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## GIBS News Letter



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# UNDERSTANDING **SYMPTOMS OF** IC/BPS

Interstitial cystitis/bladder pain syndrome (IC/BPS) is a chronic debilitating condition of unknown aetiology. It is characterized by persistent pelvic pain with lower urinary tract symptoms. The pathogenesis, as to which mechanisms perpetuate inflammation, remain unknown. It is potentially associated with urothelial malfunction and neurophysiological dysfunction. IC/BPS frequently presents with somatic and/or psychological symptoms, that commonly result in central nervous sensitisation. Typically, the condition is characterized by persistent pain perceived to be related to the urinary bladder in conjunction with urinary frequency and/or urgency. Obstructive symptoms such as slow stream, dribbling and straining are also often reported by painful bladder syndrome and interstitial cystitis (PBS/IC) patients.

#### **SUBTYPES**

IC/BPS can be categorized into two subtypes at present: (i) IC/BPS with Hunner lesions, which is also known as the ESSIC BPS type 3; and (ii) IC/BPS without Hunner lesions, which includes the ESSIC BPS type 1 and 2. These two subtypes, which are distinguished simply by the cystoscopic presence or absence of Hunner lesions, present with different, but overlapping, clinical characteristics and cannot be clinically differentiated in the absence of cystoscopic findings.

## ONSET OF SYMPTOMS AND DISEASE EVOLUTION

IC/BPS presentation can vary from being a relatively mild disease to a severe progressive disease ending up in a small shrunken bladder. Some patients of IC/BPS have rapid onset of their symptoms and fast clinical progression. This suggests that IC/BPS comprises a heterogenous group of triggering and perpetuating factors with different diseases merging into one common clinical pathway.

IC/BPS with Hunner lesions is characterized by an older age at diagnosis, more severe bladder-centric symptoms, reduced bladder capacity, fewer comorbid non-bladder conditions and more favourable outcomes on endoscopic treatment compared with IC/BPS without Hunner lesions. IC/BPS without Hunner lesions is frequently accompanied by non-bladder symptoms, including other common systemic pain problems ("bladder-beyond" pain), psychosocial health problems and affect dysregulation. Recent studies have shown that these clinical characteristics of IC/BPS without Hunner lesions strongly overlap with those of widely known somatoform disorders or functional somatic syndromes (FSSs), such as irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome and migraines.

#### SYMPTOMS OF IC/BPS URGENCY

Urgency is defined as "the complaint of sudden compelling desire to pass urine, which is difficult to defer". In IC/BPS urgency is primarily reported because of pain, pressure, or discomfort. This is different from overactive bladder (OAB) where urgency is there for a fear of leaking urine. Bladder pain is rare in patients of OAB. However, it is important to remember that both conditions can co-exist in a patient. 40% of women with OAB also report urgency because of pain, pressure, or discomfort.

#### PAIN

Urothelial denudation is a characteristic histological feature of IC/BPS with Hunnerlesions. Specifically, full layers of the urothelium are frequently sloughed off at Hunnerlesion sites. This entire loss ofthe urothelial barrier at lesion sites could permit urinary stimuli to directly come into contactwith afferent peripheral nerves in the bladder. This could be one possible explanation for thehypersensitive bladder symptoms; that is, occasionally susceptible to the change in urinecomposition in patients with IC/BPS, as consumption of specific diets exacerbate thesymptoms. Pain is described as located in suprapubic, urethral, vaginal, perineal and low back regions. In a case-control study of IC/BPS, 92% patients reported

perceiving one or more bladder or LUTS (lower urinary tract symptoms). 41% noted a bladder location of pain, 34% noted pain on bladder filling and /or decreasing with voiding, 17% mentioned other urinary tract symptoms and 8% noted only non-urinary symptoms.

Another study looked at whether patients of IC/BPS also demonstrate characteristics of visceral pain syndromes. The authors observed that IC/BPS patients also demonstrate discomfort at other sites besides the suprapubic region.

#### **VOIDING DYSFUNCTION**

Obstructive symptoms such as slow stream, dribbling and straining are often reported by IC/BPS patients. Cameron and Gajewski studied 274 patients who met the National Institute of Diabetes, Digestive and Kidney Disease (NIDDK) research definition of IC. Those who had completed pressure-flow urodynamic studies (UDPF), a urinary symptom score and had a cystoscopy with hydrodistension, were included. All patients had both cystometry and pressure-flow studies. The cut-off values of maximum flow rate (Qmax) 12 ml/sec and detrusor pressure at maximum flow (PdetQmax) 25 cm H2O were used to define BOO in these women. They observed 48.1% of their patients with IC/BPS have symptoms of bladder outlet obstruction (BOO). Detrusor Overactivity (DO) can be seen in upto 15% of patients with IC as demonstrated by the IC database study group. The exact mechanism of this is not well understood, but there is a possibility of stimulation of afferent nerves and detrusor muscle by both neuronal and non-neuronal-sensorimotor factors acting on bladder urothelium.

#### BPSASA SOMATOFORM DISORDER

Growing evidence suggests a potential connection between IC/BPS without Hunner lesions and somatoform disorders. Somatic symptoms could be linked to biological pathways that increase the risk of IC/BPS without Hunner lesions. A study that carried out MRI of the brain of patients with UCPPS and fibromyalgia, and pain-free controls showed similar abnormal brain activity in patients with UCPPS and fibromyalgia.

The relationship between specific dietary intake and symptom changes is commonly seen in IC/BPS and FSSs, in which some dietary metabolites might act as excitatory neurotransmitters, resulting in the central nervous sensitiSation. These findings suggest that IC/BPS without Hunner lesions might share its pathogenetic neurophysiological process affecting the CNS with somatoform disorders or FSSs. The underlying pathophysiology of FSSs remains unclear, but aberrant neuroimmune or endocrine processes with

certain stressors might be responsible for central nervous sensitization and systemic hypersensitivity.

#### **SUMMARY**

IC/BPS refers to a symptom syndrome complex characterized by persistent pain perceived to be related to the urinary bladder in conjunction with urinary frequency and/or urgency, and maybe associated with voiding dysfunctions. There are certain critical differences between IC/BPS with and without Hunner lesions. IC/BPS with Hunner lesions is an inflammatory disease of the urinary bladder potentially associated with enhanced immune responses and infection, whereas IC/BPS without Hunner lesions is a non-inflammatory disorder with little evidence of

bladder etiology. Categorization of IC/BPS based on cystoscopic (and histological) examination at initial diagnosis determine each subtype may change the management. For example, local fulguration and steroid injections, intravesical instillation of DMSO, and cyclosporine A administration are likely to improve patients with Hunner lesions. In contrast, neuromodulation therapy and/or a multidisciplinary treatment management of related somatoform disorders are more likely to be effective forpatients with IC/BPS without Hunner lesions. Tailored approach in this manner and could lead to better outcomes in clinical management and future research of IC/BPS.

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