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Interventional Treatments for Bladder Pain Syndrome

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Abstract

Patients with BPS persistent and unacceptable symptoms despite oral and/or intravesical therapy are candidates for more aggressive modalities. Many of these are best administered within the context of a clinical trial if possible. These may include: neuromodulation, intradetrusor botulinum toxin, oral cyclosporine and other anesthesic techniques. The last step in treatment is usually some type of surgical intervention aimed at increasing the functional capacities of the bladder or diverting the urinary stream. In this paper a review of interventional treatment´s clinical evidence is made and shows how to improve symptoms in refractory BPS.

Keywords: Bladder painful syndrome; Chronic bladder pain; Interstitial cystitis

Introduction

Although underlying pathophysiology of painful bladder syndrome/interstitial cystitis (PBS/IC) is not completely understood, it involves urothelial permeability changes primarily, along with mast cell activation and neurogenic inflammation [1]. In PBS/IC condition, damage to the protective bladder lining leads to impaired urothelial cell barrier function. Consequently, urinary solutes penetrate the epithelium and activate sensory nerve endings, leading to the manifestation of inflammation and pain [2]. Consistent with this theory, bladder epithelial cells in PBS/IC patients are shown to produce anti-proliferative factor (APF) [3], which may further contribute to the impaired urothelial cell barrier. Moreover, urothelial cells in PBS/IC patients fail to release prostaglandin E2 (PGE2), which is crucial for the protection and repair of the urothelium [4]. Other bladder epithelial abnormalities reported in PBS/IC include abnormal cellular architecture as revealed by electron microscopy [5] and abnormal uroplakin expression as assessed by reverse transcriptase PCR [6]. Mast cell may play a central role in PBS/IC pathophysiology: patients have increased number mast cell along with higher percentage (70%) of activated mast cells versus 10% in healthy controls [7]. Moreover, compounds that are indicative of mast cell activation such as Interleukin 6 (IL-6), histamine, and tryptase are increased in the urine of PBS/IC patients [8]. Interestingly, Tamm-Horsfall protein concentration in the urine of PBS/IC patients may not differ from healthy controls, but it is qualitatively different containing less sialic acid [9]; this altered protein may thus be involved in PBS/IC pathogenesis.

Neurogenic upregulation may also play a role in the pathogenesis of PBS/IC. The purinergic pathway has been shown to be upregulated in urothelial cells from PBS/IC patients [10], with peripheral and central neural upregulation [11]. However, whether the neurogenic inflammation that characterizes PBS/IC is the cause or the result of other previous events is yet unresolved.

The condition of PBS/IC could result from different environmental triggers in a genetically susceptible individual [12]; this approach may explain its increased prevalence among first-degree relatives and monozygotic twins. In this context, PBS/IC could be considered a clinical phenomenon in a genetically susceptible individual, where an environmental trigger such as trauma or infection could promote genetic events leading to an inflammatory response [13].

Treatment

First line of treatments applicable to all the affected patients includes awareness, education, self-care, and stress and pain management. Most patients may require additional therapy and oral medications or bladder instillations or pelvic floor physical therapy all considered second line options. Bladder instillations represent interventional treatments which are more commonly applied in combination therapy. Manual pelvic-floor physical therapy actually has the strongest evidence for efficacy, although availability and cost can present barriers to patients. Should these fail to yield the desired therapeutic effect, more invasive interventions, such as cystoscopy with hydro distention or sacral nerve stimulation may be opted. The use of cyclosporine or bladder injections of botulinum toxin is also alternative options available for select refractory patients. It should be noted that, apart from.

Dimethyl Sulfoxide (DMSO)

Mechanisms involving DMSO facilitates dissolution of collagen and degranulation of mast cells and it helps to reduce inflammation relax muscles and mitigate pain. Only one randomized study, reported by

been reported in case series and retrospective studies. With regard to side

effects after instillation of DMSO, most patients sense a garlic-like

odor, which disappears within a day, and about 10% of patients reported bladder irritation symptoms which resolve with or without symptomatic treatment. It is hypothesized that these transient exacerbations occur as the result of mast cell degranulation. T e number of side

Conclusion

BPS initial treatment includes patient education, dietary manipulation, nonprescription analgesics and stress reduction. When conservative therapy fails or symptoms are severe and conservative

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