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ORIGINAL RESEARCH

A rare cause of non-obstructive bilateral perinephricurinoma

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ABSTRACT
Urinoma, defined as the urine leakage beyond the urinary tract, is commonly induced by blunt trauma or urinary tract obstruction by stone, intra-abdominal malignancy, or retroperitoneal fibrosis. Spontaneous urinoma is rare and parenchymal pathologic change is rarely mentioned when urinoma is found. We present a case of a 28-year-old man with left flank pain due to spontaneous non obstructive urinoma. The patient received analgesics for flank pain and was found to have bilateral peri-renal urinoma on CT. After discontinuing the medication, urinoma subsided, and the patient was discharged with normal serum creatinine. This was the first case of urinoma induced by NSAID. Its pathophysiology and management of spontaneous urinoma are discussed.
Key words: spontaneous urinoma, NSAID, interstitial nephritis

INTRODUCTION

CLINICAL HISTORY

A 28-year-old man presented with history of left back pain for past 1 week, for which he took analgesics regularly for a week. The patient denied of any previous hospitalization in recent years. The patient visited our emergency department with aggravating pain in bilateral flanks. The patient did not have any other significant medical history. Physical examination revealed bilateral costovertebral angle tenderness. Blood examination revealed an acute decline in renal function (serum creatinine: 1.6 mg/dl). Urinalysis did not have any significant findings. Due to persistent pain in the flank regions the patient was advised CT abdomen and pelvis.

IMAGING FINDINGS

Contrast excretory urogram showed bilateral perinephricurinoma. No evidence of renal calculi or hydro-ureteronephrosis on either side. (Fig 1).
Both kidneys showed normal excretion (Fig 2).
Diagnosis of acute NSAID induced nephritis was considered and treated conservatively.

After one week the patient came for follow-up, there was improvement in renal function (serum creatinine: 0.9 mg/dl, urea:18 mg/dl) and the urinoma has subsided in follow up ultrasound (fig 3).

Discussion

Urinoma is typically a collection of fluid found in the peri-renal area [1]. It is mostly caused by trauma with breach in the integrity of the renal pelvis, calices, or ureter. Sub-capsular fluid collection as a presentation in patients with renal parenchymal disease without any cause of obstruction is rare. In literature focal segmental glomerulonephritis is the common histopathologic feature [3-5].In our patient, acute interstitial nephritis due to NSAID was the etiology of the acute kidney injury and consequent subcapsular fluid accumulation[4].Perirenal fluid is a spontaneous subcapsular transudate in patients suffering from nephropathy and in a sodium retention state, with or without renal failure[6].Subcapsular fluid collection may be noticed incidentally on imaging as a crescent-shaped collection. Distension of the renal capsule and Gerota's fascia may result in local pain. Diagnosis is made using ultrasonography or CT, which shows

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unilateral or bilateral fluid accumulation[3].Although the mechanism underlying spontaneous urinoma remains unknown, poor lymphatic drainage because of chronic analgesic use may have been the main cause in our patient. Exudative fluid and cellular infiltration into the interstitium are the hallmark of interstitial nephritis[7].In glomerulonephritis with nephrotic syndrome, urinoma occurs because of lymphangiectasia[3].Studies have shown that lymphangiectasis is the main coexisting factor for spontaneous urinoma, indicating that lymphatic vessels are crucial for the prevention of fluid accumulation[3].In inflammatory disease, lymphatic vessels are important for edema clearance. When lymphatic vascular development is hindered, the tissue edema cannot be relieved and fluid accumulation occurs in a perirenal area[8].Inflammatory cytokines are critical for lymphatic vessel development. Lyons *et al*. noted that cyclooxygenase 2 improved lymphatic vessel development in breast cancers[9].The application of aspirin also decreased sarcoma development by inhibiting angiogenesis and lymphangiogenesis[10].

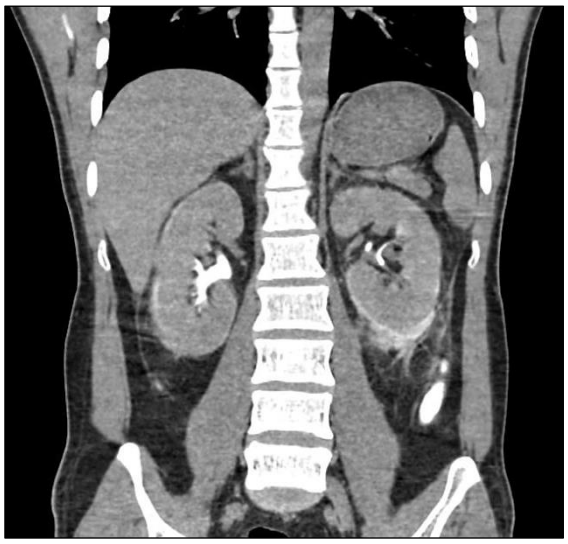

Fig 1: Delayed Excretory Urogram Coronal CT shows bilateral perinephricurinoma

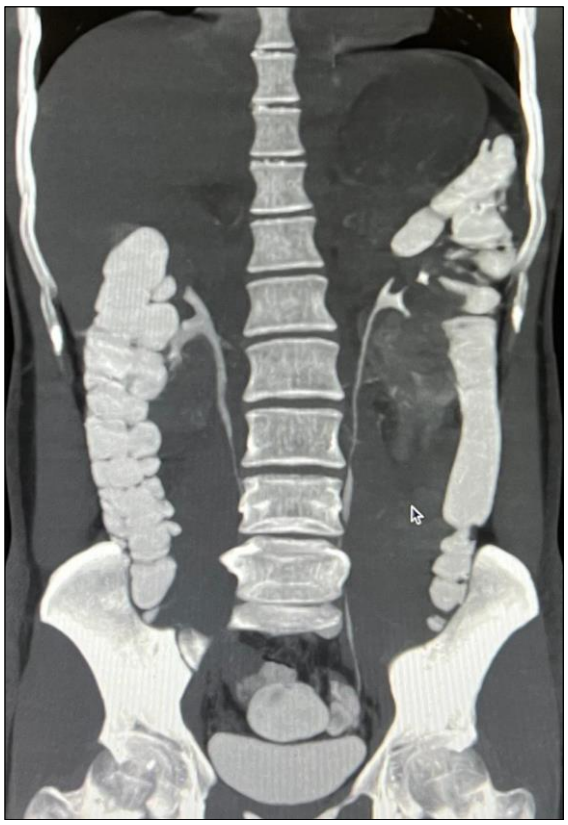

Fig 2: MIP Images showing normal excretory function of both kidneys

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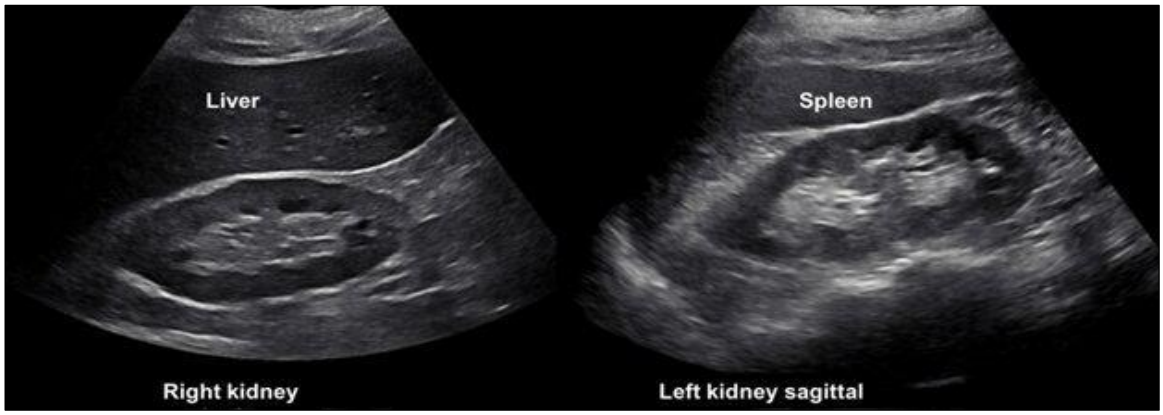

Fig 3: Follow up Ultrasound shows normal appearance of both kidneys without perinephricurinoma

Treatment of subcapsular effusion in nephrotic syndrome is achieved best by treating the underlying kidney disease and draining the subcapsular fluid if necessary[4].Simple aspiration of the fluid if the accumulation is moderate and control of the underlying glomerulopathy may suffice. There are three grades of perirenal fluid on ultrasonography:

Grade 1:A thin layer of perirenal fluid.

Grade 2:A moderate amount of collected perirenal fluid with indentations of the renal parenchyma and strands in the fluid.

Grade 3:A large fluid collection surrounding the kidney.

Severity of flank pain may not correlate with the amount of fluid accumulation in nephrotic syndrome. Therefore, in patients with glomerulonephritis and flank pain, subcapsular fluid accumulation should be considered. In contrast to posttraumatic subcapsularurinoma, there is still no consensus on the role of surgical drainage for spontaneous urinoma, unless the amount of fluid compresses the surrounding kidney or complications such as infection or hemorrhage occur. In most instances, small urinomas will reabsorb spontaneously and drainage is not necessary. Different treatment protocols to correct the underlying cause are outlined, such as drainage by percutaneous nephrostomy or placement of a stent across a ureteral defect to promote drainage of urine from the kidney into the bladder as well as drainage of the urinoma using appropriately positioned catheters[14].If the entire amount of fluid cannot be drained, treating the underlying disease without percutaneous drainage may be an alternative for regression of the subcapsular fluid accumulation.

In summary, spontaneous perirenalurinoma is a rare etiology for flank pain, and acute interstitial nephritis coexisting with lymphangiectasia is a contributing factor. In contrast to the treatment for traumatic urinoma, conservative treatment without percutaneous drainage should be the first-line management.

Written informed patient consent for publication has been obtained.

FINAL DIAGNOSIS: Non obstructive NSAID induced bilateral perinephricurinoma.

DIFFERENTIAL DIAGNOSIS: Bilateral pyelonephritis and other causes of obstructive/traumatic perinephricurinoma.

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