UTI & Overactive Bladder Application Guide

Clinical Context

Polyuria can appear as part of developing diabetes or other problems. In this Application Guide, I'll focus on undiagnosed UTI as a common driver of overactive bladder syndrome (OAB). This is often seen in functional medicine practice but may be underappreciated. For a useful discussion of causes of OAB related to surgical history or frank disease, consider this paper: Overactive Bladder Syndrome: Evaluation and Management. Leron E, Weintraub AY, Mastrolia SA, Schwarzman, P. Curr Urol. 2018 Mar; 11(3): 117–125. doi: 10.1159/000447205

Patients with OAB in a functional medicine setting often have an underlying bladder wall inflammation. This may be from a previous infection, from an oxalate sensitivity, or another cause. Whatever the cause of the inflammation, chronic bladder wall inflammation is likely to stimulate nerve endings in the bladder wall to fire. If stretch receptors fire signals into the brain, the signal will be interpreted in the brain as stretch, even if irritation, not stretch, was the stimulus that got the stretch receptor attached to that nerve to fire. This is the same effect as when you rub your eyes and see colors. The nerve endings are being stimulated by a stimulus other than the one associated with that nerve. When the patient feels sensations that give them the feeling that the bladder wall is stretched (full), they will have the urge to void urine.

Now, assuming the bladder is not in fact full of urine (as it would be in developing diabetes for example), the question is, "What is making the bladder wall inflamed?"

Active Urinary Tract Infection

It is common for the patient to have a chronic low-level UTI. This is extremely common in elderly people and is often missed, particularly if the patient's capacity for communication is impaired, or if their capacity to discern sensory signals is impaired. With infection, it's common that bacteria will form a biofilm that makes them adherent to the bladder wall. The first problem with a biofilm is that it makes the bacterial infection difficult to eradicate, as organisms deep in the biofilm go dormant, so they don't eat the antibiotic. When the antibiotic course is over, with enough time and random re-activation, the surviving organisms gradually regrow the bacterial colony. The colony will still be susceptible to the same antibiotic or natural agents, since the surviving organisms will not have eaten the previous round of antibiotic, so they won't have formed resistance. That means that when a patient tells you the same antibiotic always helps them get over their UTI, you should suspect a biofilm.

From the perspective of OAB, the concern is that infection is inflammatory. And with a biofilm, the concern is that the organisms in contact with the wall of the bladder, at the base layer of the biofilm, instead of going dormant, will irritate the bladder wall and when an inflammatory exudate forms, the organisms will eat the exudate in order to survive. So, the infectious agents are instigating inflammation of the bladder wall directly.

If there is an active infection, the first step is to address the infection. It's also important to address the biofilm component of the infection. For a discussion of how this is accomplished, review Module 15 Videos 9-12 and Module 16 Videos 8 and 13. M16V13 also contains a chart of infectious agents, biofilm disruptors, and antibiotics that have been used in combination. Thyme, stevia, NAC, ginger, oregano, quercetin, berberine, artemesia, garlic, and a host of other agents have biofilm disrupting properties. Look at the chart at the end of Module 16, Video 13. Consider the biofilm disruptors there, or use others if you have experience with them. Other natural agents often observed to be useful include the following:

Cranberry/d-Mannose (Pure Encapsulations) – 2 BID. To reduce adherence of pathogens to the bladder wall.

Buchu – 200mg BID or more as needed. To reduce pathogens and enhance urine flow.

One might imagine that it would not be useful to repeat the use of an agent that didn't fully eradicate a UTI on previous attempts. However, the opposite is often the case. The fact that an agent or combination of agents knocked down the bacterial colony count suggests that it was at least partly effective. The task is to determine if using that same approach, combined with biofilm disruptors, is more effective. A biofilm disruptor will degrade the structural integrity of the biofilm, increasing penetrance of the agent(s) you're using to kill the pathogen.

It's noteworthy that biofilm-based infections are typically characterized by strong adherence to the bladder wall and a highly organized biofilm matrix in which the pathogenic organisms are embedded. This mean that the patient may have suprapubic pain and burning upon urination, but a negative urine culture, since the organisms involved in the infection are not coming down into the urine. It is occasionally useful to give a biofilm disruptor before doing a urinalysis, but often the UA is nonetheless negative. Treating the patient for a UTI, despite the negative UA, is often useful.

It's also important to understand that that some organisms are "viable but not culturable." Biofilm researchers refer to these as VBNC's. William Costerton, the father of biofilm research, used to describe scraping infectious exudate out of diabetic foot wounds and trying to culture it, often unsuccessfully. Successful culture depends on finding the correct culture medium for growing a particular organism. Standard culture media often won't grow what's growing in the patient.

Biofilm with Residual Organisms

It's also possible that the patient has a low level of chronic inflammation, driven by a biofilm-based infection in which a small enough number of pathogenic organisms is present, embedded in a biofilm. This might not be considered an infection if the pathogen counts are not high enough. For example, a patient who was treated with an antibiotic several months ago for a UTI may now be in a position in which the pathogen count is very low but rising slowly. You can often see this kind of gradual reemergence in other infections like sinus infections or diverticulitis. As with the bladder, the key point is that the same antibiotic will work to knock back the infection each time, as discussed above. Treatment can be similar to that for a UTI, with a combination of substances to address the pathogenic organisms, coupled with biofilm disruptors.

Other Factors

As is true of many disease processes, it's necessary to inventory the patient as a whole to understand other contributors to the bladder wall inflammation. The patient may have problems with histamine elevation, driving bladder wall mast cell degranulation. The patient may have an oxalate issue. Or they may have a pattern of pain sensitization. If a painful neuropathy is part of the picture, it becomes important to use perilla to inhibit interleukin-4 (IL-4). Astragalus has also been shown to be useful to downregulate chronic pain. One or more of the following may be suitable.

Hist Reset (Pure) – 2 BID or TID. To downregulate histamine.
Perilla Extract (Pure) – 2 BID or TID. To downregulate IL-4 in the context of neuropathy if present.
Balanced Immune (Pure) – 2 or 3 TID. More if needed. To downregulate the NFkB of the inflammatory process.
Boswellia (Pure) – 2 or 3 BID. To downregulate human leukocyte elastase (HLE), a tissue destructive lysosomal enzyme released by neutrophils to kill pathogens. HLE is often central to ongoing tissue destruction in neutrophilic inflammation.
EPA/DHA Essentials (Pure) – 2 BID. To downregulate inflammation over the long term.
Vitamin D – Dose depends on the patient's baseline dose needed to bring blood levels into the top 25% of the normal range. If the patient's D level is already there, additional D may not be needed.

Address T Cell Polarization

If the patient has chronic susceptibility to infections, consider support for adequate Th1 and NK cell function. Th2 dominance is a barrier to adequate Th1/NK cell activation. If the patient is Th2 dominant, downregulating Th2 will be suitable as well. In some patients, inflammation of the bladder wall epithelia will drive the production of Th2-promoting cytokines, making it difficult to mount an adequate Th1/NK cell response with which to eradicate pathogens.

Th1 Support (Pure) – 2 or 3 at breakfast and lunch. Supports adequate Th1 response. Note: Because berberine increases AMPK, it can give the patient more energy. That's useful, but also means taking it late in the day might need to be avoided in some patients.
Innate Immune Support (Pure) – 1 or 2 BID. Upregulates natural killer cell activity as part of Th1 system activation. This helps restore adequate Th1 response.
Th2 Modulator (Pure) – 2 or 3 BID. Downregulates IL-4 and GATA3, reduces mast cell degranulation, breaks down excess mucous, supports glutathione production.
Perilla Extract (Pure) – 2 or 3 BID. Downregulates IL-4. May need dose escalation early in entrenched cases.
Epi-Integrity (Pure) – 1 or 2 scoops BID. Downregulates IL-4 and GATA3, repairs leaky epithelial barriers, supports Th1 response. Useful when epithelial dysfunction is present, as is common in the inflamed bladder.