Is a pH Of 6.73 Compatible With Life?

M Yaprak, Z Kemeç, E Çetinkaya, R Dayanan, O Çeliker, O Sünger

Citation

M Yaprak, Z Kemeç, E Çetinkaya, R Dayanan, O Çeliker, O Sünger. *Is a pH Of 6.73 Compatible With Life?*. The Internet Journal of Nephrology. 2015 Volume 10 Number 1.

DOI: 10.5580/IJNE.25148

Abstract

In patients with metabolic acidosis hemodialysis (HD) threapy should be applied if the acidosis is too deep (pH<7.1) or it cannot be taken under control despite conservative treatment. We present a chronic kidney disease (CKD) patient who was brought to the emergency room with severe metabolic acidosis (pH 6.73) and treated with emergency HD without any sequela. The 76 year old male patient was brought to the emergency room due to mental fog, disruption in general condition and respiratory distress. He had a medical history of low clearance CKD. Physical examination revealed mental fog, blood pressure of 60/40 mmHg, respiratory rate per minute of 20 and that he was dehydrated. Isotonic saline 500 cc was given and the infusion was started at 200 cc/hour. Arterial blood gas analysis showed pH:6.73, pCO2:23 mmHg, HCO3 2,9 mmol/L and lactate:3,1 mmol/L. HD was performed within half an hour. He underwent HD on the following 3 consecutive days. He completely recovered, was planned to continue HD 3 times a week and discharged from hospital on the 10th day of his hospitalization. In conclusion; Metabolic acidosis is an important and urgent HD indication in patients with CKD. We showed that pH 6.73 is compatible with life by presenting this case.

INTRODUCTION

In the treatment of renal impairment, despite conservative precautions, hemodialysis therapy (HD) can be necessary in some patients (1). HD treatment is usually used for one or more reasons in these patients. Emergency hemodialysis indications include hyperkalemia, hypervolemia, metabolic acidosis, hypercalcemia, hyperuricemia, hyperphosphatemia, metabolic alcalosis, hyponatremia, intoxication due to medicine or toxic substances and occurance of uremic symptoms or findings such as neurological (uremic encephalopathy), cardiovascular (pericarditis) and gastrointestinal (inappetency, nausea, vomiting) (1-4). HD threapy should be applied in patients with metabolic acidosis, if acidosis is very severe (pH<7.1) or it cannot be taken under control despite conservative treatment (1,3). We present a CKD patient who was brought to the emergency room with severe metabolic acidosis (pH 6.73) and treated with emergency HD without any sequela.

CASE

A seventy-six year old male patient was brought to the emergency room due to mental fog, disruption in general condition and respiratory distress. He had a medical history of low clearance CKD. In the history obtained from his son, we learned that he did not use prescribed medicines for the last 20 days and that his general condition has worsen for the last 3 days. On physical examination we found that his general condition was bad, he had mental fog, his blood pressure was only 60/40 mmHg, his respiratory rate per minute was 20 and he was in dehydrated condition. After taking blood for examination, isotonic saline 500 cc was given and the infusion was started at 200 cc/hour. The blood gas showed a pH:6.73, pCO2:23 mmHg, HCO3 2,9 mmol/L and lactate:3,1 mmol/L. Hemodialysis was planned. Until the HD treatment, 100 mEq (10 ampoules) NaHCO3 was given intravenously (IV). The blood pressure was measured as 80/50 mmHg after IV liquid and NaHCO3 treatment.

HD was performed within half an hour right after his emergency treatment. His results were as follow; urea 437 mg/dL, kre 20.9 mg/dL, sodium 146 mEq/L, potassium 6.1 mEq/L, calcium 10.2 mg/dL and phosphore17.7 mg/dL. The HD order was given as follows; time: 3 hours and blood flow rate: 200 ml/min. Ultrafiltration wasn't performed. Blood gas was taken again towards to the end of HD and pH:7.27, pCO2:45 mmHg, HCO3:20.6 mmol/L and lactate:2,2 mmol/L was obtained. HD was ended after 3 hours. Isotonic infusion was continued at a dose of 100 cc per hour. The blood pressure stabilized at about 100/60

mmHg without any inotrope support. 4 hours after HD, blood gas was taken again because of tachypnoe. Severe metabolic acidosis (pH:7.23, pCO2:9.4 mmHg, HCO3:3.9 mmol/L and lactate:3.1 mmol/L) was found but HD therapy was not performed because of risk of a dialysis disequliubrium syndrome. 100 mEq (10 ampoule) NaHCO3 was given IV in five minutes, NaHCO3 infusion was started at adose of 20 mEq per hour.

Tachypnoe and methabolic acidosis decreased after 4 hours. (pH:7.39, pCO2:21.7 mmHg, HCO3:13.0 mmol/L and lactate:1.2 mmol/L) (Table). The patient underwent HD the following 3 consecutive days. He completely recovered, was planned to continue HD 3 times a week and discharged from hospital on the 10th day of his hospitalization.

Table 1

Blood Gas Analysis of Patient

	pH	pCO2*	HCO3**	Laktat**
Application Time	6,73	23	2,9	3,1
After Dialysis	7,27	45	20,6	2,2
4 Hours After Dialysis	7,23	9,4	3,9	3,1
8 Hours After Dialysis	7,39	21,7	13,0	1,2

* mmHg

** mmol/L

DISCUSSION

HD should be applied during occurence of hyperkalemia, hypervolemia, metabolic acidosis, hypercalcemia, hyperuricemia, hyperphosphatemia, metabolic alcalosis, hyponatremia, intoxication due to medicine or toxic substances and occurance of neurological (uremic encephalopathy), cardiovascular (pericarditis) and gastrointestinal (inappetency, nausea, vomiting) symptoms or findings (1-4). HD is applied for one or more reasons (1). In our case, emergency HD was performed due to serious metabolic acidosis (pH 6,73), uremic encephalopathy (urea 437 mg/dL, kre 20.9 mg/dL) and hyperpotassemia (potassium 6.1 mEq/L).

Acid-base balance is normally provided by excretion daily acid from kidneys (5). The elimination of this acid load is basicly provided by hydrogen and amonium excretion by urine. When number of nephrons decrease in CKD, the definite acid excretion is continued by increasing amonium ion excretion per nephron in the beginning (5). However, when the glomerular filtration (GFR) rate decreases under 40-50 ml/min, total amonium excretion starts to decrease (5-7). The obvious effect of decrease in GFR is progressive metabolic acidosis due to increase in hydrogen ions (5,7,8). Alkali therapy is recomended as HCO3 level will be over 22 mEq/L in patients with CKD (9-10). Metabolic acidosis was serious (pH:6,73 HCO3 2,9 mmol/L) in our patient and renal function tests as well as potassium level were very high. This is why we applied HD with parenteral NaHCO3 treatment.

It is recomended that the duration of HD should be short, blood flow rate and surface area should be kept low in first HD because of dialysis disqulibrium risk (11,12). Thus, the period is 2-2.5 hours and blood flow rate is 150-250 ml/min during the first HD (13). However in some situations such as hyperkalemia due to rhabdomyolysis or severe metabolic acidosis this period and blood flow rate may not be enough. In such situations extending the period or increasing blood flow rate carries the potential dialysis disequlibrium risk. Therefore, we used a dialysis period of 3 hours by keeping blood flow rate at 200 ml/min with blood gas control. We applied parenteral NaHCO3 treatment with close blood gas control. Dialysis disequlibrium syndrome was not observed in our patient.

Some clinicians are afraid that HD cannot be tolerated because of low blood pressure in hypotensive patients. However, especially in patients with severe metobolic acidosis, the reason of hypotension can be the negative effect of acidosis on cardiac function (14). Thus, necessary approaches including HD should be applied to improve acidosis in these patients. We succeeded to treat out patient by appliying HD, IV NaHCO3 and fluid treatment despite the hypotension.

In conclusion; Metabolic acidosis is an important and urgent HD indication in patients with CKD. Even if CKD patients who have severe metabolic acidosis are hypotensive, they should performed HD. We showed that pH 6.73 is compatible with life by presenting that case.

References

1. Briglia, AE. Dialysis considerations in the patient with acute renal failure: ICU dialysis, in Principles and Practice of Dialysis, Henrich, WL (Ed). Third Edition, Lippincott Williams Wilkins, 2004.
2. Lameire, N, Van Biesen, W, Vanholder, R. Acute renal failure. Lancet 2005; 365:417
3. Palevsky, PM. Renal replacement therapy I: Indications and timing. Crit Care Clin 2005; 21:347)
4. Hakim, RM, Lazarus, JM. Initiation of dialysis. J Am Soc Nephrol 1995; 6:1319.
5. Warnock, DG. Uremic acidosis. Kidney Int 1988; 34:278.

6. Bailey, JL. Metabolic acidosis: An unrecognized cause of morbidity in the patient with chronic kidney disease. Kidney

Int 2005; 68(Suppl 96):S15.
7. Widmer B, Gerhardt RE, Harrington JT, Cohen JJ. Serum electrolyte and acid-base composition: The influence of graded degrees of chronic renal failure. Arch Intern Med 1979; 139:1099.
8. Uribarri J, Douton H, Oh MS. A re-evaluation of the urinary parameters of acid production and excretion in patients with chronic renal acidosis. Kidney Int 1995; 47:624.
9. Alpern RJ, Sakhaee K. Clinical spectrum of chronic metabolic acidosis: Homeostatic mechanisms produce significant morbidity. Am J Kidney Dis 1997; 29:291.
10. Mitch WE. Dietary protein restriction in patients with chronic renal failure. Kidney Int 1991; 40:326.
11. Arieff AI. Dialysis disequilibrium syndrome: Current concepts on pathogenesis and prevention. Kidney Int 1994; 45:629.
12. Ali II, Pirzada NA. Neurologic complications associated with dialysis and chronic renal insufficiency. In: Principles and Practice of Dialysis, Henrich, WL (Ed), Lippincott, Williams and Wilkins, Philadelphia, 2004, p. 507.
13. Lionel U Mailloux: Dialysis disequilibrium syndrome. www.uptodate.com
14. Mitchell JH, Wildenthal K, Johnson RL Jr: The effects of acid-base disturbances on cardiovascular and pulmonary function. Kidney Int. 1972 May;1(5):375-89.

Author Information

Mustafa Yaprak
Department of Nephrology, Batman Regional Government Hospital
Batman, Turkey
mustafayaprakdr@yahoo.com

Zeki Kemeç
Department of Internal Medicine, Batman Regional Government Hospital
Batman, Turkey

Engin Çetinkaya
Department of Emergency, Batman Regional Government Hospital
Batman, Turkey

Ramazan Dayanan
Department of Internal Medicine, Batman Regional Government Hospital
Batman, Turkey

Orhan Çeliker
Department of Internal Medicine, Batman Regional Government Hospital
Batman, Turkey

Osman Sünger
Department of Internal Medicine, Batman Regional Government Hospital
Batman, Turkey