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A positive outcome with extremely severe metabolic acidosis: A case report

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Abstract

This is an entrancing case report of extremely severe metabolic acidosis occurred to a 46-year-old female who was admitted to intensive care medicine, Ninewells hospital. She presented with vomiting and collapsed with significant deranged metabolic acidosis. Despite carrying very guarded prognosis on admission, fortunately she survived following effective management from multidisciplinary team in the hospital. This case describes the metabolic acidosis in association with the mortality including the clinical presentation, management and the outcome.

Key words: metabolic acidosis, mortality, prognosis

Introduction

Metabolic acidosis is a common biochemical disorder frequently encountered in acute and intensive care medicine. The blood pH is maintained within narrow limits (7.35- 7.45). Acidosis (pH < 7.35) is a common feature of a number of acute and critical conditions that lead to intensive care admission. Blood pH < 6.8 is considered to be extremely severe acidosis that is incompatible with life but there are very few exceptional reported cases of survival with no long-term ill effect. Severe Metabolic acidosis universally carries poor prognostic outcome.

Case Report

A 46-year-old lady who was staying fit and well got admitted to Emergency department presenting abdominal pain, severe vomiting for past 3 days. Prior to this admission, she underwent total hysterectomy for suspected ovarian tumor a month ago but later biopsy reported as benign in nature.

On arrival to Emergency department, she was in very poor condition and unable to provide history. Clinical examination revealed marked dehydration with generalized abdominal tenderness. She was very unstable at that point with Systolic Blood pressure being less than 80 mmHg, tachycardia, and requiring 15 L of nasal oxygen. Arterial blood gas showed extremely severe metabolic acidosis: pH being 6.5 with bicarbonate 2 mmol/L, base excess more than 20 and lactate 39 mmol/L. Given this current critical condition, the patient was reviewed by intensive medial team at the Emergency department. At that point of time, the intensive care team remarked that she would barely survive with this extremely low pH even under intensive care medicine and suggested to find out the cause for severe metabolic acidosis first. Emergency department team carried out urgent CT abdomen and pelvis within 2 hours of arrival which reported promptly as small bowel adhesion resulting from recent hysterectomy operation. Once small bowel adhesion diagnosis was made, she was reviewed by surgical team and decided to undergo emergency laparotomy operation to release the adhesion band. The operation executed within 4 hours of emergency admission. Following the major operation, the patient was transferred directly to intensive care medicine in need of ventilator and inotropic support.

During her stay in intensive care medicine, she also required several sessions of hemodialysis for anuria due to acute kidney injury. Surprisingly, her medical condition as well as biochemical markers were significantly improved day by day with pH being 7.36, Bicarbonate 22mmol/L, BE -3 and lactate 1 mmol/L and finally extubated her successfully with no longer inotropes requirement from 7th Day of Intensive care medicine admission.

Subsequently, she was transferred to general surgical unit for further management where she went through physiotherapy and occupational therapy that made her independent functionally. Eventually, she was discharged home after 14 days of admission.

Discussion

In general term, metabolic acidosis is an abnormal condition in which there is too much acid in the body fluid resulting in a fall in arterial plasma bicarbonate. To put it in another way, it is a serious electrolyte disorder characterized by the body's acid-base imbalance. There are three main root causes: increase in acid production, loss of bicarbonate and failure to excrete acid by kidneys. There are two main groups of metabolic acidosis: normal or high anion gaps.

Some common examples frequently found in daily clinical practice are diabetes ketoacidosis, alcoholic acidosis, sepsis, renal failure, diarrhea, renal tubular acidosis and some poisonings such as salicylates, iron overdoses and paracetamol.

With respect to clinical presentation, it depends on underlying causes. Mild acidemia itself is asymptomatic. Symptoms such as Nausea, vomiting and malaise may occur in severe metabolic acidosis of pH < 7. The most characteristic sign is hyperpnea reflecting a compensatory increase in alveolar ventilation. Severe acute acidemia brings about hypotension, ventricular arrythmia, shock and coma that lead to death.

Diagnostic approach to metabolic acidosis can go by simple steps: perform arterial blood gas and serum electrolyte measurement first and then calculate anion gap that may help narrow down differential diagnosis.

Treatment is mainly directed at the underlying cause. For instance, haemodialysis is required for renal failure and some poisonings. Treatment of acidaemia with sodium bicarbonate (NaHCO3) is clearly indicated only in certain circumstances. When metabolic acidosis results from loss of HCO3− or accumulation of inorganic acids (i.e. normal anion gap acidosis), bicarbonate therapy is generally safe and appropriate. However, when acidosis results from organic acid accumulation (i.e. high anion gap acidosis), bicarbonate therapy is controversial; it does not clearly decrease mortality in these conditions, and there are several possible risks.

With treatment of the underlying condition, lactate and ketoacids are metabolized back to HCO3−; exogenous HCO3− loading may therefore cause an "overshoot" metabolic alkalosis. In any condition, sodium bicarbonate may also cause sodium and volume overload, hypokalemia, and, by inhibiting respiratory drive, hypercapnia. Furthermore, as HCO3− does not diffuse across cell membranes, intracellular acidosis is not corrected and may paradoxically worsen because some of the added HCO3− is converted to carbon dioxide (CO2), which does cross into the cell and is hydrolyzed to H^+ and $HCO3^-$.

Despite these and other controversies, most experts still recommend giving bicarbonate IV for severe metabolic acidosis (pH < 7.0).

Treatment requires 2 calculations (same for both conventional and SI units). The first is level to which HCO_3^- must be raised, calculated by the Kassirer-Bleich equation, using a target value for $[H^+]$ of 79 nEq/L (79 nmol/L), which corresponds to a pH of 7.10:

$79 = 24 \times PCO_2/HCO_3^-$

or

Desired $HCO_3^- = 0.30 \times PCO_2$

The amount of sodium bicarbonate needed to achieve that level is-

$NaHCO_3$ required (mEq/mmol) = (desired $[HCO_3^-]$ − observed $[HCO_3^-]$) × 0.4 × body weight (kg)

For example, a 70-kg man has severe metabolic acidosis with a pH of 6.92, PCO_2 40 mmHg and HCO_3^- of 8 mEq/L (8 mmol/L). The target bicarbonate level needed to achieve a pH of 7.10 is 0.30 × 40 = 12 mEq/L (12 mmol/L). This level is 4 mEq/L (4 mmol/L) more than his current bicarbonate level of 8. To increase bicarbonate by 4, multiply 4 by 0.4 times 70 (the body weight), giving a result of 112 mEq (112 mmol) of HCO_3^-. This amount of sodium bicarbonate is given over several hours. Blood pH and HCO3−levels can be checked 30 minutes to 1 hour after administration, which allows for equilibration with extravascular HCO_3^-.

Alternatives to sodium bicarbonate include:

- Lactate, either in the form of lactated Ringer's solution or sodium lactate (is metabolized mEq for mEq to bicarbonate when liver function is normal);
- Sodium acetate (metabolized mEq for mEq to bicarbonate when liver function is normal);
- Tromethamine, an amino alcohol that buffers both metabolic (H+) and respiratory (carbonic acid [H2CO3]) acid;
- Carbicarb, an equimolar mixture of sodium bicarbonate and carbonate (the latter consumes CO2 and generates HCO3−); and
- Dichloroacetate, which enhances oxidation of lactate.

These alternatives do not offer a proven benefit over sodium bicarbonate alone and can cause complications of their own.Potassium (K^+) depletion, common in metabolic acidosis, should be identified through frequent serum K^+ monitoring and treated as needed with oral or parenteral potassium chloride. Concerning mortality, the higher the metabolic acidosis, the higher the mortality rate is. One of the retrospective clinical study found that the mortality rate was 90% for patients with pH < 7 and cardiac arrest before admission while there is in the range of 75-90% mortality for very sick patients with pH < 7 but not cardiac arrest prior to intensive care admission.

Another retrospective study conducted in critical care unit also found that Critically ill patients with metabolic acidosis are twice as likely to die as patients who do not have metabolic acidosis. A statistical analysis confirmed that both lactate and Strong ion gap (SIG) were strong independent predictors of hospital mortality. The authors recommend that both should be monitored in critically ill patients.

Renal failure is one of the common causes of metabolic acidosis. One study on renal patients was concluded that uncorrected severe metabolic acidosis, defined by serum bicarbonate concentrations less than 20 mmol/L, is associated with a 10-year risk for coronary heart disease more than 20% and high overall mortality in patients on renal replacement therapy.As discussed above, as there are several causes of metabolic acidosis, bear in mind the fact that finding out the root cause is of great importance because treatment of metabolic acidosis counts on its underlying pathology.

Conclusion

Severe Metabolic acidosis carries significant high mortality. Finding out the underlying cause is of great importance because treatment is mainly directed at the root cause. Studies concluded that patients with pH less than 7 have slim chance of survival. However, our case may be one of the very rare cases who survived with pH 6.5. The outcome may be related to her age, early arrival to hospital where she got early diagnosis and prompt and effective management.

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