Acute leukemia masquerading as xanthogranulomatous pyelonephritis

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INTRODUCTION

Xanthogranulomatous pyelonephritis (XPN) is an unusual variant of chronic pyelonephritis that is often associated with renal stone disease or urinary tract obstruction. Clinical presentation includes fever, flank pain, dysuria, malaise, weight loss, or a palpable flank mass. Blood biochemistry may reveal azotemia and abnormal liver function test. The most typical laboratory findings are anemia and leukocytosis. Removal of the xanthogranulomatous inflammatory tissue is the mainstay treatment. However, there have been some reports of medical management of XPN.[1] Herein, we describe a case presenting as XPN in a leukemia patient.

CASE REPORT

An 82-year-old male presented with left-sided flank pain along with high-grade fever with chills and dysuria for 5 days. He was normoglycemic, normotensive but a known case of chronic obstructive airway disease and on regular bronchodilators. He underwent transurethral resection of the prostate for benign hypertrophy of prostate 10 years back. His physical examination revealed poor general condition with pallor and tachycardia. On abdominal examination, a palpable tender lump of 8 cm × 5 cm was found in the left upper quadrant area. He did not have any clinically palpable lymphadenopathy or hepatosplenomegaly.

Key Words: Acute prolymphocytic leukemia, masquerading, xanthogranulomatous pyelonephritis
Urinalysis revealed occult blood (++), protein (+), and nitrite (+) along with plenty of red blood cells, numerous white blood cells, and many bacteria per high-power field. Urine culture had grown *Escherichia coli* with resistance to quinolones and sensitivity to aminoglycosides and carbapenems. Complete blood count on admission showed a white cell count of 58,000/mm³, with 70% blast cells and 20% neutrophils, 8% lymphocytes, and 2% monocytes. Hemoglobin of 7.5 g/dl, hematocrit of 23.8%, and platelet count of 40,000/mm³. Blood biochemistry showed blood urea nitrogen of 46 mg/dl and a creatinine of 1.5 mg/dl, serum glucose of 112 mg/dl with normal liver function test. Ultrasonography showed a heterogeneously dense left kidney with calculus inside with normal right kidney. Contrast-enhanced computed tomography of abdomen revealed a diffusely enlarged left kidney with hypointense areas with staghorn calculus, fibro-fatty tissue proliferation with perinephric and paranephric fat stranding suggestive of XPN [Figures 1 and 2]. On further evaluation, peripheral blood smear examination revealed large atypical mononuclear cells with large nuclei and open chromatin and prominent nucleoli resembling blast cells and the overall picture suggestive of acute prolymphocytic leukemia. A bone marrow study was done, and imprint smear of bone marrow biopsy showed 24% lymphocytes, neutrophil of 1%, prolymphocytes of 75% with scattered smudge cells and grossly reduced platelets [Figure 3]. The bone marrow biopsy showed hypercellular marrow with decreased fat space according to the age. Erythroid, myeloid and megakaryocyte series are completely replaced by monotonous lymphocytes cell population. Some cells show scanty and moderate amount of cytoplasm and prominent nuclei. The overall picture is suggestive of prolymphocytic leukemia [Figure 4].

He was started on conservative management with intravenous (IV) antibiotics (IV meropenem with renal dose adjustments) and supportive treatment. Because of the
The patient’s poor general condition and in the background setting of acute leukemia, a surgical intervention was deferred and was decided to continue on supportive management. His general conditions deteriorated, chest condition worsened and had to be shifted to the intensive therapy unit. Gradually, he went into multi-organ failure and succumbed to the disease. This is a classic case of acute leukemia masquerading as XPN.

**DISCUSSION**

XPN is a rare infective condition of the kidney leading to diffuse destruction of renal parenchyma and occurs in the presence of chronic obstruction and suppuration. The urinary tract infection with obstructing renal calculi leads to diffuse or focal renal destruction, which usually starts in the pelvis and calyces and subsequently spreads to the adjacent renal parenchyma. If it is not controlled by then, it spreads to the adjacent tissue and destroys them. It presents with nonspecific symptoms of fever with chills and rigors, ipsilateral flank pain, loss of weight, malaise, dysuria, or palpable mass in the renal region. The laboratory results and complete blood counts show decreased hemoglobin with leukocytosis along with elevated serum C-reactive protein levels, erythrocyte sedimentation rate, or liver enzyme. Although not uniformly present, pyuria and bacteriuria may be a common finding. About a third of the patients may have sterile urine, and the causative organism may only be found in the tissue cultures. Positive urine culture is present in about 70% of patients. Although *E. coli* and Proteus species are mostly isolated organisms in the urine, Staphylococcus and Pseudomonas can also be isolated. Lipid-laden macrophage (xanthoma cells) present in the final histopathology of the resected specimen is confirmatory of the diagnosis of XPN though these cells can resemble clear cells of renal cell adenocarcinoma. XPN can be confused with other chronic inflammatory.

Condition of the kidney and malignancy and definitive diagnosis is confirmed only by histopathological examination. Ultrasonography patterns of XPN correspond approximately to that of a solid mass and often have an inhomogeneous echo. There is also a report that on ultrasonography XPN is suggested by parenchymal thinning and hydronephrosis, sonographic signs of chronic obstructive uropathy caused by stones, echoes in the dilated collecting system and a perinephric fluid collection. Contrast-enhanced computed tomography has made the radiological diagnosis of XPN easy with characteristic features including a large calculus in the renal collecting system with absent contrast excretion, spherical areas arranged in a hydronephrotic pattern, with higher attenuation than urine and no enhancement, large lesions with ill-defined borders and extension beyond the expected confines of the kidney, preservation of the reniform outline, and enhancing rims surrounding the spherical low-density areas.

In our case, the initial presenting finding was suggestive of XPN. However, this was in a background of leukemia. The blood picture and the bone marrow examination were suggestive of prolymphocytic leukemia. The two diseases simultaneously presenting with XPN masquerading the features of acute leukemia is a rare occurrence with only a few reports in the literature. The diagnosis of leukemia is confirmatory from the bone marrow studies. The underlying immunocompromised state in the background of prolymphocytic leukemia may be a cause of the abnormal inflammatory response in the form of XPN. The treatment protocol for such a situation is a dilemma, as to which one to treat first and how much aggressive the treatment planning could be sketched out in the background of hematological malignancy. As in our case, in consultation with the oncologist, a more conservative approach was decided on in view of the poor general condition and advanced age of the patient. We decided to treat the XPN conservatively with IV antibiotics and the best supportive care in an intensive care unit and to address leukemia after settling the XPN. However, the patient developed multi-organ failure and expired.

**CONCLUSION**

XPN can rarely masquerade hematological malignancies. Since it is a relatively rare occurrence, the treatment protocol is not clear in the literature. More such cases needed to be reviewed to finally formulate a treatment guideline for the above.

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**Conflicts of interest**

There are no conflicts of interest.

**REFERENCES**

1. Brown PS Jr., Dodson M, Weinrub PS. Xanthogranulomatous pyelonephritis: Report of nonsurgical management of a case and review of the literature. Clin Infect Dis 1996;22:308-14.
2. Korkes F, Favoretto RL, Bróglio M, Silva CA, Castro MG, Perez MD. Xanthogranulomatous pyelonephritis: Report of nonsurgical management of a case and review of the literature. Urology 2008;71:178-80.
3. Eastham J, Ahlering T, Skinner E. Xanthogranulomatous pyelonephritis: Clinical experience with 41 cases. Urology 1994;43:295-9.
4. Nataluk EA, McCullough DL, Scharling EO. Xanthogranulomatous pyelonephritis, the gatekeeper’s dilemma: A contemporary look at an old problem. Urology 1995;45:377-80.
5. Papadopoulos I, Wirth B, Wand H. Xanthogranulomatous pyelonephritis associated with renal cell carcinoma. Report on two cases and review of the literature. Eur Urol 1990;18:74-6.
6. Das DP, Pal DK. Co-existing malakoplasia and xanthogranulomatous
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pyelonephritis of kidney: Two different spectrum of same disease process. Urol Ann 2016;6:252-4.
7. Tiu CM, Chou YH, Chiou HJ, Lo CB, Yang JY, Chen KK, et al. Sonographic features of xanthogranulomatous pyelonephritis. J Clin Ultrasound 2001;29:279-85.
8. Rajesh A, Jakanani G, Mayer N, Mulcahy K. Computed tomography findings in xanthogranulomatous pyelonephritis. J Clin Imaging Sci 2011;1:45.
9. Wen YK. An unusual case of xanthogranulomatous pyelonephritis in a leukemia patient. Clin Nephrol 2008;70:255-8.
10. Ho CI, Wen YK, Chen ML. Xanthogranulomatous pyelonephritis successfully treated with antibiotics only. J Chin Med Assoc 2008;71:643-5.