A Case Report

END STAGE RENAL FAILURE WITH SUSPECTED TUBERCULOSIS AND HOSPITAL ACQUIRED PNEUMONIA

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ABSTRACT: Immunocompromised chronic kidney disease (CKD) patients are often subjected to multiple complications. Hospital Acquired Pneumonia (HAP) is common among patient with long term warded in hospital. A 56 years old Malay women diagnosed with end stage renal failure 2 years ago was presented with intermittent fever and on/off cough with whitish sputum. Patient was admitted one week before for chest infection. The laboratory finding shows that the patient had hypochromic mycocytic anemia since her hemoglobin and hematocrit level was below normal range. The CT thorax done proposes a suggestive that the patient had chest infection or pneumonia. Chronic kidney disease patients are often associated with multiple co-morbidies. Low immune response makes CKD patients an ideal subject for hospital acquired pneumonia. Critical review on patient pharmacotherapy should be done in patients with conditions that alter drug pharmacokinetics as CKD.

Key words: chronic kidney disease, hospital acquired pneumonia, multiple co-morbidies

1. INTRODUCTION

End stage renal failure (ESRF) is last stage kidney disease, when estimated glomerular filtration rate is less than 15 mL/min/1.73m2 [1]. ESRF Patients, at this stage will require renal replacement therapy in the form of dialysis or transplantation to sustain life. This disease is characterized by a progressive deterioration in kidney function that ultimately leads to irreversible kidney damage. Complication that associated with ESRF other than increase complexity of the condition is fluid and electrolyte abnormalities, anemia, cardiovascular disease, hyperparathyroidism, bone disease, and malnutrition. Studies indicate that both innate and adaptive immune system contribute to an increased rate of infection among ESRD patients. Functional abnormalities of monocytes, neutrophils and dendritic cells are directly linked with ESRD patients [2-4]. With so many medications prescribed to ESRD patient, compliance or adherence issue become a major problem in most of the patient.

Hospital acquired pneumonia is defined as infection of the lungs that occurs >48 hours after admission that was not incubating at the time of admission [5]. Tuberculosis is caused by M. tuberculosis, an aerobic, non-spore-forming bacillus often refered to as acid-fast –bacillus (AFB). This disease usually spread through the air ,patient with low immune system will have high possibility to get infected. The symptom of TB is almost similar to pneumonia, such as cough, loss appetite, coughing up blood or mucus, weakness or fatigue, fever and night sweats [5].

Case presentation

A 56 years old Malay women, weighing 39Kg, was admitted to Hospital Universiti Sains Malaysia (HUSM) with complaining of intermittent fever and on/off cough with whitish sputum. The patient had hemoptysis and complained of shortness of breath. She was admitted to the ward one week before for chest infection or pneumonia. Patient also claimed to have anemic symptom (pallor, tremor, nausea) before loss conscious at home. On examination, subject had mild tachypneic and her vital sign were; BP (160/65mmHg), PR (100 bpm), RR (20 bpm), T (38.5OC), and saturated partial O2 (SPO2) are 92% under room air and 96% under Nasal Prongs O2 (table 1). Physical examination find that her lung presents with coarse crept at right side. Computed tomography (CT) thorax shows a multiple area of consolidation in keeping with chest infection.

Table 1: laboratory profile of patient

Laboratory profile	Patient value	Normal range
Hgb	7.5 g/dL	11.5-16.4 g/dL
Hct	23.6%	36-47%
Platelet	136 x10^3/L	150-400 x10^3/L
PO_4	0.79 mmol/L	0.8-1.45 mmol/L
Total Protein	58 g/L	66-83 g/L
Prothrombine Time	14.8s	10-13.5s
aPTT	77.0s	26-42s
PCO_2	116 mmol/L	35-45 mmol/L
HCO_3	30 mmol/L	72-100 mmol/L
O_2 Saturation	99%	90-95%

On admission, patient was diagnosed with upper GI bleeding secondary to ureamicgastropathy, end stage renal failure (ESRF) secondary to obstructive uropathy, hospital acquired

pneumonia and also with hypochromic mycocytic anemia. She was on regular hemodialysis (HD).

Multiple pharmacotherapies were initiated to stabilize the patient. Peptic ulcer disease (PUD) is a common reason for upper gastric bleeding, patient was prescribed pantataperazole (40 mg BD) to treat PUD. Antihypertensive were also initiated to control bleeding disorder of GI, this included enalapil (10mg BD) and felodipine (10mg OD). Antibiotics for hospital acuquired pneumonia was initiated that included azithromycin tazocin, meropenem and voriconazole

The patient was treated for pneumonia along with other complications aggressively, since her lab reports were improving and to avoid any further hospital acquired infection patient was discharged 4 days after the initiation of treatment and asked to take complete rest. Table 1 provides the details of medication prescribed to patient to improve her condition.

2. DISCUSSION

Hospital acquired pneumonia (HAP) occurs due to the imbalance between host defenses and microbial propensity for colonization and invasion shift in favor to the ability of pathogens to persist and invade lower respiratory tract. The source of infection is from healthcare devices or the environment (air, water, equipment and fomites) and can occur with transfer of microorganisms between staff and patients [1].

In treating HAP, third-generation cephalosporin, broad-spectrum penicillins, fluoroquinolones, aminoglycosides and carbapenems can be used as they provide broad-spectrum activity against the common aerobic pathogenscausing HAP. Other agents such as macrolides and lincosamides, linezolid and vancomycin have excellent activity against Gram-positive cocci, while demonstrating minimal activity against Gram-negative bacilli [2]. In treating current patient, the doctor prescribes azithromycin 500mg OD and IV piperacillin/tazobactam 225mg TDS then change to IV meropenem 500mg TDS. Piperacillin/tazobactam is the first choice in treating HAP which covers Pseudomonas and azithromycin is given to cover Legionella [3].

Table 2: medication given to patient in the ward

Drug Name	Dose Regimen	Indication	Metabolism	Primary Excretion
T. Isoniazid	200mg OD	Anti-TB	Hepatic	Renal
T. Rifampicin	300mg OD	PTB Smear -ve	Hepatic	Renal
T. Pyrazinamide	750mg OD		Hepatic	Renal
T. Ethambutol	800mg OD		Hepatic	Renal
T. Pyridoxine	10mg OD		Hepatic	Renal
T. Azithromycin	500mg OD	Antibiotic for HAP	Hepatic	Hepatic
IV Tazocin	225mg TDS		Hepatic	Renal
IV Meropenem	500mg TDS		Hepatic	Renal
IV Voriconazole	200mg BD		Hepatic	Renal
T. Enalapil	10mg BD	Antihypertensive	Hepatic	Renal
T. Felodipine	10mg OD		Hepatic	Renal
T. Isordil	10mg TDS	Angina Pectoris	Hepatic	Renal
T. Atovastatin	20mg ON		Hepatic	Renal
T. TrimetazidineHCl (Vastarel MR)	35mg BD		Hepatic	Renal
T. Nitroglycerin	0.5mg PRN		Hepatic	Renal
T. Pantoprazole	40mg BD	PUD	Hepatic	Renal
T. Metoclopramide (Maxolon)	10mg TDS	Antiemetic	Hepatic	Renal
KCl mist	15mL TDS	Hypokalemia	-	-
T. $CaCO_3$	1mg TDS	Hyperphosphatemia in ESRD	Hepatic	Hepatic
T. Folic Acid	5mg OD	Hematinic Agent	Hepatic	Renal
T. Vitamin B Complex	1/1 OD			
T. Ferrous Fumarate	400mg OD			

HAP Hospital acquired pneumonia; PUD peptic ulcer disease

HAP can be divided into early-onset and late-onset HAP. Early-onset HAP happens within 4 days of admission and is often attributed to community-acquired organisms that were colonizing the patient around the time of hospital admission. Late-onset HAP (5 days or more) is more likely to be caused by more resistant gram-negative bacilli, S. aureus including methicillin-resistant S. aureus (MRSA), and L. pneumophila [4]. According the guidelines for pneumonia in adult patients, antipseudomonal β-lactam plus an anti pseudomonal fluoroquinolone or aminoglycoside plus or minus linezolid or vancomycin for MRSA are the regimens for late onset of HAP [4]. Current patient was only prescribed meropenem for her HAP. Thus, fluoroquinolone or aminoglycoside should be added to the regimen for HAP.

Tuberculosis (TB) can only been diagnosed by culturing M. tuberculosis organisms from a specimen taken from the patient. TB is difficult to diagnose due to the difficulty in culturing the slow-growing organism in the laboratory. A complete evaluation for TB includes a medical history, a chest radiograph, a physical examination, and microbiologic smears and cultures. It may also include a tuberculin skin test and a serologic test. The first line treatment of tuberculosis is isoniazid, rifampicin, pyrazinamide, ethambutol and streptomycin [5]. In this case, the patient was given isoniazid 200mg stat then OD, rifampicin 300mg stat then OD, pyrazinamide 750mg stat then OD (except HD day), ethambutol 800mg stat then OD. Three specimen of sputum acid-fast bacilli (AFB) done in this patient showed negative result but the treatment was continued. This is because negative smear results do not exclude TB disease. Culture of clinicalspecimens will confirm the diagnosis in smear-positive cases and usually identify that many cases, since the culture is more sensitive (80 to 85%) compared to smear. However, the time to detection and speciation of cultures may be up to 7 weeks, particularly when the burden and metabolicactivity of the mycobacteria are very low, as is often the case in smear-negative, culture-positive disease [6]. The treatment was continued even with the AFB smear is negative as it takes some time waiting for the culture result.

Serum galactomannan was detected in this patient. Galactomannan is the molecule found in cell wall of aspergillus sp. Vorionazole is the drug of choice for primary therapy of most patients with aspergillosis as it provided improved survival and fewer side effects [7]. In patients with Clcr<50ml/min, accumulation of IV vehicle (cyclodextrin) of voriconazole occurs. After initial IV loading dose, oral voriconazole should be administered unless an assessment of benefit vs risk to the patient justifies the use of IV voriconazole. Serum creatinine should be monitored and changing to oral voriconazole therapy should be done when possible. QT prolongation has been associated with voriconazole use. Rare cases of arrhythmia, cardiac arrest, and sudden death have been reported, usually in seriously ill patients with risk factors such as electrolyte imbalance. For current patient, electrolyte abnormalities (hypokalemia, hypomagnesemia, hypocalcemia) should be corrected prior to initiating the therapy. Rifampicin is contraindicated with the use of voriconazole. Rifampicin decreases the levels of voriconazole by increasing the metabolism. This problem can be encountered by adjusting the dosing interval, 3-4 hours apart for the time taking anti-tuberculosis drugs and voriconazole.

3. CONCLUSION

CKD patients have alterations in their pharmacokinetic parameters such as drug absorption, distribution, protein binding, biotransformation & renal function. Such patients are usually with multiple co-morbidities and require close observation. The role of clinical pharmacist in applying pharmacokinetics principle in dosage adjustment, drug dosage adjustment, monitoring & assessing patient medication therapy is critical.

4. REFERENCES

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