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0032. Relationship between microcirculatory alterations and venous-to-arterial carbon dioxide differences in patients with septic shock

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Introduction

Increased venous to arterial carbon dioxide difference ($Pv\text{-}aCO_2$) have been attributed to low cardiac output states. However, mechanisms conducting to $Pv\text{-}aCO_2$ increases during normal or even high cardiac output conditions as in septic shock are not fully understood. We hypothesized that $Pv\text{-}aCO_2$ could reflect the adequacy of microvascular perfusion during resuscitated septic shock

Objectives

To test the hypothesis that $Pv\text{-}aCO_2$ could reflect the microvascular blood flow during the early phases of resuscitation in septic shock

Methods

We included 80 patients with a first episode of septic shock admitted to a mixed ICU in a University Hospital over a 12-month period. Time 0 (T0) was set at ICU admission when a pulmonary artery catheter was inserted. Arterial and venous gases analyses were performed at T0 and 6 hours after (T6). We defined $Pv\text{-}aCO_2$ as the difference between the mixed venous and arterial CO_2 partial pressures. A Sidestream Dark-Field (SDF) imaging device (Microvision Medical, Amsterdam, the Netherlands) was used to evaluate the sublingual microcirculation both at T0 and T6. At each assessment, 5 sequences of 20 seconds each were recorded and stored under a random number. An investigator blinded to the sequence order and patient's clinical course, analyzed the sequences semi-quantitatively. The vessels were separated into large and small using a cut-off value of 20 μm in diameter. We evaluated the relation between the percentage of small vessels perfused and the $Pv\text{-}aCO_2$ using linear and non-linear regressions and Spearman Rho test. A $p< 0.05$ was considered as significant.

Results

We found significant but very weak relationships between general hemodynamics or oxygen derived parameters with $Pv\text{-}CO_2$.$Pv\text{-}aCO_2$ was inversely related to the percentage of small vessels perfused both at T0 and T6 (T0: R^2:0.515, $p< 0.001$; T6: R^2:0.453, $p< 0.001$).

Conclusions

Microvascular blood flow is a key determinant of $Pv\text{-}aCO_2$ during normodynamic septic shock. $Pv\text{-}aCO_2$ could track microvascular alterations during early phases of septic shock.

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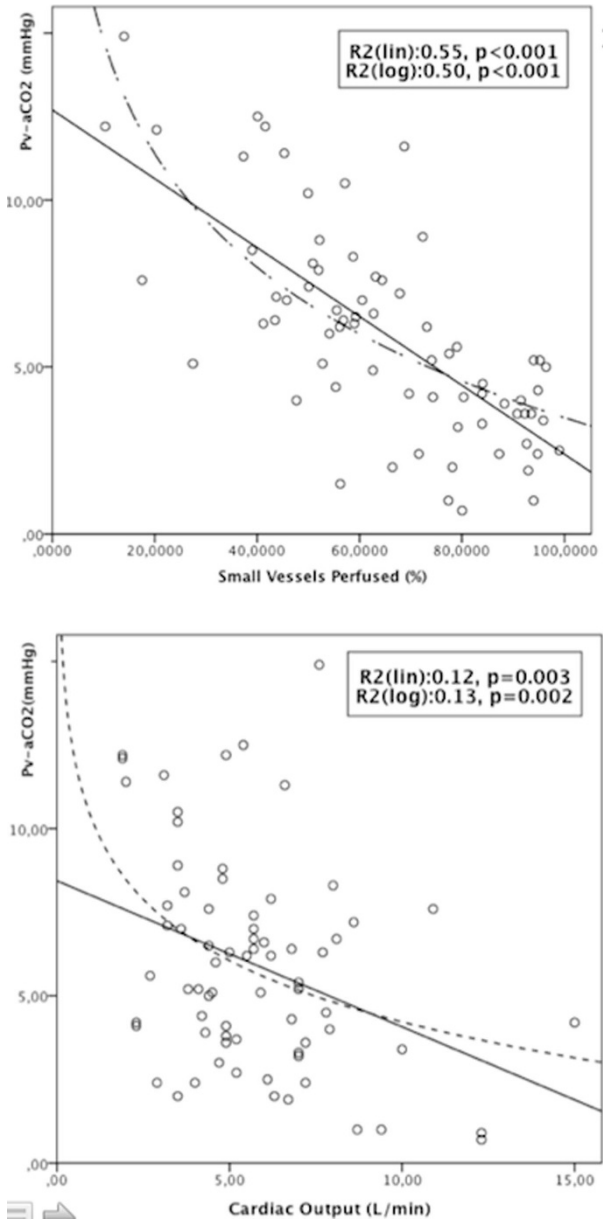

Fig. 1 Pv-aCO2 vs. SVP and CO at T0

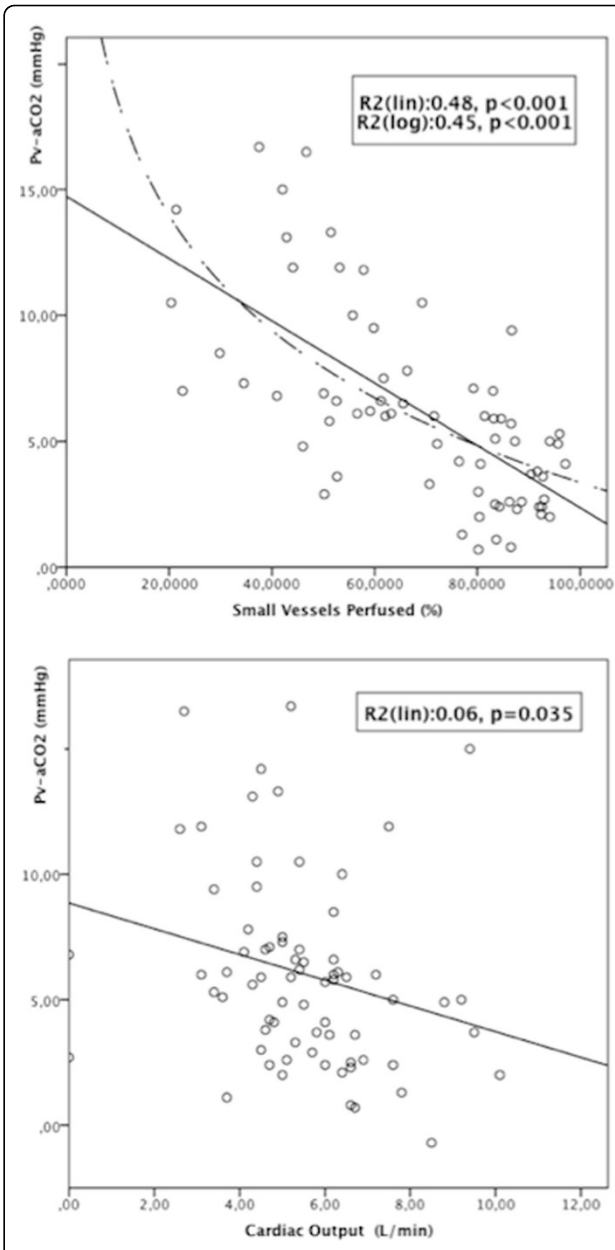

Fig. 2 Pv-aCO2 vs. SVP and CO at T6

Table 1 General hemodynamics and Oxygen-derived parameters

	T0	T6
SvO2, (%)	68.8 (61.75-75.0)	69.7 (64.4-75.9)
Cardiac Index, (L/min/m2)	3.3 (2.4-4.0)	3.2 (2.7-3.8)
iDO2, (ml/min/m2)	389.0 (293.4-500.3)	399.0(322.3-468.2)
iVO2, (ml/min/m2)	116.8 (87.3-150.4)	182.8 (106.3-240.4)
Pv-aCO2, (mmHg)	5.2 (3.6-7.2)	5.8 (3.5-7.7)

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