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Persistent Hyperkalaemia in Lupus Nephritis - A Case Report

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Abstract: ***Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease that often involves the kidneys, that is lupus nephritis. This occurs in almost 50% of SLE patients. Patients with lupus nephritis present with immune complex-induced interstitial nephritis. It was common causes of oliguric acute renal failure that leads to decreased renal excretion including potassium. We present a case of persistent hyperkalaemia related to lupus nephritis, in patient with new onset SLE admitted in Wangaya Regional Hospital. In this case, we found an interesting case where we were correcting a patient with lupus nephritis and persistent hyperkalaemia. As we know, hyperkalaemia is a life-threatening condition causing cardiac arrhythmia and muscle paralysis.***

Keywords: hyperkalaemia, lupus nephritis, systemic lupus erythematous

1. Introduction

SLE is a chronic multisystem autoimmune disease that predominantly affects women of childbearing age. Several factors are involved in the development of SLE such as genetic factors, environmental triggers, and the hormonal[1]. SLE often involves the kidneys causing lupus nephritis. Lupus nephritis occurs in almost 50% of patients with SLE and is the most common cause of kidney injury in SLE [2].

Lupus nephritis typically develops early in the disease course, generally within the first 6 to 36 months, and may be present at initial diagnosis. Lupus nephritis is more common in women than in men. In the United States, the incidence of lupus nephritis is higher prevalence (31%-55%) among black, Hispanic, and Asian women between ages 15 to 44 years old. This population tends to develop the disease earlier and experience more serious complications lead to end stage kidney disease [2, 3].

Patients with lupus nephritis present with immune complex-induced interstitial nephritis [4]. This immune process triggers kidney damage, while hemodynamic and metabolic factors activated after this initial event promote chronic, progressive damage to the kidney [5]. Interstitial nephritis is one of the common causes of oliguric acute renal failure. The distal tubules and collecting duct cells are often damaged and thus unable to excrete potassium. The distal delivery of sodium and/or the distal tubular flow rate is often decreased, causing hyperkalaemia [6].

Hyperkalaemia is a life threatening condition causing fatal cardiac arrhythmias and muscle paralysis. It usually caused by extracellular potassium shift or decreased renal potassium excretion [6]. A decreased excretion problem occurs when renal potassium excretion is limited by reductions in glomerular filtration rate, tubular flow, distal sodium delivery or the expression of aldosterone-sensitive ion transporters in the distal nephron [7].

We present a case of persistent hyperkalaemia related to lupus nephritis in patient with new onset SLE admitted in Wangaya Regional Hospital. This case presentation aims to remind the healthcare provider in managing patient with lupus nephritis which is often has non-specific symptom hyperkalaemia leading to serious complication if left untreated and the factors that leads to hyperkalaemia.

2. Case Report

A 41 years old woman admitted to Department of Internal Medicine, Wangaya Regional General Hospital, Denpasar, Bali on 5th June 2021 due to epigastric pain in the past 2 months. The pain described as a sharp pain and intermittent without any trigger at epigastric region. It is getting worse in the last 2 weeks. Patient also feels nausea and it caused her to have low appetite. The patient also complained myalgia, hair loss, rash and itching at face and body, and joint pain in the last 2 months. She has denied any weakness, fever, vomiting, palpitation, urination's abnormalities, diarrhea, and constipation. There are no history of diabetes mellitus, hypertension, dyslipidemia, or cardiovascular disease in patient and her family. Patient also denied any habit like smoking or alcohol consumption. About 2 months ago, patient had admitted to Wangaya Regional General Hospital due to bilateral leg swelling but physical and laboratories examination were normal.

The patient was in conscious state with blood pressure 120/70 mmHg, heart rate 80 beats per minute, respiratory rate 22 times per minute, temperature 36.6 degree Celsius, and oxygen saturation 98%. From physical examination, we found alopecia, malar rash (butterfly rash) at the face, discoid rash at the chest, and tenderness at epigastric region. From blood examination, we found anemia normocyte normochromic (hemoglobin 6.8 g/dL, MCV 85.1 fL, MCH 27.4 pg, MCHC 32.2 g/dL), elevated urea and creatinine levels (urea 82 mg/dL, creatinine 2/3 mg/dL), and hyperkalemia (potassium 6.7 mmol/L). The chest x-ray and electrocardiography (ECG) are normal. From the history taking, physical, and laboratories examination, we suspected SLE with anemia, hyperkalemia, acute on chronic kidney disease, and dermatitis. To diagnose SLE, the patient undergoes more examination such as urinalysis, antinuclear

antibody (ANA) test, abdomen ultrasonography (USG), and esophagogastroduodenoscopy (EGD).

The patient was given intravenous fluid (NaCl 0,9%), blood transfusion, insulin-dextrose, calcium salt (calcium gluconate), α2-agonist (salbutamol), corticosteroid (methylprednisolone), proton pump inhibitors (omeprazole), antiemetic (ondansetron), antacid, and gastro-duodenal protective agent (sucralfate) while waiting for the result of examination. Urinalysis showed proteinuria (protein 3+), hematuria (blood 3+), and erythrocytes in urinary sediment, and granular casts. This examination increases our suspicion towards SLE.

During hospitalization, patient's potassium level is very high and fluctuating. The management of hyperkalemia that given to the patient were insulin-dextrose (dextrose 40% 50 cc together with dextrose 10% 200 cc and rapid-acting insulin 20 units over 5 hours), calcium salt (calcium gluconate 3 x 1 ampule), and α2-agonist (salbutamol inhalation every 8 hours). On 8th June 2021, we re-confirm patient's potassium level through electrocardiography (ECG) and the ECG's result was normal. Therefore, we gave patient calcium polystyrene sulfonate.

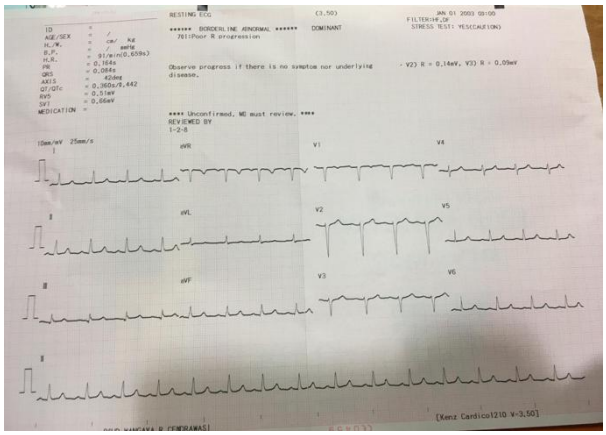

Figure 1: Electrocardiography (ECG) of Patient with Hyperkalemia

However, on 9th June 2021, patient's potassium level is increases and abdomen USG shows parenchymal liver disease with differential diagnosis fatty liver (grade II-III), bilateral nephritis, and ascites. We re-confirm patient's potassium level by cross check with laboratory outside our hospital and the results shows potassium level at our laboratory is 6.6 mEq/L and laboratory outside hospital 6.1 mEq/L. Thus, we add furosemide in order to increase the excretion potassium through renal system. Patient is also undergone serum albumin and globulin examination. The laboratories show total protein 4.4 g/dL, albumin 1.2 g/dL, and globulin 3.2 g/dL. Through this blood examination, we gave albumin 20% to the patient. On 11th June 2021, ANA Indirect Immunofluorescence (IF) speckled with titer >1:1000 and ANA Profile shows RNP/Sm and Sm are positive 3, nucleosomes (NUC) is positive 2, and SS-A native (SSA) together with histones (HI) are positive 1. EGD's result is gastritis erosive at antrum region. Potassium level has decreases from 5.5 mmol/L to 5.1 mmol/L. Therefore, the management of hyperkalemia has stopped.

The table below shows the potassium level after each of the correction.

Table 1: Potassium Level

Date	Number of corrections	Potassium (mmol/L)
05/06/21		6.7
	After 1st correction of hyperkalemia	6.0
06/06/21	After 2nd correction of hyperkalemia	5.9
	After 3rd correction of hyperkalemia	7.5
07/06/21	After 4th correction of hyperkalemia	6.6
	After 5th correction of hyperkalemia	6.0
08/06/21	After 6th correction of hyperkalemia	7.2
	After 7th correction of hyperkalemia	6.1
09/06/21	After 8th correction of hyperkalemia	7.0
	After 9th correction of hyperkalemia	6.2
	After 10th correction of hyperkalemia	6.6
10/06/21	After 11th correction of hyperkalemia	5.5
11/06/21	After 12th correction of hyperkalemia	5.2

We diagnosed the patient as SLE with nephritis lupus, gastritis, minimal ascites, anemia, and hyperkalemia. Patient's condition has stabilized and discharge from the hospital with antacid, gastro-duodenal protective agent (sucralfate), calcium polystyrene sulfonate, corticosteroid (methylprednisolone) and hydroxychloroquine.

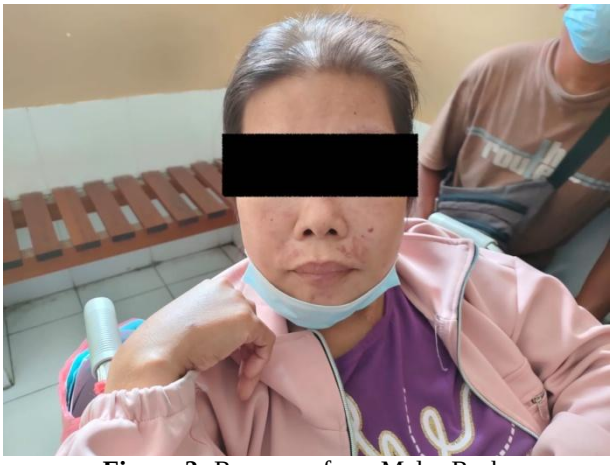

Figure 2: Recovery from Malar Rash

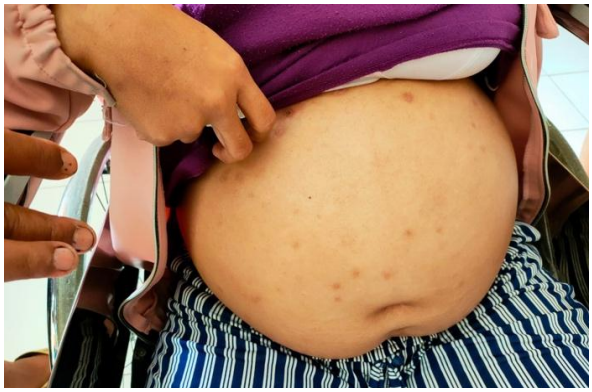

Figure 3: Recovery (Hyperpigmentation) from Discoid Rash

3. Discussion

This case illustrates new-onset SLE that experienced hyperkalemia due to renal failure in lupus nephritis.

Hyperkalemia is defined as a serum or plasma potassium level above the upper limits of normal, usually greater than 5.0 mEq/L to 5.5 mEq/L. Mild hyperkalemia is usually asymptomatic, high levels of potassium may cause life-threatening cardiac arrhythmias, muscle weakness or paralysis. Symptoms usually develop at higher levels, 6.5 mEq/L to 7 mEq/L [8].

This patient had some symptoms related to SLE and later confirmed by ANA test. At the time of routine laboratory examinations before hospitalization, high levels of potassium were found in the blood. LN is believed to be the underlying disease for hyperkalemia in this patient as it comes in most SLE patient. Symptoms related to hyperkalemia including muscle cramps, shortness of breath, palpitation, diarrhoea and weakness were denied, she was admitted for chief complaint of abdominal pain. This patient had fluctuated high potassium level for 6 days despite had given therapy since day one admitted.

The most common cause of hyperkalemia is pseudohyperkalemia, which is not reflective the true serum potassium levels. Etiology of hyperkalemia can be categorized into 3 categories: (1) increased potassium intake, (2) intracellular potassium shifts, and (3) impaired potassium excretion. Increase potassium intake from food is a very uncommon cause especially in adult patient with normal renal function. Intravenous intake through high potassium-containing fluids, medication, and massive blood transfusions can significantly elevate serum potassium levels [8].

Cellular injury, metabolic acidosis, sepsis, dehydration, insulin deficiency, diabetic ketoacidosis, and tumor lysis syndrome may cause intracellular potassium to shift into the extracellular space [8]. Decrease distal renal flow (such as acute or chronic kidney disease, congestive heart failure, and cirrhosis), hypoaldosteronism, and primary renal tubular defects (such as sickle cell disease, SLE, obstructive uropathy, hereditary tubular defects, and amyloidosis) could cause impaired potassium excretion [9]. Hyperkalemia is usually seen if glomerular filtration rate falls below 30 ml/min [8].

Tubulointerstitial lesions may be encountered in all classes of lupus nephritis. The pathomechanism is as the result of circulating immune complexes specifically interacting with tubulointerstitial autoantigens [10, 11]. Intrarenal inflammation is maintained via local cytokine and chemokine production, which attracts leukocytes into the glomerulus and interstitial. Further amplify local inflammation, renal cell loss, and nephron atrophy causing decreased renal excretory function as a late marker of underlying progressive nephron loss [11].

We diagnose this patient with the manifestations of LN including oedema, proteinuria, hypo protein, haematuria and renal failure accompanied by anaemia.The patient is an Asian woman aged 41 years which is risk factors for LN.Also the patient has no past history regarding renal diseases. Patient has persistent high potassium level without any symptoms. Another cause of hyperkalemia such as increased potassium intake, haemolytic processes, sepsis or dehydration, certain medications, and pseudohyperkalaemia had been excluded. ECG was performed to evaluate the cardiac condition, and there were no abnormalities found. High potassium level usually exhibit ECG alteration however ECG can be normal in hyperkalaemia patients[12].

Management of hyperkalaemia includes the elimination of reversible causes (diet, medications), rapidly acting therapies that shift potassium into cells and block the cardiac membrane effects of hyperkalaemia, and removal of potassium from the body (saline diuresis, oral binding resins, and haemodialysis). Treatment should be started with calcium gluconate to stabilize cardiomyocyte membranes, followed by insulin injection, and b-agonists administration [8]. In this case, we performed several times of hyperkalemia therapy management in patients due to fluctuating potassium levels in order to achieve normal potassium levels.

This patient received calcium gluconate as calcium salts antagonize the effects of potassium on cardiomyocyte membranes without affecting plasma potassium level. Medical emergency happened if there are abnormalities in ECG, or the plasma potassium level is greater than 6.5 mEq/L, so the calcium therapy is indicated to help prevent the development of potential lethal arrhythmias. Bolus of insulin and dextrose given by intravenous drip, insulin and glucose is the fastest acting drug that shifts potassium into the cells by stimulating the activity of Na^+-H^+ antiporter which leads to activation of the Na^+-K^+ ATPase, causing an electrogenic influx of potassium. Salbutamol inhalation as beta-adrenergic agonists also activates NaK-ATPase and cause potassium to shift into cells. We give furosemide as loop diuretics, in combination with saline infusion to ensure the delivery of sodium to the distal nephron can promote renal potassium excretion in patients with normal kidney functions. Another treatment calcium polystyrene sulfonate oral binding resins was given as it acts by a cumulative process throughout the gastrointestinal tract, removing potassium ions which are excreted in the faeces[6, 13]. Additional therapy such as corticosteroid and hydroxychloroquine were given to treat SLE.

4. Conclusion

Lupus nephritis is a complication that often occurs in SLE patients characterized by impaired kidney function. Impaired renal excretory function can lead to high potassium levels in body which may be life threatening. Hyperkalaemia should be suspected in lupus nephritis patient as it comes somehow with non-specific symptom. Other complications associated with SLE should be identified and managed. The weakness of this case report is we didn't performed any renal biopsy to identify the stage of LN.

5. Declarations

- Funding: No funding sources
- Conflict of interest: None declared
- Ethical approval: Not required

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