgency and pain, also manifest HPA dysregulation. Patients with IC display symptom exacerbation when exposed to stress. Specific data regarding the role of CRF directly on micturition are conflicting and incomplete. However, due to substantial cross-innervation of the colon and bladder, CRF, mediated through CRF-R1, would be expected to stimulate the bladder similar to its excitatory action on the colon⁶.

The pelvic floor pathway

We hypothise that pelvic floor dysfunction (PFD) is another link between sexual abuse history and OAB. The pelvic floor is known to be an integrated structure, influenced by psychological and physical causes. A higher prevalence of multiple pelvic floor complaints, like micturition, defecation and sexual pain, are seen in patients with sexual abuse history¹⁰. Davila et al reported significant more pelvic floor related urological complaints like dribbling, slow urinating stream and stress incontinence after SA^{11} . The pelvic floor comprises several layers, including the pelvic diaphragm (levator ani and coccygeus muscles) and the urogenital diaphragm. Each diaphragm has its own 3D shape and position with regard to the internal pelvic organs. The urogenital diaphragm consists of a deep laver, the perineal membrane, and a superficial laver, consisting of the bulbospongiosus muscle and the ischiocavernosus muscle. The levator ani muscle is made up of the iliococcygeus, pubococcygeus, and puborectalis muscles. Together with the urethral and anal sphincters, these muscles play an important role in preventing complaints of micturition, defecation, sexual dysfunction, prolapse and/or pelvic floor pain. The development of one of these complaints is referred to as PFD¹². It has been hypothesized that patients with PFD have voiding difficulties due to a higher tone at rest of the pelvic floor^{9;13,14}. Many of them have episodes of obstructive urinating complaints. As in benign prostate hyperplasia, long-lasting bladder outlet obstruction (BOO) can lead to OAB symptoms¹⁵. Obstruction-induced changes in the bladder are of two basic types. First, the changes that lead to detrusor instability or decreased compliance are clinically associated with symptoms of frequency and urgency. Second, the changes associated with decreased detrusor contractility are associated with further deterioration in the force of the urinary stream, hesitancy, intermittency, increased residual urine, and (in a minority of cases) detrusor failure¹⁶. Pelvic floor physiotherapy can be used to treat pelvic floor related BOO and thus relieving OAB symptoms¹⁷. Unfortunately randomised studies describing improvement of urological complaints in SA survivors treated with pelvic floor overactivity) resulting in BOO, resulting in voiding symptoms and later on in torage symptoms (OAB). In women with interstitial cystitis/painful bladder syndrome (IC/PBS) two randomized clinical trials showed that myofascial physical therapy significantly improved patients bladder complaints compared to treatment with global therapeutic massage, indicating a role of the pelvic floor in bladder complaints^{18,19}. We postulate an extra pathway to the existing Klausner-Steers model by adding the pelvic floor pathway. An overactive pelvic floor leads to BOO, which when long-lasting leads to OAB, just as in OAB after BPH. The biological influence of stress and abuse on the pelvic floor is not cleared out yet, but epidemiological and clinical studies point out in that direction. Figure 2.

Conclusion

PFD after SA is another link between the relation of SA and OAB. Besides CRF and OAB as a therapeutic target, maybe pelvic floor physiotherapy can improve OAB after SA. We add the pelvic floor pathway to the current Klausner-Steers model for emotional influence on the bladder.

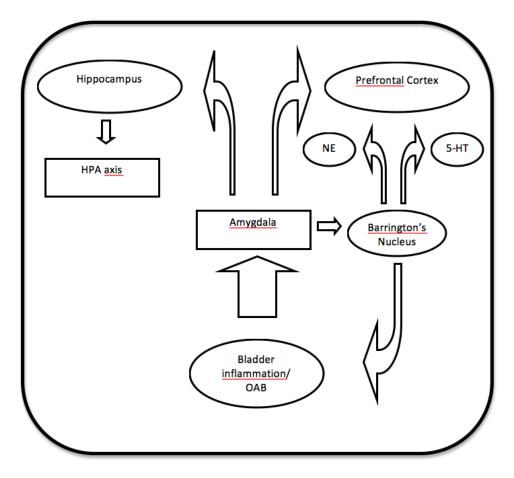


Figure 1: The Klausner-Steers model for emotional influence on the bladder

Central stress response. In this model bladder inflammation sends nociceptive input to central nervous system via afferent nerves. Nociceptive stimuli are processed in amygdala which acts as relay station for processing emotional stress and visceral pain. Signals are relayed to hippocampus where memory processing occurs and can trigger peripheral stress response via HPA axis. Signals also project to prefrontal cortex for higher order processing and directly to Barrington's nucleus, projections are sent to locus ceruleus, which is rich norepinephrine (NE), and dorsal raphe nucleus, which is rich in serotonin (5-HT) and directly to lumbosacral spinal cord where descending inputs can influence afferent processing or motor output to bladder neurons.

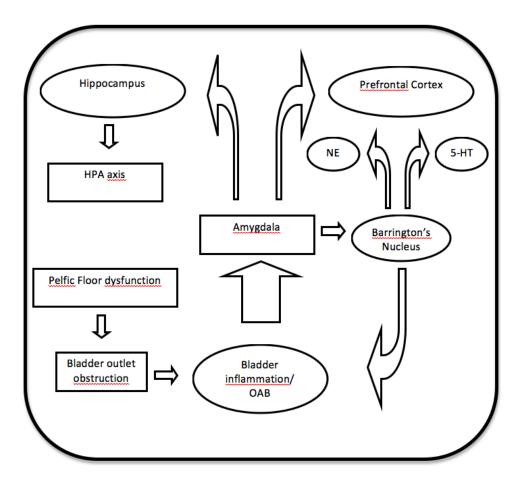


Figure 2: Adding the pelvic floor dysfunction pathway to the Klausner-Steers model

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