



Prostatic Calcification: A Rare Differential Diagnosis of Inguinal Pain

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Abstract

Background: Prostatic calcification associated with chronic bacterial prostatitis is a cause of Lower Urinary Tract Symptoms (LUTS) and chronic pelvic pain.

Case Report: We describe the case of a 56-year-old man who had severe lower urinary tract irritation symptoms along with bilateral, intermittent, deep inguinal pain for over two years. Computed Tomography (CT) of the abdomen and pelvis showed prostatic calcification and ruled out other concurrent causes of inguinal pain, namely hernia, inflammation of the lymph nodes, cancer, and mesenteric panniculitis. A diagnosis of Chronic Bacterial Prostatitis (CBP) with calcification was made after clinical (NIH-CPSI and I-PSS), laboratory (Urine, CBC, EPS, and seminogram), and transabdominal ultrasonographic evaluation, particularly for the prostatic dense inclusion. Following antibiotic and anti-inflammatory treatment, inguinal pain completely subsided, and prostatic calcification was much reduced on follow-up US.

Conclusions: Our report demonstrates a rare and significant differential diagnosis of inguinal pain and the predictive value of transabdominal ultrasonography in evaluating prolonged chronic inguinal pain in addition to the effectiveness of longer medication treatment for the symptomatic manifestations of prostatic parenchymal calcifications in patients with CBP.

Keywords: Prostatic Calcification; Pain; Inguinal; Ultrasound

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Introduction

When evaluating transabdominal ultrasonography, prostatic calcification often appears as an incidental finding in the form of echo-dense inclusions. In particular, it mostly occurs in 47-48.9% of cases with Chronic Bacterial Prostatitis/Chronic Pelvic Pain Syndrome (CBP/CPPS). Patients with prostatic calcification mainly complain of lower urinary tract symptoms, with various non-specific symptoms such as perineal discomfort, suprapubic pain, lumbosacral ache, and/or and testicular pain [1, 2].

Moreover, prostatic calcification may be linked to several pathologies (particularly hernia, lymph node inflammation, cancer, and mesenteric panniculitis) during a workup examination for pelvic and abdominal pain [3, 4]. The diagnosis of such complicated pathologies can be delayed because the symptoms mimic other, more common disorders.

We present a case of prostatic calcification in a 56-year-old man who had symptoms of bilateral, intermittently recurring inguinal pain that was initially thought to be caused by an inguinoscrotal pathology.

Case Report

A 56-year-old man without any medical or family history presented with recurrent intermittent inguinal pain for more than 2 years and lower UT irritative symptoms. Every attack lasted from 30 minutes to several hours, and the pain was intense, deep, insidious, and radiating from the lower abdomen. Inguinal pain was linked to painful micturition, more frequent urination, and sometimes suprapubic pain, which was aggravated after sexual intercourse. Pain was experienced either during or after ejaculation. The pain was not associated with a visible or palpable bulge in the groin, did not worsen with activity or coughing, and was not associated with weight loss, fever, or other

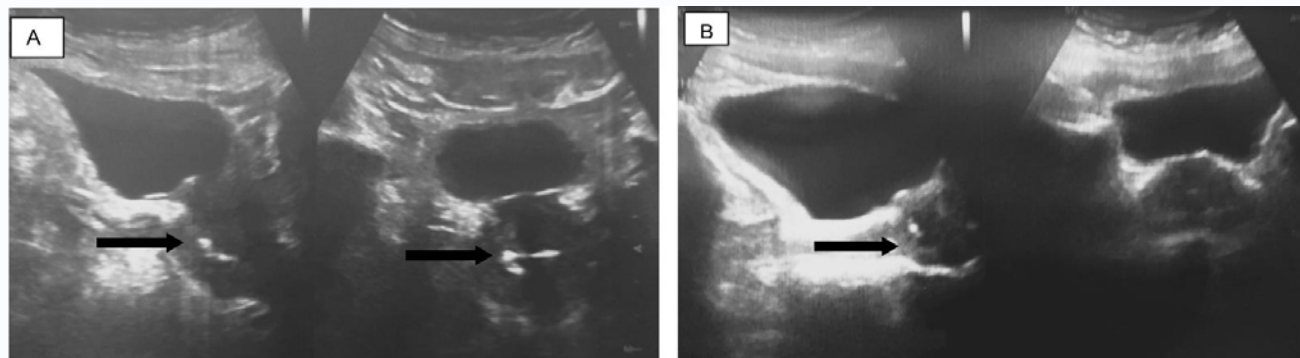


Figure 1: Transabdominal ultrasound examination of the prostate in a 56-year-old patient with inguinal pain. A: Both the Axial and sagittal images demonstrate scattered echo-dense inclusions in the prostate parenchyma on the first clinic visit (black arrow); B: Both the Axial and sagittal images demonstrate residual dense prostatic parenchymal inclusion (black arrows) on the second clinic visit after 3 months of treatment. The residual prostate calcification appeared as the hyper echogenicous foci with an estimated volume of $\leq 1.3 \text{ mm}^3$ (black arrow).

symptoms. The pain was not related to food intake, defecation, or any other activities of daily living. The frequency of pain gradually increased and affected his daily life. The pain was not associated with any other cause of inguinal pain (e.g., inguinoscrotal swelling, lymph node enlargement). He visited several hospitals for workups.

A diagnosis of bilateral nonsurgical, long-standing neuropathic pain or inguinal swelling with inflammation was strongly suspected. Nevertheless, his symptoms persisted despite alpha-blocker therapy and non-steroidal anti-inflammatory medications. No palpable inguinal lump or tender abdominal point was observed during the clinical examination. No testicular abnormalities or urethral discharge were detected during the urogenital examination. Digital rectal examination revealed a mildly enlarged prostate with a regular surface contour and firm consistency, with no palpable nodular lesions.

Samples of urine, semen, and Expressed Prostatic Secretion (EPS) were sent for bacterial culture. A urinary tract infection and pyospermia were found in the semen. However, urine, prostatic fluid, and semen bacterial cultures were negative. The patient's total serum PSA level was 1.77 ng/mL, and his free PSA was 0.43 ng/mL at the time of diagnosis. Uroflowmetry revealed mild urinary tract obstruction.

A radiologist performed an imaging evaluation using pelvic ultrasonography, which showed that the patient's prostate weighed 26 grams and had multiple coarser echoes-dense inclusions ($>3\text{mm}^3$) scattered throughout the gland (Figure 1A). Furthermore, no lesions were visible on abdominal ultrasonography. In addition to the assessment of prostatic calcification, unenhanced abdominal and pelvic computed tomography was performed to diagnose the causes of recurrent abdominal pain. Non-enhanced CT revealed two prostatic calcifications with CT attenuation values of 249 (Figure 2). A diagnosis of CBP with calcification was made after clinical assessment using NIH-CPSI and I-PSS scores. The pretreatment NIH-CPSI was 25/43, indicating moderate symptoms, and the I-PSS was 6/35, indicating mild obstructive symptoms.

We started treatment with a fluoroquinolone (ciprofloxacin) as empiric antibacterial therapy, a course of oral doxycycline, and nonsteroidal anti-inflammatory drugs for 3 months, and the inguinal pain disappeared. After 3 months, a follow-up US revealed remarkable regression and only one small hyperechogenic prostatic calcification

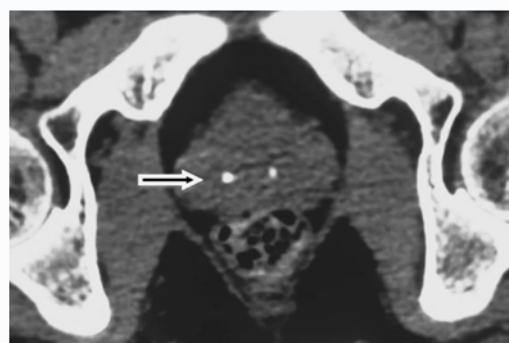


Figure 2: Axial Computed Tomography (CT) view of the prostate shows two prostatic calcifications on the first clinic visit with an estimated volume of more or less 3.5 mm^3 with an attenuation value of 249 (black arrows).

($\leq 1.3 \text{ mm}^3$) (Figure 1B). Even after treatment, his NIH-CPSI score was significantly decreased (9/43), and mild obstructive symptoms persisted, with an I-PSS of 6/35. In our case report, frequent relapses of prostatitis symptoms were reported 3 times a year. Repeated urine and EPS cultures again yielded negative results.

Discussion

Our patient presented with recurrent bilateral inguinal pain that was initially diagnosed as either inguinal pathologies or non-surgical, chronic neuropathic pain, which affects 7-10% of the population in the groin area. Prostatic calcification, a condition observed in 47-48.9% of cases using transabdominal and transrectal ultrasonography, respectively, was later diagnosed. In this case, transabdominal ultrasonography revealed prostate calcifications as echo-dense inclusions larger than 3 mm^3 , aligning with findings from other researchers, particularly in cases of CBP/CPSP [5].

As far as we are aware, this case report is the first to highlight inguinal pain as the primary symptom of prostatic calcification in a patient with CBP. Our findings suggest that calcifications are correlated with a longer duration of bilateral inguinal pain, potentially indicating a chronic infection. Nonetheless, several underlying mechanisms have been proposed for the development of inguinal pain in such cases. One of these mechanisms is the calcification of prostatic secretions, which has a calcium apatite core encircled by concentric layers. This calcification process can either lead to or be a result of chronic prostate inflammation. The calcifications

themselves serve as a hiding place for microorganisms, resulting in recurrent or challenging-to-treat CBP and local tissue inflammation. Moreover, they found that 78% of prostatic stones had bacterial imprints, which suggested that the prostate tissue had previously or currently been infected [6]. Large or numerous calcifications can obstruct prostatic ducts, resulting in fluid accumulation, elevated intraprostatic pressure, intraprostatic reflux, and further secondary inflammation and tissue damage [7]. Persistent inflammation in the area might cause persistent irritation of the nerve and spasms in the pelvic floor muscles, resulting in pain in the inguinal area, along with symptoms affecting the lower urinary tract. Furthermore, prostatic inflammation reduces the ability of prostatic tissue to maintain its pathophysiological function by interfering with smooth muscle relaxation and the prostate's microvascularization [8]. This disruption can lead to deep, radiating pain in the inguinal area.

Previous studies have indicated that most calcifications are associated with histological inflammatory changes, especially the infiltration of inflammatory lymphocytes and histiocytes. Our results demonstrated that prostate calcifications, appearing as echo-dense inclusions, were linked to inguinal pain and symptoms of prostatitis [1]. In comparison to asymptomatic prostatitis, both calcifications and the duration of pelvic pain were significantly more common in CBP. Furthermore, a noteworthy correlation exists between the relapse of prostatitis and calcification. Deep prostatic calcification may play a more significant role in the exacerbation of the disease than in its severity, particularly inguinal pain. In our case, prostatitis relapse symptoms were observed three times a year. However, the correlation between the presence of prostatic calcifications and an increase in frequency and longer duration of inguinal pain in this case suggests a more chronic course and recurrent episodes of infection [9, 10]. It is worth noting that while calcifications and inguinal pain can coexist, such pain is a common symptom with numerous other possible causes (e.g., inguinal hernias, hip joint disorders, and muscular strains). Therefore, the diagnosis relies on ruling out other causes of inguinal pain.

Furthermore, a significant correlation between increased white blood cell counts and disease symptom severity was discovered. Concurrently, the presence of echo-dense inclusions in this inflammatory type of CBP was associated with elevated leukocyte levels in the ejaculate. Our findings, which demonstrated a high leukocyte count in prostatic seminal secretions and its correlation with a prolonged duration of symptoms, confirmed this hypothesis. Patients with prostatic calcification also have higher levels of inflammatory markers in their EPS [1]. Moreover, the authors found that such patients had higher levels of IL-1 β and IL-10 in their urine and ejaculate, and that these levels were strongly correlated with the severity of prostatitis symptoms and the local inflammatory activity in the symptomatic prostatitis glands [11-13]. Our findings indicate that inguinal pain is also linked to ultrasonographic signals in the prostate tissue and pyospermia in patients with CBP. However, it cannot be ruled out that GABA and serotonin levels in the serum and ejaculate of patients with CBP may contribute to functional alterations in the neuroendocrine receptor apparatus within the prostate gland. Moreover, there is a strong correlation between their concentrations and the symptoms of prostatic calcification [13, 14].

These observations suggest that inguinal pain in the presence of prostatic calcification is probably determined by a combined effect of various factors, including the balance and interaction between

the pathogenic causes of chronic inguinal pain and the actual changes in the prostate gland. A patient's inguinal pain is caused by the development of variable degrees of central and peripheral nervous system sensitization, reactive muscular-tonic disorders, and inflammatory and degenerative processes in the prostate gland. The inflammatory process of the prostate gland predominates in the early stages of disease development, but the processes typical of chronic pain predominate in the latter stages. Consequently, an inflammatory response to pyospermia and prostatic echo-dense calcification are related as the pathology progresses, which may be reflected in our findings [13].

According to a previous study, following three months of combination therapy to remove calcification microorganisms, men with resistant CPPS and prostatic calculi showed considerable symptomatic improvement [14]. More intriguingly, under effective combination therapy (such as antibiotics or anti-inflammatory medicines), the inguinal pain was significantly improved, and the frequency of relapse had almost completely disappeared in this case report. Even the echo inclusion was significantly reduced in size after 3 months of treatment. In this case, the majority of the prostate calcification (56%) was diffusely observed throughout the gland, which showed a significant reduction in the size of the inclusion under treatment effectiveness. A larger reduction in NIH-CPSI scores in our case report was also demonstrated. Furthermore, calcification may be a sign of chronic stage disease, for which monotherapy treatments are ineffective. Consequently, combined therapy is necessary for coarser prostate stones associated with inflammation [10].

In conclusion, we describe a very rare chronic inguinal pain that manifests as prostatic calcification in men with CBP. Prostatic echo dense inclusion is a useful tool for assessing the clinical symptoms of patients with CBP and should be carefully evaluated when inguinal pain is detected. Prostatic calcification is also associated with longer duration, more severe prostatitis symptoms, and frequent exacerbations of recurrent inguinal pain in those with CBP. For optimal treatment outcomes, a longer medication period was also required in patients with prostatic calcifications.

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