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Malaguti S[1], Mako A[2], Natale F[2], Lamarche J[2], Cervigni M[2]
1. *neurourology spinal unit niguarda hospital milan,Urogynecology Dept., San Carlo-IDI Hosp., Rome, Italy. italy,* ***2.*** *Urogynecology Dept., San Carlo-IDI Hosp., Rome, Italy.*

DEFECTIVE SPINAL MODULATION OF NOCICEPTIVE PROCESSING IN PATIENTS WITH PAINFUL BLADDER SYNDROMEINTERSTITIAL CYSTITIS (PBS-IC) MAY PLAY AN ESSENTIAL ROLE IN THE PATHOPHYSIOLOGY OF THE DISEASE.

Hypothesis / aims of study

Painful Bladder Syndrome-Interstitial cystitis (PBS-IC) is the complaint of suprapubic pain related to bladder filling, accompanied by other symptoms, such as increased daytime and night-time frequency, in the absence of proven urinary infection or other obvious pathology.

Sensation depends on neurophysiologic mechanisms in several different nerves, receptors, and transmitters. Different stimuli can elicit sensations in the lower urinary tract such as bladder filling, micturition, noxious stimuli, and external stimuli.
In chronic inflammatory bladder diseases (e.g., in PBS-IC), hypersensitivity to both visceral and somatic stimuli due to hyper excitability of C-fiber afferent pathways, which are silent in the normal state during bladder filling, has been proposed as mechanism for bladder pain and urgency.

Visceral hypersensitivity can be influenced by peripheral and central mechanisms affecting pain perception. Studies using electrical stimulation and the nociceptive flexion RIII lower limb reflex have further confirmed enhanced visceral perception in other chronic pain disease (e.g. Irritable Bowel Syndrome).

Abnormal central pain processing may play an important role in the initiation and maintenance PBS/IC suggesting a central sensitization of pain pathways.

The aim of this study was to evaluate the function of pain modulating systems sub serving diffuse noxious inhibitory controls (DNICs) in PBS/IC.

Study design, materials and methods

We investigated the spinal transmission of nociceptive signals and the DNICs in 14 PBS-IC patients (12 women, 2 men; mean age 39,8 yrs) and in 10 healthy subjects (matched for sex and age) by means of nociceptive flexion RIII reflex and the Cold Pressor Test (CPT) as heterotopic noxious conditioning stimulation (HNCS).

Results

The subjective pain thresholds (Tp) and the RIII reflex threshold (Tr) were significantly lower in PBS/IC vs. controls. In controls a significant inhibition of the RIII reflex was observed during the CPT (-39%,$P<0.05$). On the contrary, PBS/IC patients showed facilitation (+35%,$P<0.05$) of the RIII reflex during CPT.

Interpretation of results

Our results give direct evidence that a hyper excitability of spinal nociceptive processes is present in PBS/IC patients and demonstrate a dysfunction in systems sub serving DNICs in PBS/IC patients. Impairment of endogenous supraspinal pain modulation systems may contribute to the development and/or maintenance of central sensitization in PBS/IC.

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Was this study approved by an ethics committee?	**No**
This study did not require eithics committee approval because	**diagnostic protocol**
Was the Declaration of Helsinki followed?	**Yes**
Was informed consent obtained from the patients?	**Yes**