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Managing Blood Glucose in Patient with Acute on Chronic Kidney Disease: A Case Report

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Abstract: ***Chronic Kidney Disease (CKD) is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. Diabetes and hypertension are the leading causes of CKD in all developed countries. Managing glucose control is the key strategy in reducing the risk or slowing the progression of diabetic kidney disease (DKD). Meanwhile, they are at risk for acute diabetes-related complication such as hypoglycemia and diabetic ketoacidosis; long-term complications such as retinopathy, neuropathy, and foot complications. Patient with diabetes and CKD should be treated with a comprehensive strategy to reduce risks of the kidney progression and cardiovascular disease.***

Keywords: Chronic kidney disease, diabetes mellitus, diabetic kidney disease

1. Introduction

Chronic kidney disease (CKD) has been recognized as a leading public health problem that both increases the risk of end stage of renal disease (ESRD), cardiovascular disease as well as other complications.[1] CKD is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. The markers of kidney damage include albuminuria, urine sediment abnormalities, electrolyte and other abnormalities due to tubular disorders, abnormalities detected by histology, structural abnormalities detected by imaging, history of kidney transplantation, and decrease glomerular filtration rate (GFR) < 60 ml/min/1.73 m^2.[2]

In 2017, the global prevalence of CKD were 697.5 million cases. About a third of patient with CKD lived in China and India. Bangladesh, Brazil, Indonesia, Japan, Mexico, Nigeria, Pakistan, Russia, the USA, and Vietnam had more than 10 million cases of CKD each.[3]Diabetes and hypertension are the leading causes of CKD in all developed countries. While in countries of Asia and sub-Saharan Africa, glomerulonephritis and unknown cases are the leading causes of CKD.[1]Managing glucose control is the key strategy in reducing the risk or slowing the progression of diabetic kidney disease (DKD). However, treatment of patients with diabetes mellitus and concomitant CKD is very challenging due to impaired renal clearance on the pharmacokinetic and pharmacodynamic profiles of antihyperglycemic therapies and to enable patient to meet their diabetes treatment goals.[4]

The aim of this case report was to evaluate glycemic control in patients with diabetic kidney disease (DKD).

2. Case Report

A 56 years old female patient was admitted to Internal Medicine Department, Wangaya Regional General Hospital, Bali, Indonesia on 4th May 2021 due to persistent fever in the past 3 days. In the past 3 days, her fever always around 40 degree Celsius and does not improve with paracetamol tablet. Patient also complained about an epigastric pain for 1 week together with nausea and vomiting. She denied any symptoms regarding urination, diarrhea, or constipation. The patient has a history of dyspepsia, uncontrolled type II diabetes mellitus (DM), and has been taking paracetamol, omeprazole, ondansetron, and antacid syrup before admitted to hospital. There is no history of hypertension, dyslipidemia, or cardiovascular disease in herself nor in her family. Patient also denied any bad habit like smoking or alcohol consumption.

Patient was conscious but physically weak with blood pressure 113/75 mmHg, heart rate 115 beats per minute (bpm), respiratory rate 22 times per minute, and temperature 39.5 degree Celsius. From physical examination, we can't find any abnormalities. From blood examination: leukocytosis (leukocyte 14.730/ul), mild anemia (hemoglobin 11.3 g/dL), thrombocytopenia (57.000/ul), elevated liver enzyme (SGPT 121 U/L SGOT160 U/L), increase blood urea nitrogen (BUN) and creatinine(urea 84 mg/dL and creatinine 2.4 mg/dL), low glomerulus filtration rate (GFR 24.79 ml/min/1.73 m^2),hyperglycemic (random blood glucose 332 mg/dL) and hyponatremia (natrium level 130 mmol/L). From thorax x-ray: cardiomegaly. We performed abdominal ultrasound on patient to assess patient's abdominal pain and found fatty liver, right hydronephrosis grade II, and left nephrocalcinosis. Therefore, we diagnosed acute on chronic kidney disease *et causa* pre-renal, type II diabetes mellitus, transaminitis, and febris*et causa* suspect bacterial infection. Patient was given intravenous fluid NaCl 0,9% with amino acids, antipyretic (paracetamol infusion), antibiotic (ceftazidime 3 x 1 gram IV and moxifloxacin 1 x 400 mg IV), insulin (glulisine injection 3 x 2 unit, glargine injection 4 unit), proton pump inhibitor (PPI) (esomeprazole 2 x 40 mg IV), antacid (3 x 1 tablespoon), antiemetic (ondansetron 3 x 4 mg IV), hepato-protector, and renal protection agent.

On the 2nd day of hospitalization, patient's condition is getting worse with blood pressure 90/55 mmHg, heart rate

98 bpm, respiratory rate 26 times per minute, temperature 38,6 degree Celsius and vasopressor was given. We consulted to anesthesiologist, cardiologist and urologist for patient's condition. We recheck patient's complete blood count, blood glucose, and urinalysis. From blood examination: leukocytosis (leukocyte 32.460/ul), anemia (hemoglobin 10.4 g/dL), thrombocytopenia (50.000/ul), and hyperglycemic (blood glucose at 10 PM 197 mg/dL, fasting blood glucose 244 mg/dL). From urinalysis: positiveleukocyte esterase (leukocyte esterase 25 leukocyte/uL), positive protein in urine (protein 1+), and bacteriuria (bacteria ++). Thus, we diagnosed as shock *et causa* sepsis and urinary tracts infection.

On 3rd day of hospitalization, the patient admitted to Intensive Care Unit (ICU). Diagnosis from cardiologist: cardiomegaly *et causa* hypertensive heart disease (HHD) and shock *et causa* sepsis. While diagnosis from urologist: suspect right nephrolithiasis, right hydronephrosis, and suspect urosepsis. During 6 days hospitalization in ICU, her condition is getting better. From blood examination: leukocyte 7.300/ul, anemia (hemoglobin 10.4%), thrombocyte 170.000/ul,), mild elevated liver enzyme (SGPT 37 U/L SGOT 40 U/L), increase blood urea nitrogen (BUN) and creatinine (urea 86 mg/dL and creatinine 2.6 mg/dL), low glomerulus filtration rate (GFR 22.88 ml/min/1.73 m^2), and hyperglycemic (blood glucose at 10 PM 197 mg/dL, fasting blood glucose 179 mg/dL). Patient was able to stabilized with intravenous fluid NaCl 0,9%, insulin (glulisine injection 3 x 4 unit, glargine injection 4 unit), antipyretic (paracetamol infusion), antibiotic (ceftazidime 3 x 1 gram IV), proton pump inhibitor (PPI) (esomeprazole 2 x 40 mg IV), antacid (3 x 1 tablespoon), hepato-protector, and renal protection agent.

On 9th day of hospitalization, the patient has moved to normal ward and decided to went through uroscopy (URS). However, during her assessment for operation, we found hypertension urgency with blood pressure200/100 mmHg. Therefore, we started to give her anti-hypertensive agent (angiotensin II receptor blocker, calcium channel blocker, and diuretic). On 15th day of hospitalization, the patient has stabilized and went through URS. Two days after operation, patient's condition is improved and able to discharge from hospital with antipyretic, anti-hypertensive agent, hepato-protector, renal protection agent, and insulin.

3. Discussion

Chronic kidney disease (CKD) is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. The markers of kidney damage include albuminuria, urine sediment abnormalities, electrolyte and other abnormalities due to tubular disorders, abnormalities detected by histology, structural abnormalities detected by imaging, history of kidney transplantation, and decrease glomerular filtration rate (GFR) < 60 ml/min/1.73 m^2.[2]

The symptoms of early stage of CKD is asymptomatic until GFR reaches 30 ml/min/1.73 m^2, the patient starts to feel lethargy, nocturia, nausea, appetite decreases, and weight loss. When GFR < 30 ml/min/1.73 m^2 will causeuremic symptoms such as anemia, hypertension, impaired calcium and phosphate metabolism, pruritus, nauseas, vomiting, etc. If GFR < 15 ml/min/1.73 m^2, it will cause serious complication and may need renal displacement therapy.[5]Diabetes and hypertension are the leading causes of CKD in all developed countries. While in countries of Asia and sub-Saharan Africa, glomerulonephritis and unknown cases are the leading causes of CKD.[1] In Indonesia Renal Registry 2015, it was recorded that hypertension,diabetes mellitus, and obstructive nephropathy were the three common causes of CKD in Indonesia.[6]CKD can be divided into 5 stage: stage 1 with GFR ≥ 90 ml/min/1.73 m^2, stage 2 with GFR 60-89 ml/min/1.73 m^2, stage 3 with GFR 30-59 ml/min/1.73 m^2, stage 4 with GFR 15-29 ml/min/1.73 m^2, and stage 5 with GFR < 15 ml/min/1.73 m^2.[5]

In this patient, we could find markers of kidney damage such as electrolyte abnormalities (hyponatremia) and decrease glomerular filtration rate (GFR 22.88 mL/min). Even though, we can't prove her abnormalities present for > 3 months. However, from patient's underlying disease (nephrolithiasis, hydronephrosis, type II diabetes mellitus) which is Indonesian's most common cause of CKD. Therefore, we diagnosed it as Chronic Kidney Disease (CKD) stage IV.

Patient with diabetes and CKD should be treated with a comprehensive strategy to reduce risks of the kidney progression and cardiovascular disease. Strategies of patient with diabetes and CKD are glycemic control, blood pressure control, lipid management, exercise, nutrition, and smoking cessation.[7]Glucose metabolism in CKD is mediated by multiple mechanisms: 1) impaired glucose disposal by muscle and peripheral tissues due to uremia; 2) reduced insulin removal by the damaged kidney; 3) persistent mild inflammatory state; 4) over-secretion of counter-regulatory hormones. Patient with advanced CKD are also predisposed to postprandial hyperglycemic due to impaired osmotic diuresis and increased muscle insulin resistance.[8] Therefore, patient with diabetes and CKD are at risk for acute diabetes-related complication such as hypoglycemia and diabetic ketoacidosis; long-term complications such as retinopathy, neuropathy, and foot complications.[7]

KDIGO recommended using hemoglobin A1c (HbA1c) twice per year in patient with diabetes and 4 times a year in patient who glycemic target. For patients for whom prevention of complications is the key goal, HbA1c < 6.5% or <7% might be preferred. For those with multiple comorbidities or increased burden of hypoglycemia, HbA1c<7.5%or <8% might be preferred. Metformin and sodium/glucose cotransporter-2-inhibitors (SGLT2i) are the first line therapy for DM, CKD, and eGFR ≥ 30 ml/min/1.73 m^2.Other additional drug such as glucagon-like peptide-1 (GLP-1) receptor agonist, dipeptidyl peptidase-4 (DPP-4) inhibitors, sulfonylurea, insulin, thiazolidinedione, alpha-glucosidase inhibitor should be guided by patient preferences, comorbidities, eGFR, and cost.[7]For patient with GFR < 30 ml/min/1.73 m^2, insulin is one of the choice. Patient with GRF is 10-50 ml/min/1.73 m^2, insulin dose should reduce by 25%. While patient with GRR < 10 ml/min/1.73 m^2, insulin dose should be reduced by 50%. In patient on hemodialysis, insulin resistance may decrease due

to uremia. Rapid insulin degradation continues and hence, the insulin requirement reduces at the time of dialysis. Some of the patient require half of the doses on dialysis days and some do not require any insulin. However, some patient will continue to need the same insulin dose even on dialysis days.[9]

In this case report, patient's GFR is 22.88 ml/min/1.73 m^2. Metformin and SGLT2i should be given if GFR < 30 ml/min/1.73 m^2. Thus, insulin is our first choice in this case report. We reduced her insulin dose by 25%. Patient's total insulin daily dose is 0,3 unit/kg body weight x 60 kg is equal to 18 unit.[9,10,11] We need to reduce by 25% which is 13,5 unit. Prandial insulin is 60% of total daily dose, while basal insulin is 40% of total daily dose.[11] Prandial insulin is 60% x 13,5 unit = 8 unit and basal insulin is 40% x 13,5 unit = 5,5 unit.Starting dose before meal is 8 unit divided by 3 equals 2,67 unit. We gave her glulisine injection 3 x 2 unit and glargine injection 4 unit, then we adjust the dose according to her blood glucose. The weakness of this case report is we didn't check patient's HbA1c to evaluate her glycemic target.

4. Conclusion

Chronic kidney disease (CKD) has been recognized as a leading public health problem that both increases the risk of end stage of renal disease (ESRD), cardiovascular disease as well as other complications. Chronic kidney disease (CKD) is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. Hypertension,diabetes mellitus, and obstructive nephropathy were the three common causes of CKD in Indonesia. Strategies of patient with diabetes and CKD are glycemic control, blood pressure control, lipid management, exercise, nutrition, and smoking cessation. Metformin and sodium/glucose cotransporter-2-inhibitors (SGLT2i) are the first line therapy for DM, CKD, and eGFR ≥ 30 ml/min/1.73 m^2. For patient with GFR < 30 ml/min/1.73 m^2, insulin is one of the choice.

5. Conflict of Interest

The authors declare no conflicts of interest.

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