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EDITORIAL: EMERGING CONCEPTS IN THE MANAGEMENT OF PROSTATITIS/CHRONIC PELVIC PAIN SYNDROME

Chronic prostatitis/chronic pelvic pain syndrome is a common problem that causes considerable morbidity and economic impact. The syndrome is characterized by chronic pain with varying degrees of urinary symptoms. The National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI) was developed using a select group of patients who were primarily referred to academic urological practices.

Assessment of its use to measure symptoms among the general population in primary and secondary care clinics is an important undertaking of the group at the University of Washington (page 580). In this sample of HMO enrollees with recent primary and secondary care visits for chronic pelvic pain the NIH-CPSI performed well in its global assessment of the patient as well as discriminating among pain, urinary symptoms and quality of life. Furthermore, there was a reasonably good correlation between these parameters and other valid measures of these indexes. However, there were several areas where the NIH-CPSI did not correlate as well. For example, the NIH-CPSI only discriminated men with low pain and disability from those with higher levels of pain and disability but did not discriminate between the low versus the moderately high disability groups. Thus, in selected patients a specific disability index score may be useful. In addition, the internal consistency coefficients were lower than those in the original validation study. These differences, in part, may be explained by the fact that the HMO enrollees tended to have less discomfort for shorter periods than the cohort initially used to construct the NIH-CPSI. Nevertheless, the NIH-CPSI total score is a valid, reliable and responsive measure of prostatitis symptoms in primary and secondary care patients.

The etiology of the chronic pelvic pain syndrome remains an enigma. Traditional thinking implicates the prostate as a primary source of the discomfort and bacteria and/or inflammatory cells as the cause of prostate malfunction. To this end, a lot of time and effort have been spent using traditional culture and more sophisticated polymerase chain reaction techniques to identify putative bacterial pathogens that could infect the prostate and lead to the development of the chronic pelvic pain syndrome. Previous studies using sophisticated polymerase chain reaction technology have failed to identify evidence of bacteria in prostatic tissue obtained from young, presumably healthy cadaver specimens. Traditional techniques to identify bacteria in the prostate are based on localization studies that identify bacteria in prostatic fluid and/or post-prostatic massage voided urine. Obviously, contamination of these specimens from urethral organisms is a common concern and the significance of low numbers of bacteria in expressed prostatic secretions is questionable.

To avoid this problem, Lee et al (page 584) performed transperineal tissue biopsies of the prostate. Contamination of the skin was negligible. Using aggressive culture techniques, they identified low numbers of nonpathogenic bacteria in about 30% of the patients and controls. Older men were more likely to have positive cultures, as were those with inflammation in the prostatic fluid. None of the counts was high enough and/or associated with recognized pathogenic bacteria and, therefore, these bacteria are probably colonizing bacteria rather than infecting strains. Since the patients in these studies were older, it is likely that in time urethral bacteria colonized the prostatic ducts. However, these bacteria apparently do not lead to a host response and, particularly, there is no evidence that they are associated with symptoms.

It is well recognized that even if pathogenic bacteria are present in the prostate, as in men with established chronic bacterial prostatitis, they do not cause chronic pelvic pain unless acute urinary tract infection develops. Taken together, these data suggest that bacteria do not have a significant role in the development of the chronic pelvic pain syndrome. The clinical observation that antimicrobial therapy reduces symptomatology in men with chronic pelvic pain syndrome is being tested in a double-blinded NIH controlled study. Since antimicrobials may have anti-inflammatory activity, it is possible that these drugs may benefit the patient by reducing inflammation rather than eradicating bacteria.

The positive correlation between serum prostate specific antigen (PSA) and prostatic inflammatory cells has been previously established. Carver et al (page 589) determined that about a third of men who present for prostate cancer screening have 10 or more white blood cells (WBCs) per high power field in the expressed prostatic secretions. The mean PSA level in men with 10 or more WBCs (2.3) was statistically significantly higher than the value (1.4) obtained in men with less than 10 WBCs in the expressed prostatic secretion. Furthermore, 18% of the men with 10 or more WBCs per high power field had a PSA of 4 or more compared to 5.2% of those with less than 10 white cells. However, no cancer was identified on biopsy in 8 patients in the inflamed group and 4 in the noninflamed group. No cultures were performed to determine if any of these men had bacteria associated with the inflammation but I would predict that no more than 5% of them did. Nevertheless, many clinicians have traditionally treated such patients with increased PSA and evidence of inflammation in the expressed prostatic secretion with antimicrobials, and occasionally observed a reduction in the expressed prostatic secretion and inflammation. If this occurs, a biopsy can be delayed but clearly the patient needs to be followed carefully because the majority with increased WBCs do not have increased PSA and, conversely, the majority with increased PSA do not have significant inflammation.

The treatment of men with chronic pelvic pain syndrome is exceedingly difficult because of the lack of understanding of the pathogenesis of this condition. Presumably, multiple factors are involved. Since obstructive voiding symptoms have a role in a reasonable number of patients, α-blockade has been used in an effort to reduce symptomatology. Cheah et al (page 592) demonstrated that 56% of patients on α-blockade showed improvement in the NIH-CPSI score but as is almost always the case, a favorable placebo response was observed in 36% of the patients. Nevertheless, the results are statistically significant suggesting that α-blockade can benefit some of these individuals. Clearly, studies of this type are hampered by the fact that presumably only those patients with significant bladder outlet obstruction and/or voiding dysfunction will benefit from α-blockade, and these individuals constitute a subset of the population of the men with chronic pelvic pain syndrome. Hopefully in the future we will be able to identify individuals with significant bladder outlet obstruction prospectively and thereby insure a better re-

sponse rate to α-blockade therapy. The NIH and the Chronic Prostatis Collaboration Clinical Research group have conducted an adequately powered study assessing the efficacy of antimicrobial therapy, α-blockade, combination therapy or no therapy in men with chronic pelvic pain syndrome. The outcome of the study will be available shortly and should provide important guidelines for the use of antimicrobials and/or α-blockade in men with chronic pelvic pain syndrome.

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