A Case Report

END STAGE RENAL FAILURE WITH MULTIPLE CO-MORBIDITY

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ABSTRACT: End-Stage Renal Disease (ESRD) is a progressive and irreversible deterioration in renal function in which the body fails to maintain metabolic, fluid and electrolyte balance. A 65 year old Malay female was admitted to Hospital Universiti Sains Malaysia (HUSM) with complaint of short of breath (SOB) and reduction in urine production. Patient serum creatinine on admission was 1261 µmol/l and urea was 37.3mmol/l. Lungs examination showed decrease breath sounds bibasally and abdominal examination showed soft and non tender region. The patient was diagnosed with end stage renal failure, diabetes mellitus, hypertension, hyperlipidemia, ischemic heart disease and community acquired pneumonia (CAP). The patient had been undergoing dialysis in past, lately the patient had been complaining shortness of breath during the dialysis procedure. The patient was admitted, closely monitored and underwent intense pharmacotherapy plan. Patients with chronic kidney disease often suffer from multiple co-morbidties. Close monitoring and multiple pharmacotherapy are required for manage such patients. hemodialysis maximizes chronic Kidney Disease patient survival and optimize patients' functioning .

Key words: end stage renal disease, hypertension, renal failure, community acquired pneumonia

1. INTRODUCTION

End-stage renal disease (ESRD) is a progressive, irreversible deterioration of renal function in which the body fails to maintain metabolic, fluid and electrolyte balance. This failure of body to maintain the balance results in uremia or azotemia (retention of urea and other nitrogenous wastes in the blood). ESRD may be caused by systemic diseases such as diabetes mellitus (leading cause), hypertension, chronic glomerulonephritis, pyelonephritis (inflammation of the renal pelvis), obstruction of the urinary tract, hereditary lesions, as in polycystic kidney disease, vascular disorders, infections, medications or toxic agents [1]. Co-morbid conditions that develop during chronic renal insufficiency contribute to the high morbidity and mortality among patients with ESRD.

Ischemic heart disease (IHD) is caused by intraluminal artherosclerotic plaque formation consist of fatty deposits of the coronary arteries. The fatty deposits called atheroma are made up of cholesterol that results in arteriosclerosis [2]. Low density lipoprotein (LDL) particles invade the endothelium and oxidized; hence further increase the risk for cardiovascular disease [3]. Patients with hypertension, hyperlipidemia and diabetes mellitus are on high risk to get IHD.

Pneumonia is increasingly being recognized among older patients and is a common finding in patients with multiple co-morbidities that include chronic obstructive pulmonary disease, diabetes mellitus, renal insufficiency, congestive heart failure, coronary artery disease, malignancy, chronic neurologic disease and chronic liver disease. These individuals are at high risk for being infected with a variety of newly identified or previously unrecognized pathogens [4].

2. CASE PRESENTATION

A 65 year old Malay female was admitted to Hospital Universiti Sains Malaysia (HUSM) with the complaint of shortness of breath (SOB) and reduced urine output. She was earlier diagnosed with end stage renal failure along with diabetes mellitus, hypertension, hyperlipidemia, ischemic heart disease and community acquired pneumonia (CAP). She has been on regular hemodialysis (HD) since last two years and relatively well.

During her last HD, she developed SOB and intradialytic hypotension and the procedure was terminated. She was then admitted and treated as Acute Coronary Syndrome with decomposed heart failure.

On examination, patient was alert, conscious and comfortable. Vital signs include blood pressure 159/72 mmHg, pulse rate 88 beats/min and temperature 37.4°C. Cardiac examination showed normal heart sounds, S_1 and S_2. Respiratory examination revealed bibasel decrease breath sounds and abdominal examination was unremarkable.

Initial lab values of patient demonstrated high creatinine, urea and potassium level. Her high white blood cell count, high platelet count, low calcium level and low hemoglobin levels (table 1).

Based on patient deteriorating condition and lab values, patient was admitted and underwent intense pharmacotherapy. Initially Oral calcium polystyrene sulfonate powder and IV lytic cocktail was given to patient for controlling electrolyte imbalances. Along with electrolyte imbalance therapy, felodipine was prescribed to control patient fluctuations in blood pressure. Since the current patient was bed crippled, a decision was taken to initiate anti-coagulant (IV heprin) therapy and statins

Table 1: Initial laboratory values of patient

Laboratory findings	Patient values	Normal range
Urea	37.3 mmol/l	2.8-7.2 mmol/l
Creatinine	1261 µmol/l	72-127 µmol/l
Potassium level	6.1 mmol/l	3.5-5.1 mmol/l
Calcium level	1.82 mmol/l	2.1-2.6 mmol/l
White blood cell count	14 x 10^3/uL	l 4-11 x 10^3/uL
Hemoglobin level	9.9 g/dL	11.5-16.4 g/dL
Low hematocrit level	29%	36-47%
Platelet count	481 x 100/L	150-400 x 100/L

(atorvastatin) were prescribed simultaneously to avoid any risk of cardiovascular complications. Proleptically, a third generation cephaloshporin was prescribed. One of the major complains of patient was the shortness of breath lately; the patient was prescribed a combivent combination inhaler to control her shortness of breath (table 2).

Blood investigations result such as electrolytes, urea and creatinine levels improved after the initiation of the appropriate treatment. Patient was discharged after 4 days and necessary instructions were given to patient and the care givers for future course of treatment.

3. DISCUSSION

Kidney failure results in various clinical manifestations, for instance fluid retention that leads to elevation of blood pressure. The accumulation of nitrogenous metabolic waste products such as urea and creatinine due to the failing renal excretory mechanism. These metabolic derangements lead to several clinical manifestations such as nausea, vomiting, alteration in conscious state and acidosis. Renal replacement therapy need to be initiated immediately to restore the metabolic derangements. Acute treatments include hemodialysis and peritoneal dialysis. Renal transplant is another option that is offered as a part of the long term management. However, this option is often limited due to scarcity of donors. Hemodialysis is a procedure that utilizes diffusive mechanism, blood separated from the dialyste via semi permeable membrane in the dialyzer. Through this mechanism urea and creatinine from blood traverses to the dialysate and purified blood returns to the patient. A vascular access is required for this procedure. The hypervolumic state also can be corrected via its ultrafilterative mechanism. As such the blood pressure will be normalized. Excess electrolyte solutes like potassium, sodium and chloride will also be removed through hemodialysis. Peritoneal dialysis is another mode of renal replacement therapy that utilizes the semi permeable peritoneal cavity and dialysate is infused and removed periodically. Certain period of time is required to allow exchanges between the solute and waste products with the instilled dialysate. These treatments restore the metabolic derangement that occurred and improved the patient's overall outcome. Unfortunately, ESRD is also associated with other medical problems such as bone disease, hypertension, neuropathy, and anemia [5].

In physiological state, the kidney regulates electrolyte, maintaining pH within normal range of 7.35-7.45 and promotes erythopoiesis via the endogenous erythropoietin produced by the alpha intercalated cells in the tubular cells. The vitamin D25-hydroxycalciferol is converted to its active form 1.25 dihydroxychoalcalciferol in the kidney. This active form promotes calcium absorption in the gut. As such this will restore the calcium level. In the renal failure, the conversion did not occur and result in hypocalcemia. As such will stimulate parathyroid gland to release parathyroid hormone in the blood. This hormone serves to stimulate osteoclastic cells in the bone to release calcium and phosphate from the bone. This condition is left untreated potentially lead to secondary hyperparathyroidism and later tertiary hyperparathyroidism. A condition known as chronic Kidney Disease-Mineral Bone Disease (CKD-MBD) will occur as a result of the calcium and phosphate imbalance. Oral activated vitamin D, calcitriol is prescribed to provide patients the required active form of 1.25 dihydroxycholecalciferol. Identification of this condition is crucial and treating it accordingly is critical [6].

Patients with more advanced kidney disease are at risk of developing hyperkalemia because of the inability of the damaged kidney to excrete a potassium load [7]. Treatments are available to improve renal excretion of potassium. The patient should be counseled on their dietary habits as to prevent this problem. These potassium chelating agents available to bind potassium in the intestine and prevent its absorption into the blood stream thereby keeping potassium in the safe normal range. The treatment often acts as a balance because the medications often required for the treatment of hypertension that may affect potassium, either increasing or lowering blood potassium. Both low potassium (hypokalemia) and hyperkalemia have potential of lethal complications because of their effect on the heart. Calcium polystyrene sulfonate is one of the treatments that can be given to the patients with hyperkalemia resulting from acute or chronic renal failure [8]. The drug is excreted as unchanged polystyrene sulfonate resin into the feces without digestion and absorption. As a result, potassium in the intestinal tracts excreted outside the body.

Severe hypertension can cause renal damage and hasten the progression of many intrinsic renal diseases. However, progressive renal disease is also associated with the onset of new hypertension or the worsening of already existing hypertension often to dangerous levels that are resistant to conventional blood pressure treatments.

Renal disease will progress more rapidly and the patient is at risk of other complications such as coronary artery disease and heart attacks as well as strokes without such control. In current case, the patient was given felodipine as the antihypertensive medications. Felodipine is the most potent calcium channel blocker and it can be used to treat mild to moderate essential hypertension [9].

Table 2: Medications prescribed to the patient

Drugs	Therapeutic class	Dose & frequency
IV Cefotaxime (claforan)	3rd Generation Cephalosporin	2g stat then 1g TDS
SC Soluble insulin (actrapid)	Short acting insulin	10 units stat then TDS
T. Felodipine	Calcium channel blocker	5mg OD
T. Atorvastatin	HMG Co-A Reductase Inhibitors	40mg ON
T. Aspirin	Anti-platelet	150mg OD
T. Pantoprazole	Proton pump inhibitor	40mg OD
T. Ezetimibe	Cholesterol lowering agent	10mg OD
T. Calcium Carbonate	Phosphate binder	1g TDS
T. Ferrous Fumarate	Hematinics	200mg OD
T. Folic Acid	Vitamin	1/1 OD
T. Vitamin B Complex	Vitamin	1/1 OD
IV Hydrocortisone	Corticosteroid	200mg QID x 1/7
Nebulizer Ipratropium Bromide /Salbutamol (combivent)	Anti-muscarinic/B agonist	4 hourly
Oral Calcium Polystyrene Sulfonate Powder (kalimate)	Ion-exchange resin for hyperkalaemia	10g TDS x 3/7
IV Lytic Cocktail	Intravenous Potassium chelating agent	IV D 50% 50mls bolus IV Actrapid 10 units stat IV Calcium Gluconate 1 g stat
SC Soluble Insulin (actrapid)	Anti-diabetic	14 units TDS
SC Isophane Insulin (insulatard)	anti-diabetic	8 units ON
T. Clopidogrel	Anti-platelet	75mg OD
IV Infusion Heparin	Anticoagulant	800 unit/L
T. Ivabradine (coralan)	Cardiotonic for angina pectoris, lowers the pulse rate	5mg BD

Many patients suffer from high cholesterol level (hypercholesterolemia) or other abnormalities of lipid metabolism such as hyperlipidemia (high lipid level). Hypercholesterolemia is associated with some forms of kidney disease, particularly those that cause large amounts of protein loss in the urine. The management of hyperlipidemia with diet is necessary and the medications are particularly important in patients with early renal failure. The current patient was treated with HMG CoA reductase inhibitors or "statins" (atorvastatin) and ezetimibe. Atorvastatin reduces levels of "bad" cholesterol (low-density lipoprotein, or LDL) and triglycerides in the blood, while increasing levels of "good" cholesterol (high-density lipoprotein, or HDL) [10]. Ezetimibe can reduce the amount of cholesterol absorbed by the body [10].

Diabetes mellitus and chronic kidney disease often coexist; together they increase the risk for cardiovascular diseases. Studies indicate that diabetes along with micro vascular complications are strongly associated with previous hyperglycemia control [11], taking this in to account it is critical to control hyperglycemia among CKD patients. In the United States, more than half of the patients with diabetes mellitus are currently on dialysis due to the renal failure as a consequence of their diabetes. The progression of diabetic kidney disease can be slow down by optimum control of blood glucose and it is beneficial to patients with diabetes who have other forms of progressive renal disease [12].

Anemia in chronic kidney disease patients, especially in ESRD is very common. They often manifest clinically as pallor and "sallow" appearance. The endogenous relative deficiency of Erythropoietin hormone produced by the alpha-intercalated cells of the tubular cells in chronic kidney disease patients leads to anemia. Erythropoetin is a hormone produced by kidneys to promote erythropoiesis in the marrow. It stimulates the production of red blood cells. The treatment available consists of periodic injections with artificial erythropoietin produced by DNA cloning technology. These injections are highly effective at maintaining a normal or near normal blood count. Hematinic agents are also used in the treatment of anemia. Ferrous Fumarate and B-complex is a once a day iron fortified with folic acid and vitamin B2 and B12 [14]. The ferrous fumarate is fully absorbable and it was the riches salt as regards to iron content. Vitamin produces satisfactory hematologic response and symptomatic improvement in patient suffering from iron deficiency. Ferrous Fumarate and B-complex can improve the blood pressure rapidly and correct the symptoms of hypochromic, microcytic and anemia.

There are many pathogens which can give rise to CAP. Typical bacterial pathogens that cause CAP are *Streptococcus pneumoniae* (penicillin-sensitive and -resistant strains), *Haemophilus influenzae* (ampicillin-sensitive and -resistant strains), and *Moraxella catarrhalis* (all strains penicillin-resistant). These 3 pathogens account for approximately 85% of CAP cases [8]. When the drug-resistant *Streptococcus pneumoniae* (DRSP) was suspected, the Centers for Disease Control and Prevention (CDC) group has identified several number of active β-lactam agents that can be used as initial empiric therapy, if the organism has a penicillin MIC of 2 mg/L. The active β-lactam agents include oral therapy with cefuroxime

(alternatively cefpodoxime), high-dose amoxicillin (1 g every 8 h), or amoxicillin/clavulanate (875 mg twice daily); intravenous therapy with cefotaxime, ceftriaxone, ampicillin/sulbactam; or, alternatively, a new antipneumococcal fluoroquinolone could be used.

4. CONCLUSION

The current case illustrates a good example of multiple co-morbidities of and ESRD patients. Hemodialyis in CKD patients may prolong patient's survival, optimize their functioning and well-being. Promoting physical, psychosocial, and vocational rehabilitation is an important survival aspect for such patients'. Patient should continue and comply with the medications especially related to the co morbidity. Continued treatment for co-morbidities, changes in life style and diet could bring positive changes in quality of life of patient.

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