Bilateral Acute Pyelonephritis in an Adult Nigerian

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Abstract

Introduction: Pyelonephritis is a common urinary tract infection. It can be acute or chronic. Most cases of acute pyelonephritis are usually unilateral but can be bilateral in certain conditions that pose as a significant risk factors like immunosuppressive states and renal calculi. If not well taken care of it can progress to chronic pyelonephritis (leading to chronic kidney disease) or urosepsis.

Method: A 52-year-old lady presented with fever, loin pains and diarrhea of 1week duration. She had dysuria and frequency of urination. She was diagnosed diabetic about 10years ago and on Oral hypoglycemic agents. Clinical examination revealed an acutely ill woman who was febrile with bilateral renal angle tenderness.

Result: Urinalysis revealed a pH of 8.0, specific gravity of 1.025, protein 2+, Leucocytes 3+, blood 1+, others were negative. Urine m/c/s showed numerous pus cells, some RBC casts and a few WBC casts; culture yielded growth of klebsiella and proteus species. Full blood count revealed leucocytosis with relative neutropenia.

Conclusion: bilateral acute pyelonephritis is not so uncommon. The risk factors include DM, HIV, Calculi, Structural abnormality, male sex and elderly age. As such a thorough investigation around these possible risk factors should be entertained in a patient with bilateral loin pains. This is to ensure proper counseling and treatment of patients.

Keywords: pyelonephritis, diabetes, urinalysis

I. Introduction

Pyelonephritis is an ascending urinary tract infection that has reached the pelvis of the kidney. The most common etiological cause includes *Escherichia coli*, *Proteus*, *Klebsiella* and *Pseudomonas*. Urine analysis and microscopy and culture is important in making a diagnosis. Antibiotic therapy especially with the quinolones is the treatment of choice.

II. Case Report

Mrs. E.J was a 52year old secondary school teacher who presented with fever of one week duration, loin pains also of one week duration and frequent stooling of five days duration. She was well until about 1 week prior to presentation when she developed fever which was continuous, high grade in nature and associated with chills and rigors. There was also associated history of vomiting and anorexia and also generalized body pains. At about the same time, she developed a left loin pain which was severe in nature. Patient also had right loin pain, though not as severe as the left. There was also associated suprapubic and epigastric pain. Pain was relieved by taking paracetamol. There was no known aggravating factor and no history of trauma to the affected region. About 2 days later she was noticed to have developed diarrhea which was watery and not blood stained. The estimated volume was about 400mls daily. There was associated history of dysuria and increased frequency of urination. There was a positive history of polyuria and polydepsia. She is a known diabetic diagnosed about 10 years ago and had been on oral hypoglycaemic agents. She is not a known hypertensive or sickle cell disease patient. She is the first of 4 children in a monogamous setting. She does not smoke cigarette neither does she take alcohol.

General examination revealed an acutely ill looking young woman, dehydrated⁺⁺, not pale, febrile (39.5°C), anicteric, acyanosed and nil pitting pedal edema. Cardiovascular system examination revealed a pulse rate of 116beats per minute, regular and normal volume. The blood pressure was 100/70mmHg and the jugular venous pressure was not elevated. Apex beat was at the 5th left intercostals space mid-clavicular line. The heart sounds were S1, and S2. Chest examination revealed a respiratory rate of 26cycles per minute. Trachea was central, percussion notes resonant. She had vesicular breath sounds with no added sounds. The abdomen was full, moved with respiration. The epigastrium was tender. There was also suprapubic tenderness. Renal angle tenderness was positive, more on the left but there was no organomegaly. There was also no ascites. She was conscious and alert, well oriented in time, place and person. There were no signs of focal neurological deficit A diagnosis of bilateral acute pyelonephritis in a known diabetic was made.

Investigations were as follows: Urinalysis revealed a pH of 8.0, specific gravity of 1.025, protein 2+, Leucocytes 3+, blood 1+, others were negative. Urine m/c/s showed numerous pus cells, some RBC casts and a few WBC casts; culture yielded growth of klebsiella and proteus species.

Full blood count revealed a total white blood cell count of $16,000/\text{mm}^3$ with 90% neutrophils and 10% lymphocytes. Platelet count was $250000/\text{mm}^3$. The PCV was 34% and ESR was 19mm/hr. The viral studies (RVS, HCV, and HBsAg) were all negative. Serum Urea and Creatinine were 21mg/dl and 0.7mg/dl respectively. Serum electrolytes revealed sodium, potassium, bicarbonates, and chloride of 132mmol/L, 3.7mmol/L, 20mmol/L, and 94mmol/L respectively. Random blood sugar was 302mg/dl and a fasting blood sugar done the following day was 179mg/dl. Lipid profile revealed total cholesterol 190mg/dl; HDL – C, LDL – C and TG were 44mg/dl, 158mg/dl and 132mg/dl respectively.

The chest x-ray was essentially normal and renal USS scan revealed kidney sizes of 12.40 x 5.80cm and 11.70 x 5.40cm for the right and left respectively. Both shows normal cortical echogenicity and corticomedullary differentiation with no evidence of stones or abscesses. She was placed on Intravenous fluids 500mls of 0.9% normal saline 4 hourly. Intramuscular injection of diclofenac 75mg was given as a stat dose and this was followed by 100mg tablet diclofenac daily with food for the next one week. She also had tablets vitamin C 100mg TDS. Intravenous antibiotics (ceftriaxone 1g daily and metronidazole 500mg 8hourly) were also administered.

In view of her uncontrolled blood sugar, she had subcutaneous pre-meal soluble insulin, 10IU in the morning after breakfast, 14IU in the afternoon after lunch and 8IU in the evening after dinner. She was placed on diabetic diet of 1800kcal/dayShe was on admission for 1 week and discharged after satisfactory improvement both clinically and biochemically. Random blood sugar on discharge was 136mg/dl and was advised on follow-up in clinic.

III. Discussion

Pyelonephritis is an ascending urinary tract infection that has reached the pelvis of the kidney. It is a form of nephritis and can also be called pyelitis¹. In the United States, approximately 250,000 cases of acute pyelonephritis is recorded each year. The most common etiological cause is *Escherichia coli* in about 80% of cases². In the elderly, *E. coli* is a less common cause and accounts for less than 60% of cases. Other causative organisms in the elderly and immunocompromised patients include *Proteus*, *Klebsiella* (like in the case of this patient) and *Pseudomonas*. Most cases of community acquired pyelonephritis are due to bowel organisms that enter the urinary tract e.g. *E. coli* in 70 – 80% of cases and *Enterococcus faecalis*. Hospital acquired infection are due to organisms like *Klebsiella* and *Pseudomonas*.

Acute pyelonephritis occurs in 1 - 2% of pregnant women increasing the risk of premature labour and low birth weight³. Most renal parenchymal infections occurs following bacteria ascent through the urethra and urinary bladder. In men, prostatitis and benign prostatic hyperplasia predisposes to bacteraemia⁴. Haemategenous acute pyelonephritis occurs most often in debilitated, chronically ill looking patients and those receiving immunosuppressive treatment. It is usually unilateral but can be bilateral when there are risk factors as mentioned below. Patients with diabetes mellitus tend to have infections caused by *Klebsiella* (as in this case), enterobacter and clostridium or candida. They are also at increased risk of developing emphysematous pyelonephritis and papillary necrosis leading to shock and renal failure⁵. Acute pyelonephritis occurs within 2 months following renal transplant in 30 to 50% of patients because of concomitant immunosupression and post surgical vesicouretonic reflex⁶. Acute pyelonephritis is considered complicated in men because they have a higher probability of urinary tract abnormalities, benign prostatic hyperplasia and age related decrease of antimicrobial activities in the prostatic secretions.

The clinical features are a wide spectrum ranging from mild illness to sepsis syndrome⁷. It is important to note that most cases of pyelonephritis starts from lower urinary tract infection e.g. cystitis and prostatis. Usually, the patient present with dysuria, abdominal pains, radiating to the back on the affected side, tenderness in the suprapubic area and a positive renal punch test (costovertebral angle tenderness), all of which our patient presented with. In many cases, systemic symptoms such as fever associated with chills and rigors, headache and vomiting are seen. In severe cases, pyelonephritis delirium may be present¹. Severe cases of pyelonephritis can lead to sepsis. When pyelonephritis or other urinary tract infection leads to sepsis, it is termed urosepsis.

Diagnosis is made from evidence of urinary tract infection from urinalysis or culture along with signs of upper urinary tract infection (fever, chills, flank pains and costovertebral angle tenderness). The risk factors for complicated cases of acute pyelonephritis (or bilateral acute pyelonephritis) include immunosuppressive states (Diabetes mellitus, HIV etc.) catheters in situ, urinary calculi, vesicoureteric reflux anatomical abnormalities, elderly, infants and male sex. In a study of young and middle aged women presenting to an emergency department with fever, pyuria and other features of upper urinary tract infections, 98% had acute pyelonephritis⁸. Up to 30% of elderly patients with acute pyelonephritis have no fever. Urinalysis and culture confirms the diagnosis of acute pyelonephritis. A consensus definition of pyelonephritis established by the infectious disease society of America (IDSA) is a urine culture showing at least 10,000 CFU/mm³ and symptoms compatible with diagnosis. Urine specimen generally is obtained by a midstream clean catch technique and one study showed that cleansing does not decrease contamination rates in adults⁹. Positive

leucocyte esterase test or nitrite test is very important in making diagnosis. Haematuria may be present in patients with cystitis and pyelonephritis. Urine culture is positive in 90% of cases and specimen should be collected before initiation of antibiotics. Blood culture has been recommended in some cases but studies have shown that it however did not result in changes in the management strategies of acute pyelonephritis patients. Leucocyte esterase test and nitrite test are 74 - 96% and 35 - 88% specific respectively and 94 - 98% and 92 - 100% sensitive respectively¹⁰.

Antibiotic therapy is the treatment. Outpatient treatment with oral antibiotics can be given to patients with uncomplicated acute pyelonephritis¹¹. Those with complicated cases like our patient are admitted and given intravenous antibiotics initially for a couple of days and follow-up with oral drugs for about 2 weeks duration. Fluroquinolones are the antibiotics of choice. The resistance to fluroquinolones remains very low $1 - 3\%^{12}$. They are absorbed very well from the gastrointestinal tract and have excellent kidney functions. Acceptable alternatives include amoxicillin – clavulinic potassium and trimethoprim – sulfamethaxazoli. Quinolones are avoided during pregnancy and augmentin used instead. The common causes of initial treatment failure are resistant organisms and nephrolithiasis.

References

- [1]. Ramakrishnan K, Scheid DC (2005). "Diagnosis and management of acute pyelonephritis in adults". Am Fam Physician 71 (5): 933–42.
- [2]. Stamm WE, Hooton TM. Management of urinary tract infections in adults. N Engl J Med. 1993;329:1328–34.
- [3]. Gilstrap LC 3d, Ramin SM. Urinary tract infections during pregnancy. Obstet Gynecol Clin North Am. 2001;28:581-91
- [4]. Stamm WE. Urinary tract infections and pyelonephritis. In: Harrison TR, Braunwald E, eds. Harrison's Principles of internal medicine. 15th ed. New York: McGraw-Hill, 2001:1620–6.
- [5]. Roberts JA. Management of pyelonephritis and upper urinary tract infections. Urol Clin North Am. 1999;26:753-63.
- [6]. Bergeron MG. Treatment of pyelonephritis in adults. Med Clin North Am. 1995;79:619–49.
- [7]. Bass PF 3d, Jarvis JA, Mitchell CK. Urinary tract infections. Prim Care. 2003;30:41-61.
- [8]. Pinson AG, Philbrick JT, Lindbeck GH, Schorling JB. Fever in the clinical diagnosis of acute pyelonephritis. Am J Emerg Med. 1997;15:148–51.
- [9]. Lifshitz E, Kramer L. Outpatient urine culture: does collection technique matter? Arch Intern Med. 2000;160:2537–40.
- [10]. Pollock HM. Laboratory techniques for detection of urinary tract infection and assessment of value. Am J Med. 1983;75:79–84.
 [11]. Bach D, van den Berg-Segers A, Hubner A, van Breukelen G, Cesana M, Pletan Y. Rufloxacin once daily ciprofloxacin twice daily in the treatment of patients with acute uncomplicated pyelonephritis. J Urol. 1995;154:19–24.
- [12]. Nicolle LE. Urinary tract infection: traditional pharmacologic therapies. Am J Med. 2002;113(suppl 1A):35S–44S.