Respiratory Medicine Case Reports 16 (2015) 106–108

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr

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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ARTICLE INFO

Article history:
Received 26 July 2015
Received in revised form
5 September 2015
Accepted 8 September 2015

Keywords:
CO_2
Shock
Pulmonary embolism

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 CO_2 measurement and alveolar-arterial CO_2 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 CO_2 ($EtCO_2$) and arterial CO_2 partial pressure ($PaCO_2$) measurements.
Results: In cases with pulmonary embolism-related shock (n = 3), the gradient between $PaCO_2$ and $EtCO_2$ was increased (37 vs 0.2 mmHg). There was a similar trend for a higher $PaCO_2$ value (60 vs 32.2 mmHg) and a lower $EtCO_2$ value (23 vs 32 mmHg).
Conclusion: An initial CO_2 measurement might be an easily available tool for the early diagnostic work-up of clinical shock.

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 CO_2 measurement can show a correlation between decreased CO_2 transport in the blood and hemodynamic compromise. The measurement of either expired CO_2 or arterial CO_2, 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 CO_2 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 CO_2 elimination at the mouth and an increased difference between arterial and alveolar CO_2 [2,3]. We aimed to assess if expired CO_2 and/or alveolar-arterial CO_2 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 CO_2 ($EtCO_2$), from a commercial time-based capnography, and arterial CO_2 partial pressure ($PaCO_2$) 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

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http://dx.doi.org/10.1016/j.rmcr.2015.09.004

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. 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). Jugular 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 (the $P(a-Et)CO_2$ gradient) was 37 mmHg in the three patients with a massive PE and 0.2 mmHg in the nine patients with another diagnosis. The isolated $EtCO_2$ measurement showed a similar trend to lower values in patients with PE (23 vs 32 mmHg) and the isolated arterial $PaCO_2$ showed a similar trend to higher values (60 vs 32 mmHg). Under the reserve of this small sample, an early and easy initial CO_2 measurement might help the physician in the diagnostic work-up of a severe and unclear shock.

The calculation of alveolar dead space volume using capnography was previously shown to be of interest in the diagnosis of PE when combined with pretest clinical probability and D-dimer results [4,5]. Kline et al. also demonstrated a correlation between the increase in alveolar dead space volume and the severity of pulmonary vascular occlusion, by means of the vascular defect percentage on perfusion lung scans [6]. However, this literature refers to patients with adequate blood pressure [4–6]. Capnography was also shown to be a reliable noninvasive alternative in monitoring the efficacy of thrombolysis in massive PE when the diagnosis has been established [7,8]. Our results extend the application range of the expired CO_2 measurement by suggesting its potential in the assessment of clinical shock of unclear aetiology.

Clinical signs guide the physician in the initial management of shock. In the patient data analysed, obstructive shocks due to massive PE were significantly linked with the presence of jugular vein distension. This finding was potentially interesting; however, large prospective data already proved the lack of specificity of jugular vein assessment in PE [9]. Thus, the CO_2 measurement was the only paraclinical parameter of interest identified in these cases of shock.

There is major potential interest for capnography measurements in patients with shock when the early diagnosis is difficult to establish. Even if this difficulty might subjectively depend on the physician's experience, we consider our short series as representative of the most challenging urgent clinical situations physicians must face, in terms of rare and complex diagnoses (like aortic root dissection with tamponade) or clinical severity (like cardiac arrest, severe acidosis with pH < 7.0, and requirement of mechanical ventilation). Any easily obtained $EtCO_2$ or $P(A-Et)CO_2$ value might be useful in those particularly stressful situations, as it could refine the differential diagnosis and point towards the next diagnostic tests to realize.

Our case series has major limitations. First, although it was of a prospective nature, the results were retrospectively analysed. Inclusions were non-consecutive, implying a selection bias. Second, our small sample of patients prevents generalization of the results. Third, we have interpretation bias, as our 2 groups are not representative of the same population, as proven by the difference in mortality rates or pH values.

Table 1
Patients' characteristics, initial vital signs, established diagnosis, and outcome.

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

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.

Table 2
Patients' CO_2-related measurements.

	Patients with pulmonary embolism (n = 3)	Patients without pulmonary embolism (n = 9)
Patients' characteristics		
Mechanical ventilation required	2	3
Jugular vein distension	3	1
pH (mean)	6.99 ± 0.41	7.34 ± 0.12
survival	3	4
CO_2 analysis (mmHg, mean ± SD)		
$PaCO_2$	60 ± 26	32 ± 12
$EtCO_2$	23 ± 13	32 ± 11
(Pa-Et)CO_2 gradient	37 ± 23	0 ± 7

Abbreviations-$PaCO_2$: arterial partial CO_2 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)CO_2 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)CO_2 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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