Case report

CO2 measurement for the early differential diagnosis of pulmonary embolism-related shock at the emergency department: A case series

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ABSTRACT

Objective: An early differential diagnosis is mandatory when facing a patient with clinical shock of unclear aetiology, in order to guide proper treatment. We assessed if the expired CO2 measurement and alveolar-arterial CO2 calculation could improve the differential diagnosis of shock during its initial presentation, particularly in separating pulmonary embolism from other causes of shock.

Methods: We analysed the charts of 12 patients who presented with clinical shock and had end-tidal CO2 (EtCO2) and arterial CO2 partial pressure (PaCO2) measurements.

Results: In cases with pulmonary embolism-related shock (n = 3), the gradient between PaCO2 and EtCO2 was increased (37 vs 0.2 mmHg). There was a similar trend for a higher PaCO2 value (60 vs 32.2 mmHg) and a lower EtCO2 value (23 vs 32 mmHg).

Conclusion: An initial CO2 measurement might be an easily available tool for the early diagnostic workup of clinical shock.

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1. Introduction

When managing clinical shock, either in the pre-hospital or emergency department setting, it is always a challenge to assess the appropriate diagnosis, treatment, and prognosis as rapidly as possible. Physicians dealing with respiratory emergencies and life-threatening clinical situations may encounter difficult situations where the initial clinical signs, symptoms, and monitoring do not indicate any clear aetiology while the patient’s vital signs continue to deteriorate. Moreover, diagnostic tests like echocardiography or spiral CT of the chest may not be immediately accessible due to the patient’s instability or local conditions.

The CO2 measurement can show a correlation between decreased CO2 transport in the blood and hemodynamic compromise. The measurement of either expired CO2 or arterial CO2, or the difference between these two measures, has been studied since the 1970s as a surrogate marker of shock, but the potential of this marker for improving the diagnostic or prognostic evaluation of shock has yet to be confirmed in the clinical literature [1]. Massive pulmonary embolism (PE) results in low CO2 transport, due to hemodynamic compromise, together with an alveolar dead space effect (increase in poorly perfused, but well ventilated lung areas). Both conditions contribute theoretically to additional decreased expired CO2 elimination at the mouth and an increased difference between arterial and alveolar CO2 [2,3]. We aimed to assess if expired CO2 and/or alveolar-arterial CO2 gradient measurements can improve the differential diagnosis of shock by distinguishing PE from other aetiologies during the early clinical presentation in an emergency setting.

2. Methods

We analysed a non-consecutive and monocentric case series. Patients were considered for analysis following three conditions: they presented with arterial hypotension (defined as systolic arterial blood pressure <90 mmHg) and clinical signs of circulatory shock (neurological alteration, marbled skin, cyanosis, prolonged capillary refill time); there was no clear aetiology at the time of admission to the emergency department or the pre-hospital setting; end-tidal CO2 (EtCO2), from a commercial time-based capnography, and arterial CO2 partial pressure (PaCO2) were measured at admission, before the establishment of a final diagnosis. Both spontaneously ventilating and invasively ventilated patients were included.

All patients underwent a cardiac echography as first bedside
diagnostic test. The final diagnosis was established using gold-standard procedures. In particular, a spiral CT was used to confirm PE when patient’s clinical condition was more stable.

The end point of this case series was the correlation between the initial \(P(a-Et)CO_2\) gradient and the diagnosis of PE induced-shock.

3. Results

The expired \(CO_2\) was measured in 12 patients admitted to the emergency department with circulatory shock. Their main initial characteristics, the initial management, and final diagnosis are presented in Table 1. The median age was 66 years and the sex ratio was 1:1. Massive PE was the cause of the shock in three patients. Other shocks were defined as septic (\(n = 4\)), cardiogenic (\(n = 2\)), obstructive due to aortic root dissection and secondary pericardial tamponade (\(n = 2\)), and neurogenic (\(n = 1\)). The final aetiologies of the shocks were confirmed by computed tomography pulmonary angiography (PE), medullar MRI (vasoplegia by medullary section), isolation of microbiological pathogens (sepsis), and cardiac echography (aortic root dissection, cardiogenic shock). At the time of expired \(CO_2\) measurement, all patients required high oxygen supplement. Seven patients were spontaneously ventilating and five were mechanically ventilated, for cardiorespiratory arrest (\(n = 3\)), respiratory failure (\(n = 1\)), and coma from neurological origin (\(n = 1\)). No patient suffered from chronic respiratory failure.

The patients’ \(CO_2\)-related measurements and characteristics are presented in Table 2 for patients with and without PE. The \(PaCO_2\) was higher and the \(EtCO_2\) lower in cases with PE, but the differences were not significant. However, patients with PE-related shock had a significant increase in the \(P(a-Et)CO_2\) gradient (37 vs 0.2 mmHg). Jugal venous distention was observed in the three patients with massive PE and in one patient with shock due to a different aetiology. Survival and mechanical ventilation requirement rates did not significantly differ between the two groups.

4. Discussion

We presented a short series of 12 patients in circulatory shock without a clear aetiology at the pre-hospital or emergency department admission. The mean difference between arterial and expired \(CO_2\) measurement, all patients required high oxygen supplement. Seven patients were spontaneously ventilating and five were mechanically ventilated, for cardiorespiratory arrest (\(n = 3\)), respiratory failure (\(n = 1\)), and coma from neurological origin (\(n = 1\)). No patient suffered from chronic respiratory failure.

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Table 1

| Age | Sex | Arterial pressure (mmHg) | Heart rate (bpm) | GCS | pH | Lactate (mmol/l) | Initial \(PaCO_2\) (mmHg) | Initial \(EtCO_2\) (mmHg) | \(P(a-Et)CO_2\) gradient (mmHg) | Diagnosis | Outcome at 30 days |
|-----|-----|--------------------------|------------------|-----|----|-----------------|--------------------------|--------------------------|----------------------------|-----------|-------------------|
| 1   | 61  | 70/50                    | 155              | 3   | 7.1| 11.4            | 37                       | 39                       | -2                        | Septic shock | Death             |
| 2   | 47  | 70/40                    | 140              | 15  | 7.3| 5.9             | 38                       | 45                       | -7                       | Septic shock | Death             |
| 3   | 49  | 50/30                    | 50               | 3   | 6.69| 18              | 65                       | 37                       | 28                       | Massive PE  | Survival          |
| 4   | 73  | 80/40                    | 140              | 14  | 7.41| 10.6            | 14                       | 20                       | -6                       | Septic shock | Death             |
| 5   | 73  | 45/30                    | 44               | 3   | 7.4| 3.2             | 23                       | 22                       | 1                        | Aortic root dissection & tamponade | Survival |
| 6   | 30  | 75/50                    | 123              | 3   | 6.82| 13.1            | 83                       | 20                       | 63                       | Massive PE  | Survival          |
| 7   | 66  | 65/43                    | 70               | 3   | 7.23| 11.5            | 54                       | 44                       | 10                       | Cardiogenic shock (STEMI) | Death     |
| 8   | 67  | 50/30                    | 41               | 14  | 7.47| 3.7             | 29                       | 28                       | 1                        | Aortic root dissection & tamponade | Survival |
| 9   | 54  | 75/30                    | 40               | 15  | 7.3| 0.8             | 20                       | 21                       | -1                       | Cardiogenic shock (beta-blocker overdose) | Survival |
| 10  | 69  | 70/30                    | 108              | 14  | 7.46| 1.1             | 32                       | 12                       | 20                       | Massive PE  | Survival          |
| 11  | 66  | 70/45                    | 90               | 14  | 7.45| 1.4             | 37                       | 24                       | 13                       | Septic shock | Survival          |
| 12  | 69  | 60/35                    | 48               | 3   | 7.4| 10              | 38                       | 45                       | -7                       | Neurogenic shock (medullary death) | C1–C2 section |

Abbreviations – GCS: Glasgow Coma Scale; PE: pulmonary embolism; STEMI: ST-elevation myocardial infarction. \(PaCO_2\): arterial \(CO_2\) partial pressure. \(EtCO_2\): End-tidal \(CO_2\).
In conclusion, the analysis of this short series of patients aimed to focus on the potential clinical value for physicians facing life-threatening clinical situations to measure P(A–Et)CO₂ in cases of shock, especially if the diagnosis is initially challenging. Our data are far too limited in order to draw any clinical conclusion. Our results could however question if an initial P(A–Et)CO₂ difference remaining under the normal range (less than 5 mmHg) could guide the clinician to another diagnosis than PE in case of a clinical shock of unclear aetiology at the time of admission.

Conflicts of interest and source of funding

None.

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