Emphysematous pyelonephritis

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ABSTRACT

Urinary tract infection is very common and in most cases is self limiting. However, in some cases, the infection may progress and leads to serious complication requiring admission and treatment. Emphysematous pyelonephritis is a rare but serious complication of urinary tract infection. The symptoms are similar to those seen in pyelonephritis and without radiological imaging, the diagnosis can be missed with serious consequences. In severe cases, nephrectomy may be indicated. We report a case of emphysematous pyelonephritis in a previously well obese 28-year-old lady who was successfully managed with intravenous antibiotic. Her underlying risk factor was undiagnosed diabetes mellitus.

Keywords: Abscess, gas forming abscess, renal abscess, pyelonephritis

INTRODUCTION

Emphysematous pyelonephritis (EPN) is a rare life threatening condition caused by gas forming organisms. Symptoms of EPN resemble that of acute pyelonephritis and unless there is imaging, diagnosis of EPN will be missed. [1] Treatment includes intensive antimicrobial therapy with or without drainage and in some cases may require nephrectomy.

CASE REPORT

A 28-year-old obese lady presented to the emergency department with a two day history of dyspnoea, fever, lower chest pain and a one day history of vomiting. She also had a cough and mild haemoptysis. On further questioning she admitted to having intermittent right flank pain for the past one week that was associated with decreased urine output, dysuria and haematuria. Her past medical history was significant for bronchial asthma that was well controlled. On examination, she was alert, but had tachycardia (140 beats per minute), mild sclera icterus and diaphoresis. Blood pressure was normal. Abdominal examination showed left upper quadrant tenderness but was otherwise normal. Blood investigations showed renal impairment with serum urea of 14.6mmol/L (range 5-7.5), serum creatinine of 277 µmol/L (range 35-88), hyperglycemia of 17.1 mmol/L, elevated serum C-reactive protein (CRP) of 39.26mg/L and deranged liver profiles with mildly elevated total serum bilirubin of

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44mmol/L and elevated serum GGT of 113. Urine was cloudy with negative leukocytes. There was presence of protein at 1 gm/l (++) and urobilinogen (++). Urine microscopic examination shows WBC (+++) (27-36 hpf) and "packed" (red blood cells). Ultrasound of kidneys were normal. She was started empirically on intravenous amoxicillin/clavulanic acid (1.2 gm b.i.d) and ceftazidime (1 gm t.i.d) to cover for melioidosis which is common in our setting.

The breathlessness improved overnight and she could mobilise the following day. Over the next few days, her condition improved. Blood culture on admission had isolated *Klebsiella pneumoniae*, resistant to ampicillin but sensitive to all other tested antibiotics. The antibiotic was de-escalated and ceftazidime was stopped. Despite appropriate antibiotics and general improvement, she continued to have intermittent temperature spikes to a maximum of 38°C. Her condition suddenly deteriorated with rigors, hypotension (95/45 mmHg) and tachycardia.

A computed tomography scan (CT) showed mild hydronephrosis of the right kidney and a calculus (1.7 x 1.0 cm) in the right ureter. The right kidney was also markedly swollen with air bubbles noted in the infralateral aspect. The imaging findings were that of EPN. The patient was referred to urology and intravenous aminoglycoside (Netilmicin 150 mg t.i.d) was added to her management with good effects. Three days later, the patient became afebrile and was discharged with endocrine follow up for her diabetes mellitus. However, the patient defaulted at clinic follow up. When last contacted by telephone, she had reported to be well.

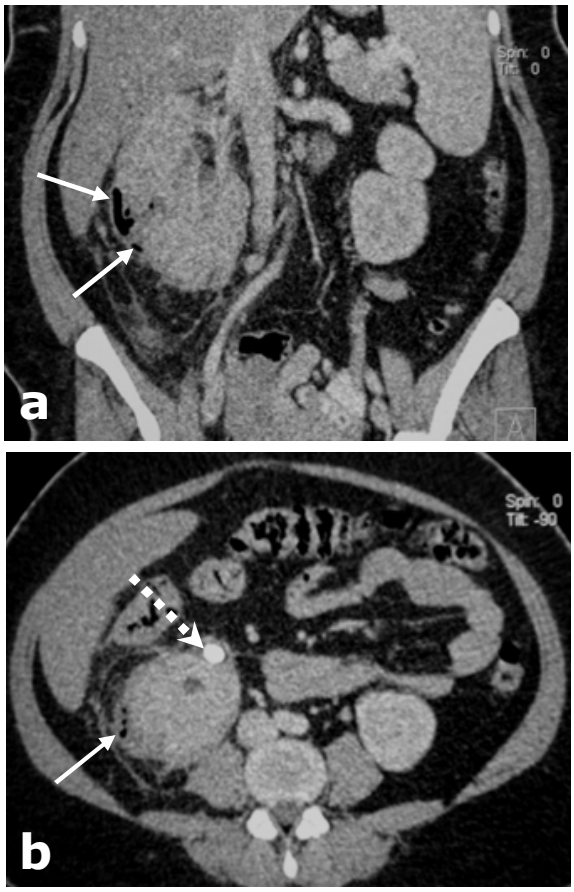

Fig. 1: a) Reconstructed computed tomography scan showing swollen right kidney and air pockets (arrows) in the inferior lateral border, b) Axial computed tomography scan showing calculus (broken arrow) and air pockets (arrow).

DISCUSSION

EPN is rare and is associated with significant morbidity and mortality (18.8 to 25%) compared to non-EPN. The risk factors associated with high mortality include bilateral involvement, significant renal parenchymal necrosis with either no fluid content or a streaky/ mottled gas pattern on imaging, conservative therapy (fluid resuscitation and antimicrobial), presence of thrombocytopenia and septic shock. [1, 2] Some advocate early surgical therapy. However in milder cases such as ours, appropriate intensive antimicrobial therapy may be adequate.

EPN is strongly associated with poorly

controlled diabetes mellitus and is more common in females with a gender ratio of 6:1. [1, 3] The presence of hyperglycemia in poorly controlled diabetes provides a microenvironment conducive to the development of this condition when the infection is caused by appropriate organisms. This has been well documented in literature reporting on gas forming pyogenic liver abscesses. [4] Apart from gas forming anaerobes such as the Clostridium species, *Klebsiella* species and *Eschericeria coli* are the two most commonly implicated organisms. These two organisms can produce formic hydrogenlyase, an enzyme required in the production of gas in the presence of an acidic environment and hyperglycemia.

Clinical presentations of EPN are non-specific and resemble those of non-emphysematous acute pyelonephtirtis. Fevers, chills, flank or abdominal pain, nausea, and vomiting are common. In severe cases, patients may present with fulminant sepsis leading to multi-organ failures especially if both kidneys are involved. Imaging typically show enlarged kidney with gas pockets that can involve the various parts of kidney. The findings on CT imaging have been used to classify and predict the prognosis of EPN. [1] The classification categorise EPN into five classes depending on location of gas and whether one or both kidneys are involved. Class 1 represents the presence of gas in the collecting system only (i.e. emphysematous pyelitis), Class 2, the presence of gas in the renal parenchyma without extension to the extra-renal space, Class 3A, extension of gas or abscess to the peri-nephric space (defined between the fibrous renal capsule and the renal fascia), Class 3B, extension of gas or abscess to the para-renal space, (defined as the space beyond the renal fascia and/or extension to adjacent tissues such as the psoas muscle) and finally Class 4 with bilateral EPN or a solitary functioning kidney with EPN.

Due to high morbidity and mortality [2], diagnosis need to be made early and appropriate aggressive therapies started as soon as possible. Approaches to management have also been suggested according to the above referenced classification. [1, 5] Appropriate antibiotics may be adequate for Class 1 without abscess or obstruction. Percutaneous catheter drainage is recommended for Class 1 category with abscess or obstruction, Class 2 or low risk Class 3A or B categories (with either none or one of the following risk factors: thrombocytopenia, acute renal failure, impaired consciousness or shock). Nephrectomy therapy needs to be considered for those who had failed percutaneous drainage or who have two or more of the stated risk factors. Generally nephrectomy should only be considered if antibiotic and drainage procedures have failed and this is especially applicable for those with Class 4 disease.

In conclusion, EPN is a very rare complication of urinary tract infections. Index of suspicion needs to be high for early diagnosis and it is particularly important that appropriate empiric therapies are started early to avoid progression that may ultimately require nephrectomy.

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