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Case Report

Successful treatment of an urgent dialysis patient with uremic encephalopathy

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ABSTRACT

Diabetes has become a global pandemic and is believed to be one of the most common risk factors for chronic kidney disease, which leads to end-stage renal disease, of which a significant number will develop, requiring renal replacement therapy. Uremic encephalopathy is the most frequent complication of untreated chronic kidney disease, is defined as cerebral dysfunction due to toxin accumulation. If this occurs, renal replacement therapy, which is urgent dialysis, is essential for lowering the patient's mortality. We report a 48-year-old female patient with uncontrolled type 2 diabetes who presented with delirium, poor physical performance and muscle mass loss.

Keywords: Diabetes, End-stage renal disease, Uremic encephalopathy, Urgent dialysis

INTRODUCTION

According to kidney disease: improving global outcomes (KDIGO) 2022, 40% or more of people with diabetes will develop chronic kidney disease (CKD) with a significant number developing kidney failure requiring dialysis or transplantation. The International Diabetes Federation estimated that 537 million people would have diabetes by 2021. As is commonly known, CKD is defined as a decreased estimated glomerular filtration rate (eGFR) of 90 ml/min/1.73 m^2 and abnormalities of kidney structure present for >3 months, with implications for health. CKD is classified based on cause, GFR category, and albuminuria category, abbreviated as CGA.[1]

Renal fibrosis plays a central role in the pathogenesis of diabetic kidney disease (DKD), along with changes in renal hemodynamic, oxidative stress, inflammation, hypoxia, and an overactive renin-angiotensin-aldosterone system (RAAS).[2]

Uremic encephalopathy refers to a variety of central nervous system abnormalities caused by impaired kidney function, which can occur as a result of either progressive CKD or an acute kidney injury (AKI). The syndrome is likely caused by uremic solute retention. Patients presenting with uremic syndrome typically complain of nausea, vomiting, fatigue, anorexia, weight loss, muscle cramps, pruritus, or changes in mental status**,** whereas urgent dialysis is required for the patient.[3]

CASE REPORT

A 48-year-old female with prior uncontrolled type 2 diabetes (T2DM) presented to the emergency department in a delirious state almost 4 hours and looked pale. No previous incidents, collisions, seizures or bleeding were reported. The patient has been taking metformin 500 mg once daily. During this time, medication from the public health care system was consumed, but there was no routine control in place during these five years. No urinary issues were noted. A non-fasting blood glucose test was performed immediately, and the result was 106 mg/dl.

Upon physical examination, the patient had a body mass index (BMI) of 18.02, which fell within the underweight range. She had hypotension with tachycardia, afebrile, with her respiration rate was 24 breaths/min, and oxygen

saturation was 98% at 3 litres/min with the nasal cannula. Her conjunctivae were pale. The chest examination revealed the lungs were clearly bilaterally, no wheezes, rales, or rhonchi, no murmurs or gallops were detected. Abdominal examination revealed the abdomen non-distended, normal active bowel sounds, soft, no hepatosplenomegaly, with no enlarged kidneys and distended bladder were palpable. Gynaecologic examination was also unremarkable. No pitting oedema were found. A vasoconstrictor drug was prescribed after a fluid challenge was unsuccessful.

Laboratory findings revealed, blood urea nitrogen (BUN) 239 mg/dl, level of serum creatinine (sCr) 10.4 mg/dl with estimated glomerular filtration rate (eGFR) 4 ml/min/1.73m^2, sodium serum (Na^+) 129 mmol/l, potassium serum (K^+) 6.3 mmol/l, chloride serum (Cl^-) 98 mmol/l, haemoglobin (Hb) 6.1 g/dl, leukocyte 8.81×10^3 u/l, platelet 338×10^3 u/l, with the interpretation of arterial blood gas was metabolic acidosis (pH=7.01). She was then strongly encouraged to participate in dialysis with blood transfusion during dialysis. Initially, the family refused to allow the patient to undergo urgent dialysis, but after receiving more information related to dialysis, they eventually agreed. After the initial hemodialysis, the patient's consciousness improved significantly, and she was able to communicate with the physicians and family members. Hereafter, the patient's kidney function monitored on occasion, laboratory yielded BUN 169 mg/dl then decreased 89 mg/dl, sCr 6.6 mg/dl then decreased 3.3 mg/dl, with eGFR 7 ml/min/1.73 m^2 then increase 17 ml/min/1.73 m^2, and Hb 8 g/dl. Furthermore, dialysis would be scheduled based on indications and guidelines.

DISCUSSION

Blood vessels in the kidneys can be damaged by high blood glucose. When the blood vessels are damaged, they perform less effectively. Numerous individuals with diabetes also develop high blood pressure, which can cause kidney damage. Regular monitoring of the kidneys' function is essential. This is why patients who progress to stage 3 or beyond of DKD may benefit from referral to nephrologist due to the disease's complexity and the risk of adverse outcomes. CKD is linked to numerous complications with other organs and has a negative impact on our body. This contributes to morbidity and mortality. Cardiovascular disease, electrolyte imbalance, anemia, acid-base balance abnormalities, and uremic syndrome are examples of complications.[4,5] The situation appears to be like this with regard to this particular patient.

A joint group of American Diabetes Association (ADA) and KDIGO representatives reviewed and developed a series of consensus statements to guide clinical care based on the ADA and KDIGO guidelines. The published guidelines align on CKD screening and diagnosis, glycemia monitoring, lifestyle therapies, treatment goals, and pharmacologic management. Comprehensive care with pharmacotherapy that improves kidney outcomes is recommended. Also, it is written that metformin is recommended for patients with type 2 diabetes (T2DM), CKD, and eGFR ≥30 ml/min/1.73 m^2. However, the dose should be reduced to 1,000 mg daily in patients with eGFR 30–44 ml/min/1.73 m^2 and in some patients with eGFR 45–59 ml/min/1.73 m^2 who are at high risk of lactic acidosis.[2] If this is not also observed, the decrease in renal function can occur sooner, moreover, it hastens kidney failure. The most common complication is uremic encephalopathy.[6]

Changes in mental status associated with T2DM are also difficult to diagnose due to the prevalence of overlapping and confounding conditions such as diabetic ketoacidosis, hyperosmolar hyperglycaemic state, lactic acidosis, and hypoglycaemia. In this study, the patient's blood sugar level was normal and there was no prior trauma that caused delirium. For DKD, as kidney failure progresses, there is decreased hydrogen ion secretion and impaired ammonium excretion, followed by the accumulation of phosphate and additional organic acids. In turn, the resulting increased anion-gap metabolic acidosis may lead to hyperventilation, lethargy, anorexia, muscle weakness, and changes in mental status, the result of which is delirium. Patients with CKD also experience significant muscle mass loss, weakness, and diminished physical performance.[7,8] In this case, we observed the patient as underweight with significant muscle mass loss, had changes in mental status, elevated levels of urea and creatinine in the blood, and blood gas analysis indicated metabolic acidosis. Aside from that, urea is one of the waste products of the kidneys. Urea concentrations rise and cross the blood-brain barrier (BBB), also resulting in uremic encephalopathy.[9]

In critically ill patients with kidney failure, absolute indications for urgency dialysis include severe acidosis (pH<7.14), fluid overload with oliguria unresponsive to diuretics, hyperkalemia (>6 mEq/l), pulmonary oedema, and signs/symptoms of uremia (with approximately the level of BUN >100 mg/dl).[10,11] On this basis, the patient is advised to undergo urgent dialysis.

Consultation and discussion with a nephrologist are essential for providing a more detailed perspective on CKD and renal replacement therapy in order to reduce hospitalization and mortality. It is also expected that there will be a discussion about dispelling myths that may still exist in society.[12]

CONCLUSION

Medical professionals should be aware that the prevalence of CKD leading to ESRD rises in association with the number of patients diagnosed with diabetes. Patients with diabetes-related CKD should be monitored more closely and educated promptly about their disease, particularly those who have already experienced kidney failure, as the probability of undergoing dialysis is quite significant. It is

beneficial to provide education on a step-by-step basis because the myth of dialysis is quite frightening in today's society.

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