Dominant Polycystic Kidney Disease with Acute Pyelonephritis due to Multi-drug Resistant Staphylococcus D Group and Candida albicans

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Autosomal dominant polycystic kidney disease (ADPKD) is a common hereditary kidney disease. Approximately, 85% of families with ADPKD have a mutation in PKD1 gene, located on chromosome 16; these people have the PKD1 disease. The remaining 15% have the mutation in PKD2 gene on chromosome 4; this is called PKD2 disease. In some cases, it is not possible to detect which gene is mutated. PKD2 disease is milder; therefore, it often presents later in life and is sometimes not diagnosed at all. Thus, it is likely that more than 15% of all people with ADPKD have PKD2 disease, without a family history of the disease.

ADPKD appears to cause abnormal cell growth that leads to cysts on the kidneys. In ADPKD, cyst formation begins as an expansion, or ballooning of a tubule in a small proportion of nephrons. The cyst subsequently enlarges, usually due to fluid secretion into the cyst, thereby replacing the normal kidney tissue and leading to progressive renal failure. Other problems involving kidney can occur, including high blood pressure, kidney infection (Pyelonephritis), blood in the urine (hematuria), and kidney stones.

INTRODUCTION

Autosomal dominant polycystic kidney disease (ADPKD) is a common hereditary kidney disease. Approximately, 85% of families with ADPKD have a mutation in PKD1 gene, located on chromosome 16; these people have the PKD1 disease. The remaining 15% have the mutation in PKD2 gene on chromosome 4; this is called PKD2 disease. In some cases, it is not possible to detect which gene is mutated. PKD2 disease is milder; therefore, it often presents later in life and is sometimes not diagnosed at all. Thus, it is likely that more than 15% of all people with ADPKD have PKD2 disease, without a family history of the disease.

Pyelonephritis is commonly observed in ADPKD due to bacterial invasion of the renal parenchyma. Elderly patients may present with typical manifestations of pyelonephritis, or they may experience fever, mental status change, flank and abdominal pain, and generalized deterioration.

In the present case, infection of Staphylococcus D group is observed, with resistance to routinely used antibiotics of choice. Staphylococci has developed more resistance to several antibiotics, as compared to other Gram-positive organisms. The widespread antibiotic resistance across various strains of Staphylococcus aureus or across different species of Staphylococcus has been attributed to horizontal gene transfer of genes encoding antibiotic/metal resistance and virulence. A new antibiotic adjuvant entity ceftriaxone/sulbactam/disodium edetate (ELORES) counteracts multi-drug resistant (MDR) pathogens by its synergistic activity and was considered a drug of choice for treating the present case. In a comparative study among different antibiotics cefepime, ceftriaxone, piperacillin + tazobactam, amoxicillin + clavulanic acid Elores, and ceftoperazone + sulbactam in plaktonic and sessile cells of Staphylococcus epidermidis and S. aureus showed ELORES to significantly reduce the minimum inhibitory concentration and minimum biofilm eradication concentration values. Additionally decreasing the over-expression of efflux pump, makes ELORES a preferable choice in treating overexpressing efflux pump and metallo-beta-lactamase (MBL) producing pathogens.
A 70-year-old male patient was admitted to our hospital with chief complaints of fever, decreased urine output, shortness of breath with swallowing on ankle and foot since 7 days. The patient is a known case of diabetes mellitus, hypertension, autosomal polycystic kidney disease and chronic liver disease, on irregular medications. On general examination, the patient was found to have pallor, pedal edema, and increased jugular venous pressure. His general condition was poor, febrile, pulse 112/min, blood pressure 100/60 mmHg. Systemic examination revealed bilateral normal vascular breath sounds with decreased bronchial sounds at bases. Cardiovascular system was within normal limit, S1S2 normal and no murmurs. Per abdomen examination revealed two finger firm spleen with free fluid. The patient was conscious oriented with no focal neurological deficit. Urine and blood samples were sent for laboratory investigation. Chest radiography revealed bilateral pleural effusion. Computerized tomography scan of abdomen showed chronic liver disease and bilateral polycystic kidney disease with pyelonephritic changes and no cyst infection. Based on clinical finding a provisional diagnosis of acute on chronic kidney disease; acute pyelonephritis was done.

Complete differential blood cell counts revealed: White blood cells 8600/mm³ (with 6200/mm³ neutrophils), red blood cells 4×10³/mm³, hematocrit 32%, hemoglobin 6.9 g%, platelets 12,6000/mm³ and raised erythrocyte sedimentation rate 29 Mm/FHR. Other laboratory results included raised blood urea nitrogen 73 mg/dl, raised serum creatinine 3.3 mg/dl, plasma Na⁺ 114 mEq/l, K⁺ 4.7 mEq/l, total bilirubin 1.9 mg/dl, with normal aspartate aminotransferase and alanine aminotransferase level, and hypoalbuminemia and globulinemia. Blood culture showed no growth. In urine, wet mount yealy budding cells (Candida albicans), plenty of pus cells and red blood cells were noted. On culture of urine sample, Streptococcus Group D was identified showing resistance to erythromycin, clindamycin, quinupristin, streptomycin and levofloxacin. Vancomycin, linezolid, and teicoplanin showed intermediate sensitivity while ELORES (ceftriaxone, sulbactam and disodium edetate) showed sensitivity. Histopathologist and physician’s advice was taken and followed. In the light of culture sensitivity test ELORES was administered i.v. 1.5 g, B.D. with 90 min infusion, for 7 days, along with injection fluconazole 400 mg every alternate day. Hemodialysis and isolated ultrafiltration sessions were carried along with other supportive therapy. The patient responded to the therapy and was discharged on the 7th day post admission.

**CASE REPORT**

**DISCUSSION**

Pyelonephritis is a common complication associated with ADPKD, extensively involving kidney parenchyma. Moreover, diabetic patients have been found to have 5-fold frequency of acute pyelonephritis. Studies show increased incidence of pyelonephritis in diabetic patients (6.8%), as compared to non-diabetic cases (1.6%). Gram-negative organisms are more commonly found in UTI infections as compared to Gram-positive. However, the Gram-positive organisms, especially Enterococci and S. aureus, can cause severe renal parenchymal damage.

**Streptococcus** infection is more common in diabetic subjects with pyelonephritis. The affected kidney is usually enlarged because of inflammation and edema. Infection is focal and patchy, beginning in the pelvis and medulla and extending into the cortex as an enlarging wedge. Cells mediating chronic inflammation appear within a few days. Medullary and subcortical abscesses may develop. Papillary necrosis may be evident in acute pyelonephritis associated with diabetes. Positive urine cultures (i.e. >10 organisms/ml) should be treated in diabetic individuals even if asymptomatic. The choice of antibiotic should reflect the sensitivity of the organism with no treatment difference between diabetic and non-diabetic individuals although the longer duration of treatment is preferred in diabetic patients. Untreated infection may lead to renal parenchymal infection which may impair renal function.

As per IDSA guidelines, infection of Gram-positive cocci, ampicillin-sulbactam with or without an aminoglycoside is recommended. Aminoglycosides should be avoided in patients with a pre-existing renal disease. If parenchymal involvement including abscesses is observed, longer courses of antibiotics (intravenous or oral), or sequential therapy may be necessary. Typically, intravenous antibiotics are continued until the patient is afebrile for at least 24-48 h.

In our case, aminoglycosides, fluoroquinolone, and lincosamide class of antibiotics were resistant. Thus, a newer antibiotic adjuvant entity (ELORES) was used which showed good sensitivity and positive clinical outcome.

**CONCLUSION**

From the present case, ELORES can be considered as a good option to treat MDR pathogens causing UTI. ELORES exhibits various mechanism of bacterial inhibition; efflux pump inhibition, increasing membrane porosity and porin channels, conjugation inhibition and activity on ESBL and MBLs, the empirical course of antibiotic therapy of ELORES
is required, making it an ideal choice for treating MDR pathogens.

REFERENCES


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