

Unknown use of end-tidal CO₂ in metabolic emergencies in pediatric patients

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ABSTRACT

The authors describe two cases of metabolic acidosis, caused by diabetic ketoacidosis in the first case and by dehydration following gastroenteritis in the second one. Both patients were followed with noninvasive end-tidal CO_2 (ETCO $_2$) monitoring. A correlation between ECO_2 and PCO_2 and PCO_3 has been established in the literature. Noninvasive $ETCO_2$ is used in only 5–6% of metabolic emergencies. In contrast, users described its use as easy and convenient.

 $\textbf{Key words} : \texttt{end-tidal CO}_{2}, \texttt{metabolic emergencies}, \texttt{pediatric patients}, \texttt{Noninvasive ETCO}_{2}$

INTRODUCTION

Capnography is the measurement of the partial pressure of CO₂ in the air exhaled by the patient. We distinguish end-tidal CO₂ (ETCO₂), which represents the maximum numerical value at the end of the expiration, and the shape of the CO₂ wave, also called capnogram, which is a graphic presentation of the inhaled and exhaled concentration or partial pressure of CO₂. Changes in ETCO₃ values are useful for assessing the severity of a pathology as well as the response to treatment. This measurement is more commonly performed during mechanical ventilation but could also easily be used noninvasively.[1] The authors describe how following ETCO² allows monitoring of treatment for dehydration in two common pediatric situations: diabetic ketoacidosis and acute gastroenteritis. This technique allows a noninvasive, quantified metabolic monitoring of the rehydration process.

CLINICAL CASE 1

A 15-year-old patient was admitted for abdominal pain, nausea, and vomiting in a diabetes setting. The first biological tests revealed the following: arterial pH, 7.08; base excess (BE), -20 mmol/L; pCO₂, 28.4 mmol/L; HCO₃-, 8.4 mmol/L;

glycemia, 23.1 mmol/L; blood betahydroxybutyrate, 7.6 mmol (Nl < 0.5)/L. Ketoacidosis was treated with a hydration of 3 L/m² and a continuous infusion of rapidacting insulin. Continuous noninvasive monitoring of ETCO₂ was performed using a Viamed Capnometer (Viamed Limited, Keighley, United Kingdom) using nasal cannulas. The initial value was 17 mmHg, which increased to 32 mmHg at the end of continuous IV insulin infusion. During this 6-h period, we observed a correlation between ETCO₂ and pH, PCO₂, HCO₃⁻, and respiratory rate (Figure 1).

CLINICAL CASE 2

An 8-month-old patient was admitted to the emergency department with fever and diarrhea for 4 days. The patient exhibited clinical signs of dehydration with deep apathy and a weight loss approaching 10% of the body weight. A first measurement of capillary blood gases showed a pH of 7, 32, pCO₂ of 24 mmol/L, HCO₃⁻ of 16 mmol/L and EB of -12. A rehydration by nasogastric tube was tried but discontinued because of failure (diarrhea of >20 cc/kg/h and a drop of capillary pH to 7.24). Intravenous rehydration was initiated with ETCO₂ monitoring. The initial value was 23 mmHg, which increased to 30 mmHg.

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10.2478/jtim-2019-0015

Quick Response Code:



By then, the patient was transferred to a normal ward for further rehydration. During this 6-h period, we observed a correlation between ETCO₂ and pH, pCO₂, HCO₃⁻, and respiratory rate (Figure 2).

DISCUSSION

These two observations illustrate the way in which continuously monitored ETCO₂ reflects a significant degree of correlation with the metabolic variations of these two patients. The improvement in the hydration

state resulted in an increase in bicarbonate. As a result of this increase, the respiratory compensation fades, which results in a decrease in respiratory rate and a proportionate increase in CO₂ and ETCO₂ values.

A survey conducted in a network of pediatric emergency departments in the United States and Canada showed that 88% of emergency departments have access to ETCO₂ to monitor intubated patients and 53% have a noninvasive system. Only 20% of these hospitals use it for moderate sedation, 16% for trauma and 6% for acid-base metabolic disorders.^[2]

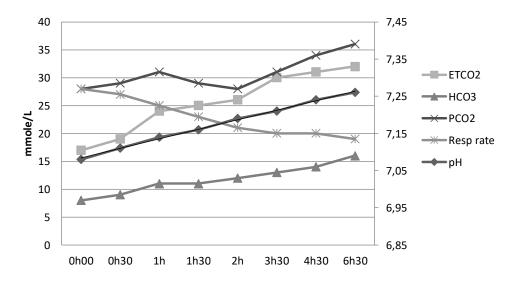


Figure 1: Correlation between ETCO₂, and pH, pCO₂, HCO₃, values, and respiratory rate during rehydration during ketoacidosis rehydration. Improvement of metabolic acidosis following administration of fluid and insulin results in elevated bicarbonate with a decrease in respiratory compensation marked by a decrease in respiratory rate and an increase in CO₂ and ETCO₂.

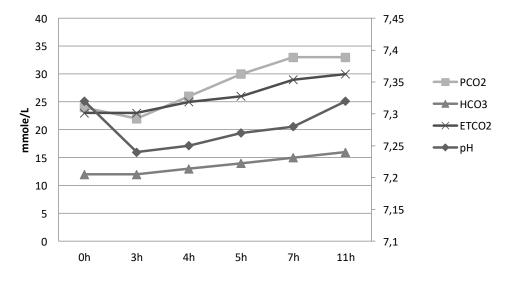


Figure 2: Correlation between ETCO₂ and pH, pCO₂, HCO₃, values during post-gastroenteritis rehydration. Rehydration results in increase of bicarbonate and a decreased respiratory compensation with a decrease in respiratory rate and elevation of PCO₂ and ETCO₂.

The correlation was established between noninvasive ETCO, and capillary PCO, with a difference of ~2 mmHg (P<0.001). ^[3] The ventilatory response in moderate ketoacidosis without lactic acidosis was investigated by showing a correlation between pH and arterial CO,.[4] The same author has shown a relationship between the measurement of arterial CO, and bicarbonate and proposed the following equation: $PaCO_2 = 1.5 \times [HCO_3]$ + 8.^[5,6] The use of ETCO₂ has been studied in ketoacidosis, where a correlation is observed among ETCO2, pH, and bicarbonate (P < 0.001).^[7] It was also shown that the risk of cerebral edema was related to bicarbonate at time 0 and remained correlated at the sixth hour. [7] Similarly, a multicenter trial evaluating the risk of cerebral edema in pediatric patients with ketoacidosis showed a decrease in the relative risk of edema of 3.4 (95%CI: 1.9–6.3; *P*<0.001) for every decrease of 7.8 mmHg of PCO₂.^[8] This shows again the interest of the instantaneous monitoring of ETCO, in this pathology.

In diarrhea with vomiting and dehydration, the measurement of HCO₃ is an important element in assessing the degree of dehydration of a pediatric patient. ETCO₂ and bicarbonate were independently correlated in gastroenteritis dehydration. A good discriminating value based on ROC curves has been demonstrated for bicarbonate thresholds of 13, 15, and 17 mmol/L (AUC at 0.94, 0.95, and 0.90, respectively).^[9]

The use of ETCO₂ is perceived as easy by pediatric emergency physicians.^[3] The reason given for not using it is the lack of equipment. The same survey was conducted in pediatric intensive care units and showed the same results. The 100% of respondents find it easy to use and 5% used it in acid-base disorders.^[10]

CONCLUSIONS

In agreement with the literature, the monitoring of $ETCO_2$ is uncommon in acid-base disorders $(5-6\%)^{[3,10]}$ but is a

reliable, easy-to-use, and noninvasive and does not require painful repeated capillary punctures for the pediatric patients. Further studies are necessary to highlight this technique among emergency practitioners for the treatment of metabolic disorders.

Conflict of Interest

None declared.

REFERENCES

- Maconochie IK, de Caen AR, Aickin R, Atkins DL, Biarent D, Guerguerian AM, Kleinman ME, et al. Part 6: Pediatric basic life support and pediatric advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2015; 95: e147 68.
- Langhan ML, Chen L. Current Utilization of Continuous End-Tidal Carbon Dioxide Monitoring in Pediatric Emergency Departments. Pediatr Emerg Care 2008; 24: 211-3
- Abramo TJ, Cowan MR, Scott SM, Primm PA, Wiebe RA, Signs M. Comparison of pediatric end-tidal CO2 measured with nasal/oral cannula circuit and capillary PCO2. Am. J. Emerg Med 1995; 13(1): 30-3.
- Fulop M. The ventilatory response in uncomplicated diabetic ketoacidosis. Crit Care Med 1977; 5: 190-2.
- Fulop M. A guide for predicting arterial CO2 tension in metabolic acidosis. Am J Nephrol 1997; 17: 421-4.
- Guh JY, Lai YH, Yu LK, Shin SJ, Tsai JH. Evaluation of ventilatory responses in severe acidemia in diabetic ketoacidosis. Am J Nephrol 1997; 17: 36-41.
- Durr JA, Hoffman WH, Sklar AH, el Gammal T, Steinhart CM. Correlates of Brain Edema in Uncontrolled IDDM. Diabetes 1992; 41: 627-32.
- Glaser N, Barnett P, McCaslin I, Nelson D, Trainor J, Louie J, et al. Risk factors for cerebral edema in children with diabetic ketoacidosis. The Pediatric Emergency Medicine Collaborative Research Committee of the American Academy of Pediatrics. N Engl J Med 2001; 344: 264-9.
- Nagler J, Wright RO, Krauss B. End-tidal carbon dioxide as a measure of acidosis among children with gastroenteritis. Pediatrics 2006; 118: 260-7.
- Langhan M. Continuous end-tidal carbon dioxide monitoring in pediatric intensive care units. J Crit Care 2009; 24: 227-30.

How to cite this article: Redant S, Angoulvant F, Honore PM, Attou R, Biarent D, De Bels D. Unknown use of end-tidal CO2 in metabolic emergencies in pediatric patients. J Transl Int Med 2019; 7: 76-8.