

BRIEF REPORT

Recurrent Psoas Syndrome Secondary to Urolithiasis and Indwelling Ureteral Stent

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Strong evidence-based guidelines exist for the evaluation and treatment of the acute medical consequences of urolithiasis, however, less emphasis has been placed on the assessment of acute and chronic musculoskeletal effects. In our case report, a 35-year-old male who develops a chronic psoas syndrome secondary to an episode of ureterolithiasis and ureteral stent placement is subsequently treated with long-term osteopathic manual medicine (OMM). Our case suggests that urolithiasis may be associated with the development of psoas syndrome – a condition that may be amenable to OMM. The specific treatment modalities discussed may be utilized to decrease symptomatology for patients presenting with similar findings.

INTRODUCTION

Urolithiasis affects approximately 1 in 11 people, with men being affected more frequently than women (10.6% versus 7.1%, respectively).¹ The most common cause of urinary calculi is dehydration. Stones also have a propensity to form when urinary levels of calcium, oxalate, cystine, and/or uric acid are elevated.¹ Patients with urolithiasis commonly present with exquisite flank pain, hematuria, difficulty urinating, groin pain, nausea, and vomiting. Differential diagnoses are vast and various organ, nervous, and muscular pathologies must be considered.

Symptomatology and physical exam findings help guide diagnosis, but the definitive diagnosis is obtained with imaging. Computed tomography (CT) without contrast is the best modality for identifying genitourinary (GU) stones, with ultrasound and X-ray also being helpful, yet inferior.² Stones may be managed with medical expulsive therapy (MET) or surgery. Many factors contribute to treatment choice including the patient's pain, vitals, and size of the stone. Psoas syndrome is a pain condition caused by injury to the psoas musculature that may have associated spasms and specific somatic dysfunctions (SD). The syndrome is likely underdiagnosed and should be considered as a differential for low back or flank pain radiating to the groin. Patients with psoas syndrome commonly complain of difficulty standing erect or lying prone, pain in the contralateral gluteal region, and radiation of pain down the opposite leg that stops above the knee. Common etiologies include prolonged flexion stress of the lumbar spine, rapid elongation of the psoas, and compensation for other SD's.

In addition, psoas syndrome can present secondary to a variety of viscerosomatic reflexes like urolithiasis, appendicitis, prostatitis, and salpingitis. Osteopathic manipulation can be used to aid in the treatment of psoas syndrome.³ We present the first reported case of a patient with chronic psoas syndrome secondary to urolithiasis and an indwelling ureteral stent, along with useful treatment options for practitioners.

REPORT OF CASE

A 35-year-old Caucasian male presented to the emergency department with sudden onset left flank pain that radiated to his left groin. The pain began after voiding, 30 minutes prior to arrival, and was accompanied by nausea and vomiting. The patient had just finished mowing the lawn when the pain started. The pain remained constant, sharp, and severe. He denied any recent fevers, chills, hematuria, dysuria, penile discharge, testicular pain, or heavy lifting. Past medical history was only significant for an episode of epididymitis 15 years prior. Surgical history was unremarkable. Family history revealed cardiac disease, but no GU diagnoses. He was afebrile, tachycardic, and hypertensive. On physical exam the patient was in distress due to pain and had marked left flank pain on palpation. Labs revealed leukocytosis ($13 \times 10^9/L$), a creatinine of 1.1 mg/dL, and a normal serum uric acid. Urinalysis was deferred due to the patient's inability to void. An ultrasound of the kidneys and ureters displayed moderate left hydronephrosis, a dilated left proximal ureter, and a possible stone in the distal left ureter. These findings were confirmed with an intravenous pyelogram, which revealed a 2.2cm obstructing left ureteral calculus. Our patient had a left ureteral stent placed the following day, with definitive stone treatment seven days later. In the interim, he sought medical attention for a new type of pain that developed in

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the left lower back with radiation to his left posterior belt line and left groin. The pain was described as episodic, severe, and cramp-like. Physical exam unveiled an inability to stand fully erect and tenderness to palpation deep within the left iliac fossa. L1-5 were side-bent left, with a segmental dysfunction at L1 of FRISI. His left hip was significantly restricted to extension. He was diagnosed with an acute psoas syndrome which was successfully treated with combination therapy of OMM, anti-inflammatories, and muscle relaxants. Over the course of the next thirty years, he has presented with thirteen recurrent episodes of acute-on-chronic psoas syndrome; a frequency of approximately one episode every one and a half years. Almost every recurrent episode was professionally evaluated with appropriate laboratory and imaging tests to rule out urolithiasis or other possible differentials. Each of the thirteen episodes were diagnosed solely as acute-on-chronic psoas syndrome. Every episode has been amenable to serial OMM treatments and combination therapy of anti-inflammatories and muscle relaxants. Several of the OMM modalities utilized in our patient's care are discussed later in this article.

DISCUSSION

The psoas major muscle originates from the transverse processes of L1-L5, the lateral bodies of T12-L5, and the associated intervertebral discs. The muscle inserts distally on the lesser trochanter. Nervous supply to the psoas muscle is derived from the ventral rami of L1-L4. The ureter receives nervous supply from nearby autonomic plexuses, which utilize many of the same lumbar afferents and efferents as the psoas. Each ureter lies directly on the anterior surface of the respective psoas musculature.⁴ Therefore, it is reasonable to theorize that inflammation generated within our patient's ureter resulted in subsequent irritation of the underlying psoas musculature leading to psoas syndrome. Although the etiology of most cases of acute or chronic psoas syndrome are multifactorial and organic in origin, it is likely that urolithiasis and stent irritation could lead to psoas syndrome as well. This report adds to the current literature as it is the first to make a direct link between the latter.

Many OMM techniques can be applied to the treatment of psoas syndrome. The modalities discussed here are the mainstays of our treatment approach and include a thoracoabdominal diaphragm release, psoas muscle energy (*Figure 1*) lumbar muscle energy (*Figure 2*), and iliopsoas counterstrain.

The thoracoabdominal diaphragm has inferiorly-spanning tendinous arches that directly cover the superior aspects of the psoas musculature, as well as crural attachments to the upper lumbar vertebrae.⁵ This intimate relationship often leads to dysfunction of the diaphragm secondary to psoas injury. We utilize a breathing-guided integrated neuromuscular release to improve diaphragmatic excursion. The technique is performed with the patient in the supine position; the clinician's hands wrap broadly around ribs 6-10 bilaterally. The indirect barriers of all diaphragmatic planes are engaged and held by the clinician while the patient maintains the phase of their respiratory cycle that is deemed the least resistant. This process may be repeated several times until restoration of diaphragmatic motion occurs.

FIGURE 1:

Prone positioning for muscle energy of the hypertonic psoas



FIGURE 2:

Muscle energy for a side-bending dysfunction of the lumbar spine



FIGURE 3:

Pre- and post-treatment sagittal findings



Figure 1 (See page 37) displays the prone positioning for muscle energy of the hypertonic psoas. Approach this technique with care, as many patients in the acute phase may be unable to tolerate aggressive lengthening of their psoas musculature.

Figure 2 (See page 37) represents muscle energy for a side-bending dysfunction of the lumbar spine. The counterstrain point for the iliopsoas lies deep within the iliac fossa. Treatment of this point requires the patient to be positioned supine with both hips and knees passively flexed to 90°, ankles crossed, and thighs externally rotated. Maintaining correct positioning will shorten the psoas muscle fibers effectively resetting the facilitated segment, which may manifest as reduction in pain experienced by the patient.

Figure 3 (See page 37) displays the pre- and post-treatment sagittal findings from our patient. The post-treatment image on the right demonstrates lengthening of the psoas musculature with resultant improvement in erect posture.

CONCLUSION

Urolithiasis may result in inflammation of the ureter with subsequent irritation of the underlying psoas musculature, leading to psoas syndrome. In this case report, our patient demonstrates not only an acute episode, but also recurrent psoas syndrome episodes secondary to a ureteral calculus and an indwelling ureteral stent. When caring for patients with a history of urolithiasis and chronic pain, it is important for physicians to consider the diagnosis of psoas syndrome. OMM may be utilized to reduce patient symptomatology and improve somatic dysfunction related to psoas muscle spasm in both the acute and chronic settings.

AUTHOR DISCLOSURES:

No relevant financial affiliations

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