Published in final edited form as:

J Urol. 2008 May ; 179(5): 1857–1862. doi:10.1016/j.juro.2008.01.028.

Monocyte chemoattractant protein-1 (MCP) and macrophage inflammatory protein-1 α (MIP) as possible biomarkers for the chronic pelvic pain syndrome

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Abstract

PURPOSE—The chronic pelvic pain syndrome (CPPS) is characterized by pelvic pain, voiding symptoms and varying degrees of inflammation within expressed prostatic secretions (EPS). We evaluated the chemokines MCP-1 (CCL2) and MIP-1 α (CCL3) in EPS to identify marker elevations associated with both inflammatory (IIIA) and non-inflammatory (IIIB) CPPS. In addition, chemokine levels were correlated with clinical pain as determined by the NIH chronic prostatitis symptom index (CPSI).

MATERIALS AND METHODS—EPS were collected by digital rectal examination and evaluated by ELISA for MCP-1 and MIP-1 α in 154 patients; controls (n = 13), BPH (n = 54), CPPS IIIA (n = 37), CPPS IIIB (n = 50). MCP-1 and MIP-1 α levels were compared between IIIA, IIIB, and the control subgroups and correlated against the CPSI and pain sub-score using a Spearman test.

RESULTS—Mean levels of MCP-1 in the control, inflammatory BPH, non-inflammatory BPH, inflammatory CPPS, and non-inflammatory CPPS were 599.4, 886.0, 1636.5, 3261.2, and 2272.7 pg/ml, respectively. Mean levels of MIP-1 α in the control, inflammatory BPH, non-inflammatory BPH, IIIA CPPS, and IIIB CPPS were 140.1, 299.4, 238.7, 1057.8, and 978.4 pg/ml, respectively. For each cytokine, both CPPS subtypes had statistically higher levels than the control group and BPH patients (p=0.0002). Receiver operating curves utilizing MCP-1 levels greater than 704 pg/ml and MIP-1 α greater than 146 pg/ml identified patients with CPPS with an accuracy of 90% from control

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patients. MIP-1 α levels ($p=0.0007$) correlated with the pain sub-score of the CPSI while MCP-1 ($p=0.71$) did not.

CONCLUSIONS—MCP-1 and MIP-1 α within the prostatic fluid in both CPPS subtypes provide candidate future biomarkers for CPPS. In addition, MIP-1 α elevation in EPS provides a new marker for clinical pain in CPPS patients. Given these findings, prostatic dysfunction likely plays a role in the pathophysiology of some patients with this syndrome. These chemokines may serve as effective diagnostic markers and modulators against the chemokines could provide an attractive treatment strategy in individuals with CPPS.

Keywords

chronic pelvic pain syndrome; prostatitis; cytokines; monocyte chemoattractant protein; macrophage inflammatory protein

INTRODUCTION

Prostatitis is the most frequent urologic diagnosis in men under the age of 50, accounting for 8% of all office visits to urologists¹. The majority of prostatitis cases are classified as chronic pelvic pain syndrome (CPPS, NIH Category III). CPPS is characterized by suprapubic, pelvic, or perineal pain and lower urinary tract voiding symptoms, in the absence of bacterial infection. It is subclassified into categories IIIA (inflammatory) and IIIB (non-inflammatory). While various mechanisms including immunologic dysfunction, infectious, and neurologic dysfunction have been cited in the development of the syndrome, the etiology and pathogenesis of CPPS is poorly understood.

Inflammatory mediators such as cytokines and chemokines have been suggested in the pathogenesis of CPPS. Cytokines are regulatory proteins released by various cellular subtypes that promote intercellular communication and immune responses. Chemokines are a subset of cytokines that recruit and activate immune cells to sites of inflammation. We have previously found elevated levels of IL-1 β , TNF- α , IL-8 (CXCL8), and ENA-78(CXCL5) in the prostatic fluid of only CPPS IIIA patients, but not in IIIB and control patients^{2, 3}. The chemokine monocyte chemoattractant protein (MCP-1/CCL2) has specific chemotactic and activating activity for monocytes⁴. MCP-1 levels have been elevated in other disorders associated with inflammation including rheumatoid arthritis, abdominal aortic aneurysms, and coronary artery disease^{4, 5}. Macrophage inflammatory protein (MIP-1 α /CCL3) is another chemokine which is chemotactic for macrophages and T-cells⁶. It has been elevated in rheumatoid arthritis, systemic lupus erythematosus, and ulcerative colitis. In addition, it also plays an important role in inflammation induced hyperalgesia^{7, 8}.

This is the first study to identify elevations in the chemokines MCP-1 and MIP-1 α in the expressed prostatic secretions (EPS) of both CPPS IIIA and IIIB patients. Previous studies of other cytokines (e.g. IL-1 β , TNF- α) have shown elevation in only IIIA patients, but not in IIIB patients^{2, 3, 9}. The elevations in both subtypes provide evidence that MCP-1 and MIP-1 α may be candidate biomarkers for this syndrome. MIP-1 α levels also correlated with pain scores in both CPPS subtypes. This suggests that prostatic dysfunction is partially contributing to this syndrome.

MATERIALS AND METHODS

Serum and EPS samples were collected by digital rectal examination (DRE) from urology clinic patients who had no urologic disease (controls, $n = 13$), BPH ($n = 54$), CPPS IIIA [≥ 10 white blood cells per high power field (WBC/hpf) in EPS] ($n = 37$), or CPPS IIIB [<10 WBC/hpf in EPS] ($n = 50$) (see table 1). Classification was based on history, symptoms, DRE of the

prostate, negative urine culture, and WBC/hpf in the EPS. Controls included men undergoing vasectomy or routine urologic evaluation with no history or symptoms of urinary tract inflammation, palpably normal prostates and normal PSA values (<4.0 ng/ml) for patients 50 years of age or older. Men with BPH had typical obstructing voiding symptoms of BPH and palpably enlarged prostates. The BPH patients were subdivided by their EPS WBC count into inflammatory (≥ 10 WBC/hpf) and non-inflammatory BPH (<10 WBC/hpf). Men with CPPS had a history of at least 3 months of pelvic or perineal pain and EPS with or without inflammation.

A complete history and physical examination including a four-glass urine collection¹ were done on each patient. Control patients, CPPS IIIA, and CPPS IIIB patients completed the NIH Chronic Prostatitis Symptom Index (CPSI). The CPSI is a self-administered validated tool that assesses patient's baseline status and is comprised of 3 domains: pain, voiding symptoms, and impact of symptoms. The total CPSI can range from 0 to 43 and the pain sub-score can range from 0 to 21. A change of 4 to 6 points is considered clinically significant¹. The first voided urine specimen represented the urethral washout and was limited to 10 ml. The second voided specimen was collected late in the urinary flow. Prostatic fluid was obtained by digital massage within 10 minutes of voiding and collected in a sterile manner on a glass slide. The fluid was transferred to a cryovial, stored at -20°C for approximately 2 hours and then transferred for storage at -70°C until thawed for analysis. The residual fluid on the slide was placed under a coverslip and examined promptly with the high power (400x) microscopic lens for cells and particles in 3 to 5 fields. The average number of WBC/hpf was recorded. Microscopic examination of the urine sediment was done after centrifugation for approximately 3 minutes. Localization of bacteria was done according to the method of Mears and Stamey¹⁰, using clean collected specimens streaked on blood and MacConkey's agar plates. Standard microbiologic methods were used to quantify and identify all organisms.

Enzyme linked immunosorbent (ELISA) assays were used to measure MCP-1 and MIP-1 α in the EPS and serum of patients (CYTIIMMUNE Sciences, College Park, MD or R&D Systems, Minneapolis, MN). Most samples were assayed for both cytokines but due to inadequate volume, some samples were only assayed for one cytokine. Each assay was performed using dilutions of recombinant human MCP-1 and MIP-1 α . Results were compared to a standard curve prepared using recombinant human cytokines. Values were computed taking the dilution factor into account. Assays were read at 490 nm in an ELISA plate reader (Molecular Devices Corporation, Sunnyvale, CA).

The distributions for the cytokine levels, age, CPSI, and WBC were examined. MCP-1 and MIP-1 α levels were log transformed in all analyses given their right-skewed distributions. A two-sample t test was performed to compare the MCP-1 and MIP-1 α levels between the IIIA, IIIB, and control group. Receiver operation curves (ROC) were developed to determine the optimal cutoff point of the cytokine levels in discriminating the IIIA and IIIB groups from the control group. The Spearman correlation coefficients and p-values were reported to show the correlation between cytokine levels, CPSI, CPSI pain sub-score, and WBC levels in EPS. All analyses were performed using SAS version 9.1 (Cary, North Carolina).

RESULTS

Demographics

The mean age, age distributions, CPSI, and WBC counts are shown in table 1. There was a statistically significant difference in age in the BPH group compared to the other subgroups. The CPSI and pain subscore between the IIIA and IIIB group were not statistically different.

Chemokines in prostatic fluid and serum

MCP-1 levels in the various groups are illustrated in figure 1a. MCP-1 was detectable, based on a threshold of detection of 15.6 pg/ml, in 10/10 controls, 54/54 BPH patients, 24/24 patients with CPPS IIIA, and 38/38 patients with CPPS IIIB. MCP-1 was not measured due to inadequate volume in 13 CPPS IIIA and 12 CPPS IIIB patients. The variability in MCP-1 ranged from 22 pg/ml to 17502 pg/ml. The MCP-1 levels in the control, inflammatory BPH, non-inflammatory BPH, CPPS IIIA, and CPPS IIIB are 599.0 ± 919.0 (mean $\pm$ standard error), 886.0 ± 183.5 , 1636.5 ± 244.8 , 3261.2 ± 517.9 , and 2272.7 ± 461.8 , respectively. Pairwise comparisons were performed to determine the extent of differences between each intergroup. Controls and BPH patients with or without inflammation had the lowest levels. CPPS IIIA and IIIB patients had MCP-1 values statistically greater than control and BPH patients ($p=0.0002$). IIIA patients also had higher MCP-1 values than IIIB patients ($p=0.05$). IIIA patients also had higher MCP-1 values than IIIB patients ($p=0.05$), although the difference is not statistically significant after the Bonferroni adjustment for multiple comparison.

MIP-1 α levels in the various groups are illustrated in figure 1b. MIP-1 α was detectable, based on a threshold of detection of 7.8 pg/ml, in 13/13 controls, 52/54 BPH patients, 32/32 patients with CPPS IIIA, and 44/44 patients with CPPS IIIB. MIP-1 α was not measured due to inadequate volume in 5 CPPS IIIA and 6 CPPS IIIB patients. The variability in MIP-1 α ranged from 23 pg/ml to 14832 pg/ml. The MIP-1 α levels in the control, inflammatory BPH, non-inflammatory BPH, CPPS IIIA, and CPPS IIIB are 140.1 ± 37.0 (mean $\pm$ standard error), 299.4 ± 57.1 , 238.7 ± 41.9 , 1057.8 ± 261.5 , and 978.4 ± 379.8 , respectively. Pairwise comparisons were performed to determine the extent of differences between each intergroup. Controls and BPH patients with or without inflammation had the lowest levels. CPPS IIIA and IIIB patients had significantly higher values compared to controls and BPH patients. There was no difference in MIP-1 α levels between IIIA and IIIB patients. The serum levels of MIP-1 α and MCP-1 were not statistically different between CPPS IIIA, CPPS IIIB, and control patients (figure 1c).

MCP-1 and MIP-1 α as biomarkers for CPPS

ROC curves were developed to identify cutoff values to discriminate CPPS IIIA or IIIB patients from control patients in our cohort. For MCP-1, a level greater than 704 pg/ml is predictive of CPPS (accurate for 52/53 patients, positive predictive value of 98%). For MIP-1 α , a level greater than 146 pg/ml is predictive of CPPS (accurate for 55/59 patients, positive predictive value of 93%). The ROC curve for utilizing both cut points is shown in figure 2. The area under the ROC curve for patients with MCP-1 levels greater than 704 and MIP-1 α greater than 146 approaches 90%, which is highly accurate for this group of patients. Utilized together, the elevation in both chemokines discriminates patients with CPPS from the control group.

Chemokine correlation with CPSI, pain sub-score, and WBC count of EPS

A Spearman correlation was performed between NIH-CPSI and pain subscore versus MCP-1 and MIP-1 α levels. In addition, a correlation between WBC count in EPS versus MCP-1 and MIP-1 α levels was also performed. MIP-1 α showed a positive correlation with the CPSI ($r=0.41$, $p=0.0002$) and the pain subscore ($r=0.38$, $p=0.0007$, figure 3), while MCP-1 did not correlate with either (table 2). Meanwhile, MCP-1 positively correlated with the WBC count of EPS ($r=0.44$, $p=0.003$), while MIP-1 α did not (table 2).

DISCUSSION

Chemokines are a subset of cytokines that play a major role in regulating the immune response by recruiting inflammatory cells, including macrophages, monocytes, and neutrophils. Chemokines have been implicated in the pathogenesis of such conditions as rheumatoid arthritis, abdominal aortic aneurysm, and coronary artery disease^{4, 5, 11}. MCP-1 and MIP-1 α

recruit monocytes and macrophages via their release from fibroblasts and macrophages in joints of rheumatoid arthritis patients and perpetuate the inflammatory process^{4, 6}. The role of inflammation in CPPS is still debated. Our previous studies have shown elevations of various chemokines in individuals with inflammatory CPPS (IIIA patients), but did not identify patients with the syndrome (both IIIA and IIIB).^{2, 3}

Both chemokines evaluated in this study were elevated in CPPS IIIA patients, compared to control and BPH patients. This is consistent with previous studies from our group and others. In addition, both chemokines were significantly elevated in CPPS IIIB patients. In addition, the elevated levels of cytokines were noted in EPS, but not in the serum. The measurement of chemokines within EPS can serve as an additional diagnostic tool in the evaluation of CPPS. The utilization of both chemokines to identify CPPS patients (both IIIA and IIIB) provides future biomarkers for the diagnosis of CPPS. The receiver operating curves utilizing a MCP-1 greater than 704 pg/ml and MIP-1 α greater than 146 pg/ml provides a test with greater than 90% accuracy for identifying CPPS patients from the control patients in our study cohort. Further studies with large number of patients and controls can help identify specific cutpoints to differentiate CPPS patients from control and BPH patients. These chemokines in the future may help identify confounding patients who have the chronic pelvic pain syndrome, from other patients with similar symptoms of a different origin. For example, a patient with major depressive disorder or irritable bowel syndrome may have similar symptoms but doesn't have prostatic dysfunction. The use of these cytokines within EPS may help differentiate these patients from individuals with CPPS and help direct treatment. The elevation of these chemokines in both subtypes suggests the prostate plays a role in the pathogenesis of CPPS and further study of these biomarkers will aid in understanding the pathophysiology of this disease process.

The paradoxical finding of elevated levels of the inflammatory chemokines within the non-inflammatory CPPS (as classified by < 10 WBC/hpf in EPS) patients was a novel finding. CPPS IIIB patients maybe at an earlier stage of the disease process prior to substantial neutrophil infiltration. During the early stages, patients have pain and voiding symptoms associated with minimal inflammation and few WBCs in the prostatic fluid. As the disease progresses, patients may have neutrophil infiltration of the prostate and WBCs are noted in the prostatic fluid. Hence, both CPPS subtypes may represent part of the same clinical spectrum and the chemokines MCP-1 and MIP-1 α may be initiating the early inflammatory process. Cytokine modulators including MCP-1 receptor antagonists, anti-MCP-1 antibodies, and MIP-1 α receptor antagonists that are currently in clinical trials may serve as potential targets for therapeutic intervention against inflammatory and hyperalgesic pathways in both CPPS subtypes¹².

Our study also represents the first of its kind to reveal a correlation between clinical pain and cytokine levels. MIP-1 α levels positively correlated with the CPSI pain subscore in our cohort. MIP-1 α has recently been shown to play an important role in inflammation induced hyperalgesia^{7, 8}. Putative mechanisms suggested include desensitizing the mu-opioid receptor on peripheral sensory neurons. The elevation of MIP-1 α in both subtypes and correlation with pain sub-score suggest that prostatic dysfunction may contribute to the pain symptoms of the syndrome. MIP-1 α can also be followed clinically to assess responses to therapy in a future study. Therapeutic strategies against MIP-1 α represent an attractive therapeutic strategy that targets both the inflammation and hyperalgesia pathways.

There are some limitations to our study. The MCP-1 and MIP-1 α had a wide range of values as noted by the high standard errors; in an attempt to correct for this, we utilized logarithmic analysis to correct for the right-sided skewing of the data from a few samples with very high levels of cytokines. The cytokine levels were measured from a relatively small number of CPPS

patients at a single referral center and due to the small EPS volume, both cytokines were not measured in every patient. The CPSI data was also absent from the BPH controls. Another limitation was the relatively small number of controls in our cohort. Also, the cross-sectional nature of the study precluded following patients longitudinally over time to check their chemokine levels with disease activity. In addition, we evaluated only two chemokines in this study while numerous chemokines may play a role in the pathogenesis of CPPS. Additionally, other chemokines that bind to MCP-1 and MIP-1 α corresponding receptors may provide additional insight to understanding the pathogenesis of CPPS.

CONCLUSIONS

The identification of increased MCP-1 and MIP-1 α levels in the EPS provides potential biomarkers to aid in the identification of patients with CPPS in the future. The identification of specific cutpoints in larger cohorts, the monitoring of chemokine levels with disease progression, and improvement of chemokine levels with treatment would validate these chemokines as biomarkers for CPPS. Their elevation in both subtypes suggests a pivotal role of the prostate in the pathogenesis of CPPS. In addition, the correlation of MIP-1 α with the pain sub-score of the CPSI suggests an important role for MIP-1 α in the development of chronic pain. The identification of MIP-1 α elevation in the pathogenesis of other disorders with pain suggests a novel therapeutic target in CPPS patients. As current treatment strategies against CPPS are often unsuccessful, novel approaches against these chemokines might be efficacious in the future.

Acknowledgments

Study was supported by funds from the Veteran's Administration Research Service, NIH grants AI40987, HL58694, and AR48267

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Key of definition for abbreviations

CPPS	chronic pelvic pain syndrome
MCP-1	monocyte chemoattractant protein 1
MIP-1α	macrophage inflammatory protein-1 α
CPSI	chronic prostatitis symptom index
EPS	expressed prostatic secretions
CCL	chemokine ligand

Figure 1.

a – Monocyte chemoattractant protein (MCP-1) levels in expressed prostatic secretions (EPS) of controls, inflammatory benign prostatic hyperplasia (BPH), non-inflammatory BPH, chronic pelvic pain syndrome with inflammation (CPPS IIIA) and chronic pelvic pain syndrome without inflammation (CPPS IIIB). The lower limit of detection, as determined by the lowest detectable assay standard by ELISA assay is 15.6 pg/ml. Error bars show standard error.

* - indicates statistically significant difference

b – Macrophage inflammatory protein (MIP-1α) levels in expressed prostatic secretions (EPS) of controls, inflammatory benign prostatic hyperplasia (BPH), non-inflammatory BPH, chronic pelvic pain syndrome with inflammation (CPPS IIIA) and chronic pelvic pain syndrome without inflammation (CPPS IIIB). The lower limit of detection, as determined by the lowest detectable assay standard by ELISA assay is 15.6 pg/ml. Error bars show standard error.

* - indicates statistically significant difference

c – MCP-1 and MIP-1α levels in the serum of CPPS IIIA, CPPS IIIB, and control patients.

Figure 2.

Receiver operating curve (ROC) to discriminate IIIA/IIIB patients from control patients in our cohort employing MCP-1 cutpoint of 704 pg/ml and MIP-1 α cutpoint of 146 pg/ml (Area under ROC curve – 0.892)

Figure 3.
Spearman correlation between CPSI pain sub-score and MIP-1 α levels.

Table 1

Patient demographics

Diagnosis	# of pts	Mean age (yrs)	Range (yrs)	CPSI	CPSI pain sub-score	WBC in EPS
Control	13	43.7	24-68	1.4	0	3.3
BPH (≥10 WBC)	19	68.9	54-89	N/A	N/A	18.7
BPH (<10 WBC)	35	70.8	54-83	N/A	N/A	1.8
IIIA	37	44.1	21-68	21.9	10	18.5
IIIB	50	44.9	30-64	23.5	11	4

Control - no urologic disease

BPH – benign prostatic hyperplasia

- sub-classified into patients with ≥ 10 and < 10 white blood cells/high field (WBC/hpf) in expressed prostatic secretions (EPS)

- CPSI score not available on BPH patients

IIIA – chronic pelvic pain syndrome IIIA, ≥ 10 WBC/hpf in EPS

IIIB – chronic pelvic pain syndrome IIIB, < 10 WBC/hpf in EPS

CPSI – chronic prostatitis symptom index (score ranges from 0 to 43)

EPS – expressed prostatic secretions

Table 2

Correlation of macrophage inflammatory protein (MIP-1 α) and monocyte chemoattractant protein (MCP-1) levels with chronic prostatitis symptom index (CPSI), CPSI pain sub-score, and white blood cell (WBC) count of expressed prostatic secretions (EPS)

Spearman correlation coefficient			
	CPSI	Pain sub-score	WBC of EPS
MIP-1 α	0.40995 (p = 0.0002)	0.37394 (p = 0.0007)	0.12362 (p = 0.3731)
MCP-1	0.03647 (p = 0.7748)	0.04695 (p = 0.7103)	0.44476 (p = 0.0032)