A Case Of Pyelonephritis In A Primiparous Indian Woman Following An Uneventful Caesarean Section

ABSTRACT

Acute pyelonephritis is a bacterial infection causing inflammation of the kidneys and is one of the most common diseases of the kidney. Pyelonephritis occurs as a complication of an ascending urinary tract infection (UTI) which spreads from the bladder to the kidneys and their collecting systems. Symptoms usually include fever, flank pain, nausea, vomiting, burning on urination, increased frequency, and urgency. The 2 most common symptoms are usually fever and flank pain. Acute pyelonephritis can be divided into uncomplicated and complicated. Complicated pyelonephritis includes pregnant patients, patients with uncontrolled diabetes, kidney transplants, urinary anatomical abnormalities, acute or chronic kidney failure, as well as immunocompromised patients, and those with hospital-acquired bacterial infections. It is important to make a distinction between complicated and uncomplicated pyelonephritis, as patient management and disposition depend on it. [1]

The main cause of acute pyelonephritis is gram-negative bacteria, the most common being *Escherichia coli*. Other gram-negative bacteria which cause acute pyelonephritis include *Proteus, Klebsiella, and Enterobacter*. In most patients, the infecting organism will come from their fecal flora. Bacteria can reach the kidneys in 2 ways: hematogenous spread and through ascending infection from the lower urinary tract. Hematogenous spread is less common and usually occurs in patients with ureteral obstructions or immunocompromised and debilitated patients. Most patients will get acute pyelonephritis through ascending infection. Ascending infection happens through several steps. Bacteria will first attach to urethral mucosal epithelial cells and will then travel to the bladder via the urethra either through instrumentation or urinary tract infections which occur more frequently in females. UTIs are more common in females than in males due to shorter urethras, hormonal changes, and close distance to the anus. Urinary tract obstruction caused by something such as a kidney stone can also lead to acute pyelonephritis. An outflow obstruction of urine can lead to incomplete emptying and urinary stasis, which causes bacteria to multiply without being flushed out. A less common cause of acute pyelonephritis is vesicoureteral reflux, which is a congenital condition where urine flows backward from the bladder into the kidneys.[1]

Acute pyelonephritis can have several complications such as renal or perinephric abscess formation, sepsis, renal vein thrombosis, papillary necrosis, or acute renal failure, with one of the more serious complications being emphysematous pyelonephritis (EPN). Emphysematous pyelonephritis is a necrotizing infection of the kidney usually caused by *E. coli* or *Klebsiella pneumoniae* and is a severe complication of acute pyelonephritis. EPN is usually seen in the setting of diabetes and occurs more frequently in women.[1]

Keywords: Acute pyelonephritis, E.coli, women, immunocompromised

I. INTRODUCTION

Acute pyelonephritis is a sudden and severe kidney infection. It causes the kidneys to swell and may permanently damage them. Pyelonephritis can be life-threatening. Symptoms usually appear within two days of infection. Common symptoms include a fever greater than 102°F (38.9°C), pain in the abdomen, back, side, or groin, painful or burning urination, cloudy urine, pus or blood in the urine, urgent or frequent urination and fishy-smelling urine[2].

E. coli is the most common bacteria causing acute pyelonephritis due to its unique ability to adhere to and colonize the urinary tract and kidneys. *E.coli*has adhesive molecules called P-fimbriae, which interact with receptors on the surface of uroepithelial cells. Kidneys infected with *E. coli* can lead to an acute inflammatory response which can cause scarring of the renal parenchyma. Though the mechanism in which renal scarring occurs is still poorly understood, it has been hypothesized that the adhesion of bacteria to the renal cells disrupts the protective barriers, which leads to localized infection, hypoxia, ischemia, and clotting in an attempt to contain the infection. Inflammatory cytokines, bacterial toxins, and other reactive processes further lead to complete pyelonephritis and, in many cases, systemic symptoms of sepsis and shock[1].

Histopathology will usually reveal necrosis or putrid abscess formation within the renal parenchyma. The renal tissues are infiltrated with neutrophils, macrophages, and plasma cells. However, the architecture is not completely disorganized[1].

A good history and physical is the mainstay of evaluating acute pyelonephritis, but laboratory and imaging studies can be helpful. A urinary specimen should be obtained for a urinalysis. On urinalysis, one should look for pyuria as it is the most common finding in patients with acute pyelonephritis. Nitrite production will indicate that the causative bacteria is *E.coli*. Proteinuria and microscopic hematuria may be present as well on urinalysis. If hematuria is present, then other causes may be considered, such as kidney stones. All patients with suspected acute pyelonephritis should also have urine cultures sent for proper antibiotic management. Blood work such as a complete blood cell count (CBC) is sent to look for an elevation in white blood cells. The complete metabolic panel can be used to search for aberrations in creatinine and BUN to assess kidney function. The imagining study of choice for acute pyelonephritis is abdominal/pelvic CT with contrast. Imaging studies will usually not be required for the diagnosis of acute pyelonephritis but are indicated for patients with a renal transplant, patients in septic shock, those patients with poorly controlled diabetes, complicated UTIs, immunocompromised patients, or those with toxicity persisting for longer than 72 hours. Ultrasonography can be used to detect pyelonephritis, but a negative study does not exclude acute pyelonephritis. Regardless, ultrasound can still be a useful study when evaluating for acute pyelonephritis because it can be done bedside, has no radiation exposure, and may reveal renal abnormalities, which can prompt further testing or definitive treatment[1].

Acute pyelonephritis can be managed either as an outpatient or inpatient. Healthy, young, non-pregnant women who present with uncomplicated pyelonephritis can be treated as outpatients. Inpatient treatment is usually required for those who are very young, elderly, immunocompromised, those with poorly controlled diabetes, renal transplant, patients, patients with structural abnormalities of the urinary tract, pregnant patients, or those who cannot tolerate oral intake. The mainstay of treatment of acute pyelonephritis is antibiotics, analgesics, and antipyretics. Nonsteroidal anti-inflammatory drugs (NSAIDs) work well to treat both pain and fever associated with acute pyelonephritis. The initial selection of antibiotics will be empiric and should be based on the local antibiotic resistance. Antibiotic therapy should then be adjusted based on the results of the urine culture. Most uncomplicated cases of acute pyelonephritis will be caused by *E. coli,* for which patients can be treated with oral cephalosporins or TMP-SMX for 14 days. Complicated cases of acute pyelonephritis require intravenous (IV) antibiotic treatment until there are clinical improvements. Examples of IV antibiotics include piperacillin-tazobactam, fluoroquinolones, meropenem, and cefepime. For patients who have allergies to penicillin, vancomycin can be used. Follow up for non-admitted patients for resolution of symptoms should be in 1 to 2 days. Follow up urine culture results should be obtained only in patients who had a complicated course and are usually not needed in healthy, non-pregnant women. Any patient that had a complicated UTI should be sent for follow up imaging to identify any abnormalities that predispose the patient to further infections[1].

II. CASE PRESENTATION

A 27-year-old postpartum primiparous female patient came to the emergency department with complaints of breathlessness since 3 days sudden in onset, gradually progressive, increased on rest. It was not associated with chest pain, cough, fever or sweating. She was very anxious about her condition. There were no similar complaints in the past. The patient had undergone lower segment caesarean section 5 days back with no complications. The baby was a male of birth weight 3.65 kg and it cried immediately after birth. On examination she was moderately built and nourished, conscious and oriented to time, place and person. She was afebrile, pulse 66 bpm, blood pressure 130/90 mm hg and respiratory examination and cardiovascular examination normal. Per abdomen shows distended abdomen, with a water proof dressing in the lower abdomen and on auscultation bowel sounds are heard. Head to neck examination reveals moderate pallour and bilateral pedal edema.

The patient's blood was drawn and sent for a complete blood count (CBC) , renal function test (RFT), liver function test (LFT), to evaluate her condition. There was a mild increase in total leukocyte count, neutrophils, with c- reactive protein being 103.7 mg/dl indicating an acute infection. The RFT revealed urea 50.2 mg/dl, creatinine 2.3 mg/dl, uric acid 9.7 mg/dl which were all above the normal range. LFT revealed albumin 3.0 g/dl(low), alkaline phosphatase 168.0 and gamma glutamyl transferase 48.8 U/L both of which are on the higher side of normal metrics. Creatinine levels were repeated everyday which showed a gradual decline over 4 days of admission from 2.6 to 1.2 mg/dl. The patient was suspected of having an acute urinary tract infection, specifically being pyelonephritis in the postpartum period.

The patient received treatment consisting of Inj. Paracetamol 1gm IV stat and BD(Paracip) , Nebulisation of ipratroipium bromide, levosalbutamol with budesonide stat(duolin and budecort) , Inj. Pantalrozole 40 mg stat and OD(Pan) Inj. Piperacillin and tazobactum(Tazocin), Tab. Oseltamivir 75 mg OD (Fluvir), Syp. Liquid paraffin 30 ml BD (Cremaffin), Biscodyl suppository stat (Dulcolax suppository), Inj. Furosemide 20 mg IV stat and OD (Lasix), Inj. Metronidazole 1gm IV BD (Metrogyl), Cap. Fluoxetine 20 mg OD (Fludac) and Syp. Disodium hydrogen citrate 2 tablespoon with water TID (Citralka). Rehydration was achieved by intravenous infusion (IVF) of Dextrose normal saline (DNS) at the rate of 30 ml/hr stat.

After her primary care in the emergency room, the patient was shifted to the ward. A gynaecologist and nephrologist opinion was taken and their advice was followed. Breast feeding was continued as advised by the gynaecologist. The patient's symptoms were resolved after 4 days of ward care. As she regained health and returned to normal state, the patient was discharged in a hemodynamically stable condition. The patient was advised to take postpartum care and follow the advice on discharge. Tab. Duonem 200 mg twice a day for 10 days (faropenem), Tab. Chymoral forte thrice a day for 5 days (a combination of trypsin and chymotrypsin), Cap. Fludac 20 mg once daily for 10 days (Fluoxetine), Tab. Dolo 650 mg whenever required (paracetamol) and Metrogyl-P ointment for local application (metronidazole) was given. The patient was also advised to get an RFT and CBC done before meeting the consultants.

III. DISCUSSION

Pyelonephritis is a bacterial infection of the renal parenchyma. It may occur postpartum if bacteria ascend from the bladder[3] . Ascending route of infection is the underlying cause found in the cases of acute pyelonephritis. Conditions that contribute to contamination of urine in the bladder include sex (short urethra in infant girls), age, poor personal hygiene, instrumentation and other potential factors.Vesicoureteral reflux (VUR) can be found in a significant number of young girls with urinary tract infections. This can further contribute to the retrograde spread of bacteria from the bladder to the kidney. The etiologic agent is Escherichia coli in more than 80% of cases of acute pyelonephritis, but other Gram-positive and Gram-negative bacteria can be found in some instances. Hematogenous acute pyelonephritis is most often diagnosed in incapacitated and

chronically ill patients, but also in those receiving immunosuppressive therapy. Fungal and staphylococcal infections may sometimes spread to the kidney from distant foci in the bone or skin.

The infection may begin as asymptomatic bacteriuria during pregnancy and is sometimes associated with bladder catheterization to relieve urinary distention during or after labor.[5]

A population-based study of acute pyelonephritis in the United States found overall annual rates of 15-17 cases per 10,000 females and 3-4 cases per 10,000 males. In women aged 18−49 years, the estimated incidence is 28 cases per 10,000 [4].

Complications of acute pyelonephritis include papillary necrosis, urinary tract obstruction, pyonephrosis, perinephric abscess. [6]

Treatment is adjusted accordingly and continued for a total of 7 to 14 days; oral antibiotics are used after the initial IV antibiotics.Women should be encouraged to consume large amounts of liquids.A urine culture should be repeated 6 to 8 weeks after delivery to verify cure. If episodes of pyelonephritis recur, imaging should be considered to look for calculi or congenital malformations. Imaging during pregnancy is usually with ultrasonography; imaging after pregnancy is usually with contrast CT. [3]

IV. CONCLUSION

In this case we describe a case of acute pyelonephritis after a lower segment caesarean section that was managed symptomatically with medications that decrease the infection by eradicating the organism which caused it. Identifying and reporting such cases is crucial to researching and developing management strategies for future reference.

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