A case report of a COPD patient presented in severe metabolic alkalosis

Ashish Shrestha[1], Naresh Gurung[1], Ashish Karthak[1], Sanjeet bhattarai[1], Rakesh Lama[1], Utsav Kumar Shrestha[1], Kishor Khanal[2], Anup Ghimire[2], Ashim Regmi[2], Sanjeet Krishna Shrestha[1]

[1]Department of pulmonary, critical care and sleep medicine, Nepal Mediciti Hospital
[2]Department of critical care, Nepal Mediciti Hospital

ABSTRACT:

Chronic Obstructive Pulmonary Disease (COPD) patients generally present with respiratory acidosis and type 2 respiratory failure. Here we present a case of 65 years old female, who is a known case of COPD and presented in our emergency department with severe metabolic alkalosis (pH 7.730, HCO3- greater than 99.9mmol/l). She was referred from other center after the development of seizure. Urinary sodium was sent which indicated the cause of metabolic alkalosis was contraction alkalosis; we then treated the patient with IV fluids, antibiotics and Mechanical Ventilator. Arterial Blood gas analysis was initially done every 4 hours till the pH was corrected. After pH was corrected the patient was extubated to intermitted Non Invasive Ventilation (NIV) for type 2 respiratory failure. With pulmonary rehabilitation we could discharge the patient with inhalers; without the need for NIV or supplementary oxygen. This is one of the rare cases where the patient presented with a very high bicarbonate level, high partial pressure of carbon dioxide in arterial blood, and high pH. The patient was successfully managed with IV fluids and mechanical ventilation.

Keywords: Chronic Obstructive Pulmonary Disease, Metabolic Alkalosis, Respiratory Failure, Non Invasive ventilation

INTRODUCTION:

Mortality associated with metabolic alkalosis is 45% in pH of 7.55, and increases to 80% if the pH is raised to 7.65[1]. To maintain the alkaline pH both the generation and maintenance of alkalosis are necessary. Alkalosis is generally caused by the loss of hydrogen ions from the gastrointestinal tract or by the kidney due to the use of loop diuretics. Alkalosis is maintained generally due to the inability of the kidney to excrete the excess bicarbonate because of hypovolemia, chloride depletion, hypokalemia, hyperaldosteronism, and renal failure. The evaluation of volume status and measurement of urinary chloride ion and plasma levels of renin and aldosterone are crucial for identifying the cause(s) of metabolic alkalosis.

Here we present a case of 65 years old female who presented with seizure and dyspnea, where we found her to be in severe metabolic alkalosis probably due to nonjudicial use of diuretics. The patient was presented with a pH of 7.730, which is among the highest alkaline pH reported in literature where life was sustained.

CASE REPORT:

AT EMERGENCY

65 years female was referred to our center for increased dyspnea and abnormal body movement. She was previously being treated for Chronic Obstructive Lung Disease (COPD), Hypertension (HTN), and clinical depression. She was on hydrochlorothiazide 25mg once a day, Sertraline 50mg once a day, Aripiprazole 5mg once a day, Mirtazapine 7.5 mg twice a day, amitriptyline 25mg once a day, Rotacap foracort (formoterol (6mcg) + budesonide (400mcg)) once a day and rotacap Tiotropium 18mcg once a day. At presentation her vitals were

Table 1. Vitals of the patient on presentation

Spo2	93% at 3 litre of oxygen
Respirator rate	32-36/ min
Blood Pressure (BP)	110/70mmhg
Heart rate	120/min
Temp	98°F

Arterial Blood Gas (ABG) revealed that the patient was in type 2 respiratory failure but in severe metabolic alkalosis with HCO3 >99.9mmol/l.

Corresponding author:
Ashish Shrestha, MD
dr.ashish.shrestha@outlook.com

Table 2. ABG on presentation

pH	7.730
Pco2	72.1mmHg
Po2	92mmHg
Hco3	>99.9 mmol/l

Pco2: partial pressure of carbon dioxide

Po2: partial pressure of oxygen

Her laboratory reports on the day of admission were as:

Table 3. Laboratory reports on presentation

Random glucose	215 mg/dl
Total protein	7.2 g/dl
Albumin	4.1 g/dl
SGPT	83 U/l
SGOT	94 U/l
Total bilirubin	0.8 mg/dl
Direct bilirubin	0.1 mg/dl
Alkaline phosphatase	63 U/l
CRP	11.5 mg/l

Nt-proBNP: N-terminal pro b-type natriuretic peptide

Due to increased respiratory effort and tachycardia the patient was intubated at Emergency and was shifted to Intensive Care Unit (ICU). We admitted the patient with intravenous (IV) antibiotics (meropenem, clindamycin), a bronchodilator (IV methylprednisolone, nebulization of budesonide), Deep Venous Thrombosis prophylaxis (Enoxaparin), Proton pump inhibitor (pantoprazole), and levetiracetam. Potassium was replaced as per replacement protocol.

BRIEF HISTORY OF PRESENT ILLNESS

It was the first month according to the Nepali calendar, our 65 years lady decided to go to the Hindu pilgrimage at Kashi, India After a few days at Kashi, she started to develop shortness of breath, and that's when she decided to return to her hometown in Nepal. After arriving home, she also had four episodes of diarrhea then she decided to go to the hospital where she was admitted to ICU; there she was treated for type 2 respiratory failure with Non Invasive Ventilator (NIV), a bronchodilator, diuretics, and IV antibiotics. After four days at ICU, she was shifted to the medical ward where she had three episodes of abnormal body movement, up rolling of eyes, tongue bite along with postictal confusion. She was then referred to our center for further management.

AT ICU

The major issues that were present in this case were type 2 respiratory failure, severe metabolic alkalosis, hypokalaemia, and seizure. Ventilatory support was adjusted to address the type 2 respiratory failure. The patient gave a history of diarrhea, use of diuretics, and severe hypokalemia at presentation; in an otherwise healthy woman we initiated the treatment with the preliminary diagnosis contraction alkalosis; further hypokalemia was aggravated by the alkalosis. Thus, the urine chloride was sent and normal saline was started at 42ml/hour. The patient had no history of seizure disorders but as she was presented with a history of abnormal body movement; a CT head was done. CT head reported age-related cerebral atrophy; we attributed severe metabolic alkalosis as a cause of the seizure. Calcium gluconate and magnesium sulfate were also replaced. Endotracheal (ET) aspirates, blood, and urine culture were also sent.

ABG was repeated four hours after the normal saline and pH was reduced to 7.653 but bicarbonate was 92 mmol/L. Around the same time, the urine chloride report was completed and was <15mmol/l and thus normal saline was increased to 84ml/hr.

Table 4. ABG after 4 hours of presentation

pH	7.653
Pco2	71.6 mmhg
Po2	153 mmhg
Hco3	92 mmol/l

Pco2: partial pressure of carbon dioxide

Po2: partial pressure of oxygen

Cardiac echo was also done: mild Tricuspid Regurgitation (TR) with normal Pulmonary artery pressure; TR pressure gradient was 16mmHg; normal right ventrile, left ventricle; Left ventricle ejection fraction was 60%; Inferior vena cava (IVC) was 12 mm with normal respiration variation. Repeat ABG was done after 4 hours of 84 ml/hr normal saline with further reduction in pH as shown below.

Table 5. ABG after 8 hours of presentation

pH	7.535
Pco2	88 mmhg
Po2	70 mmhg
Hco3	78.3 mmol/l

Pco2: partial pressure of carbon dioxide

Po2: partial pressure of oxygen

ABG was then planned 12 hourly and is follow:

Table 6. ABG after 20 hours and 32 hours of presentation

	After 20 hours	After 32 hours
pH	7.609	7.415
Pco2	46 mmhg	72.8 mmhg
Po2	52 mmhg	89 mmhg
Hco3	47.7 mmol/l	43 mmol/l
Potassium	2.7 mmol/l	3.6 mmol/l

Pco2: partial pressure of carbon dioxide

Po2: partial pressure of oxygen

After normalization of pH, normal saline was then stopped, and ABG was planned every 24 hr. As the patient has a Glasgow Coma Scale (GCS) of 15 and was on minimal ventilatory support; given the history of COPD in the patient, we planned to extubate the patient to intermitted NIV. The urine, blood, and sputum culture were all negative for any growth. In the fungal KOH of sputum, the fungal element was also not seen. We restarted her antidepressive medications after one day of extubation, the patient continued her intermitted NIV as she had type 2 respiratory failure. We shifted the patient to the ward after another 48 hr, where she had pulmonary rehabilitation and overnight NIV. With pulmonary rehabilitation, we could keep her off NIV and supplementary oxygen therapy; and was subsequently discharged on oral medications and inhalers. Her ABG on discharge was as follow

Table 7. ABG on discharge

pH	7.382
Pco2	48.5 mmhg
Po2	113 mmhg
Hco3	26.9 mmol/l

Pco2: partial pressure of carbon dioxide

Po2: partial pressure of oxygen

DISCUSSION

This patient developed metabolic alkalosis due to the non-judicial use of diuretics at the other center she was referred from, further she was already on hydrochlorothiazide as regular medicine. COPD patients presented with dyspnea are generally started with diuretics in view of reducing preload and decreasing pulmonary edema which in turn improves respiratory health. The patient also presented with diarrhea which have further compromised the volume status of the patient. On the day of admission to ICU, her IVC was only 12 mm. With the initiation of IV normal saline, her ABG improved. Her hypercapnic ABG was titrated slowly to counter her pH induced by increased bicarbonate.

The patient was hypovolemic so treatment is aimed at reversing the underlying cause. The patient was successfully managed with IV normal saline. After two days in ICU, pH was normalized and after the sixth day she was shifted to the ward. She was discharged home after a week in the ward. The patient presented with severe alkalosis but had an increased pco2 of 72.1 mmhg. We titrated pco2 slowly to counter the increased alkalosis. She was further in NIV overnight even in the ward. Her type 2 respiratory failure was addressed by a bronchodilator and most important pulmonary rehabilitation. Thanks to our physiotherapy team, with proper pulmonary rehabilitation she was discharged without the need for NIV or supplement oxygen[2].

The patient also had three episodes of seizures at other centers on the day she was referred to our center. Severe alkalosis is a well-established cause of seizures. Many cases have also been reported with severe alkalosis being associated with myoclonus[3], twitching tenany due to decrease serum calcium. Metabolic alkalosis can have central nervous system manifestations ranging from confusion to coma, peripheral neuropathic symptoms of tremor, tingling and numbness, muscle weakness and twitching, and arrhythmias to seizure, particularly when associated with hypokalemia and hypocalcemia. Tetany is thought to be due to decreased serum ionized calcium concentration and also has been demonstrated to be due to an increase in pH-dependent myofibrillar calcium sensitivity[4]. Also, severe alkalosis reduced ionized serum calcium level[5], and cases have been reported with tetany associated with alkalosis[6], thus the replacement of calcium is also essential. We decided to continue her levicetaram on discharge and have planned to decide on its discontinuation on follow-up.

Our case also presented with severe hypokalemia; hypokalemia, and metabolic alkalosis are both the entity of the same vicious cycle. metabolic alkalosis causes hypokalaemia due to the shift of potassium ion intracellularly or by the renal loss[7]. Whereas hypokalaemia induces metabolic alkalosis as hydrogen ion are exchanged for potassium[8]. Thus, the replacement of potassium and maintaining optimal potassium level is also crucial in the management of alkalosis. The patient also presented with type 2 respiratory failure. Alkalosis precipitates hypoventilation, which might not be an issue for a patient without lung diseases but for our patient who is a known case of COPD, weaning of ventilator might be difficult if alkalosis was not corrected.

Our case presented with a pH of 7.730, not the highest recorded in literature but our case was unique because as per our knowledge, none of the previously reported cases has a bicarbonate level higher than 99.9 mmol/l. In literature one of the highest pH reported was 7.87[9], induced by gastrointestinal losses. With timely intervention, the patient can be saved even though presented in non-physiological pH.

CONCLUSION

COPD patient presenting with metabolic alkalosis might not be a usual presentation, but when they do thorough medical history needs to be explored. Many COPD patients are under diuretics and if it is accompanied by other causes which compromise volume status, judicious use of diuretics is essential.

REFERENCES

1. Anderson LE, Henrich WL. Alkalemia-associated morbidity and mortality in medical and surgical patients. South Med J 1987; 80(6): 729-733. e-pub ahead of print 1987/06/01; doi: 10.1097/00007611-198706000-00016
2. Bolton CE, Bevan-Smith EF, Blakey JD, Crowe P, Elkin SL, Garrod R et al. British Thoracic Society guideline on pulmonary rehabilitation in adults: accredited by NICE. Thorax 2013; 68(Suppl 2): ii1. doi: 10.1136/thoraxjnl-2013-203808

3. Simons P, Nadra I, McNally PG. Metabolic alkalosis and myoclonus. Postgrad Med J 2003; 79(933): 414-415. e-pub ahead of print 2003/08/05; doi: 10.1136/pmj.79.933.414
4. Westerblad H, Allen DG. Mechanisms underlying changes of tetanic [Ca2+]i and force in skeletal muscle. Acta Physiol Scand 1996; 156(3): 407-416. e-pub ahead of print 1996/03/01; doi: 10.1046/j.1365-201X.1996.196000.x
5. Goyal A, Anastasopoulou C, Ngu M, et al. Hypocalcemia. [Updated 2022 May 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430912/.
6. Urbanek A, Borucka K, Ogrodowczyk-Bobik M, Kajdaniuk D, Janyga S, Marek B. Recurrent tetany in the course of metabolic alkalosis in patient with short bowel syndrome. Endokrynol Pol 2021; 72(4): 406-407. e-pub ahead of print 2021/06/01; doi: 10.5603/EP.a2021.0058
7. Aronson PS, Giebisch G. Effects of pH on potassium: new explanations for old observations. J Am Soc Nephrol 2011; 22(11): 1981-1989. e-pub ahead of print 2011/10/08; doi: 10.1681/asn.2011040414
8. Kamel KS, Halperin ML, Goldstein MB. Fluid, electrolyte and acid-base physiology e-book: a problem-based approach, Elsevier Health Sciences, 2010.
9. Tugrul S, Telci L, Yildirim A, Yanar H, Cakar N. Case Report of Severe Metabolic Alkalosis: Life-CompatibleNew Level. Journal of Trauma and Acute Care Surgery 2010; 68(3).