The complex kinetics of blood endocan during the time course of sepsis and acute respiratory distress syndrome

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The complex kinetics of blood endocan during the time course of sepsis and acute respiratory distress syndrome

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Dear Editor,

Several works have explored the blood concentrations of endocan in sepsis and acute respiratory distress syndrome (ARDS). However, data from the literature seem apparently conflicting, with high endocan levels being associated with either good or poor prognosis according to the different studies. Indeed, endocan levels on intensive care unit (ICU) admission correlate with the severity of sepsis [1]. In septic shock patients without ARDS at admission, high levels of endocan are found predominantly in patients who do not develop ARDS [2]. Ioakeimidou et al. reported that progression to ARDS in septic patients was associated with the increase of blood endocan during follow-up [3]. Furthermore, Orbegozo et al. and Tsangaris et al. reported that higher endocan levels measured at the clinical onset of ARDS were associated with poor respiratory outcomes [4, 5]. The above-stated observations suggest that endocan's predictive values may sound more complex than a simple association between high plasmatic levels and the development of poor outcomes.

To better understand the evolution of endocan over the time course of sepsis and ARDS, we conducted a post hoc analysis of the kinetics of blood endocan over 72 h, based on the data from a previously published cohort of 72 septic patients without ARDS on baseline [2]. Among the 72 patients enrolled in this cohort, 11 subjects developed an ARDS at 72 h (8 mild, 3 moderate, 1 severe).

In patients without ARDS, endocan continually decreased during the 72-h time course following enrollment, with median [IQR] values falling from 9.2 [5.6–14.8] ng/mL on enrolment to 3.9 [2.6–7.7] ng/mL 72 h later (Fig. 1a). In patients progressing to mild ARDS, endocan moderately increased from 2.5 [1.3–3.4] ng/mL on enrolment to 4.1 [2.3–7.3] ng/mL at 72 h (Fig. 1b). We observed a higher increase of blood endocan in patients progressing to moderate and severe ARDS, with median [IQR] values rising from 4.7 [2.5–5.4] ng/mL on enrollment to 11 [9.5–12.6] ng/mL at 72 h (Fig. 1c).

This study highlights the kinetics of endocan in severe sepsis and ARDS, thus helping to understand the apparently conflicting results observed in the literature. However, the interpretability of this work remains limited given the small effectives in each subgroup of ARDS, yet it may be used jointly with other data from the literature to elaborate a model of endocan's kinetics during severe sepsis and ARDS. Therefore, further explorations are required to comfort these results.

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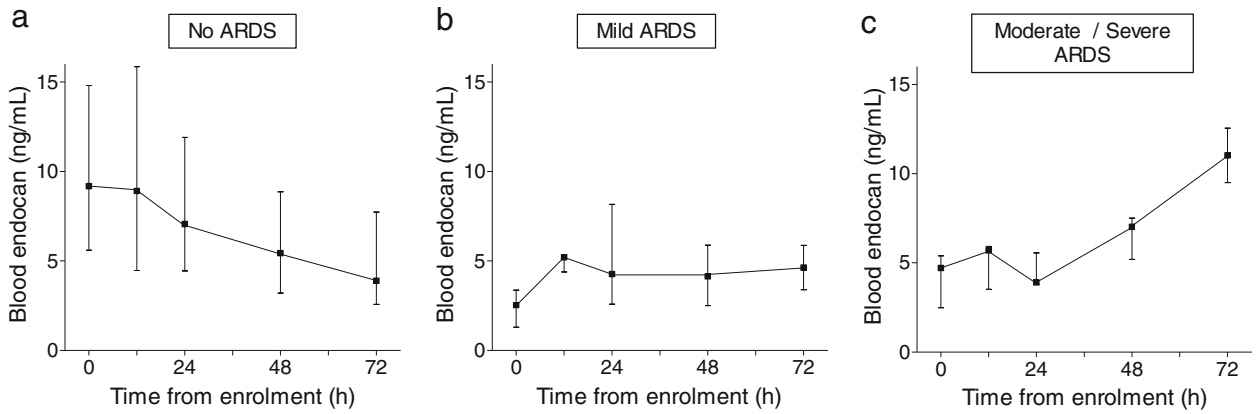

Fig. 1 Kinetics of endocan in severe sepsis according to the presence and severity of ARDS at 72 h. Diagnosis and severity of ARDS was assessed in accordance with the Berlin definition on baseline, and at 12 h, 24 h, 48 h, and 72 h following enrollment. For each patient, the severity of ARDS corresponds to the worst level of severity reached during follow-up. Median [IQR] values of blood endocan are represented at each time point of follow-up in patients with no ARDS (**a**), mild ARDS (**b**), and moderate to severe ARDS (**c**) at 72 h. Variation of blood endocan over 72 h was significantly different between these three subgroups (Kruskal-Wallis test, $p < 10^{-2}$

Abbreviations
ARDS: Acute respiratory distress syndrome; ICU: Intensive care unit

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Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions
AG and EP designed the whole study, conducted the data analyses, and drafted the manuscript. ND, LP, and SD performed the biological measurements. DM supervised the whole project. All authors read and approved the final manuscript.

Ethics approval and consent to participate
This study has been approved by the ethics committee of Lille University Hospital (approval number CP03/07). All the participants to this study gave their informed consent prior to enrollment.

Consent for publication
Not applicable.

Competing interests
The Endomark H1 and DIYEK C1 ELISA kits used in this study were provided by Lunginnov. P. L. is the cofounder of Lunginnov. N. D. and L. P. are staff members of Lunginnov. The other authors declare that they have no conflicts of interest.

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References
1. Scherpereel A, Depontieu F, Grigoriu B, Cavestri B, Tsicopoulos A, Gentina T, et al. Endocan, a new endothelial marker in human sepsis. Crit Care Med. 2006;34:532–7.
2. Gaudet A, Parmentier E, Dubucquoi S, Poissy J, Duburcq T, Lassalle P, et al. Low endocan levels are predictive of acute respiratory distress syndrome in severe sepsis and septic shock. J Crit Care. 2018;47:121–6.
3. Ioakeimidou A, Pagalou E, Kontogiorgi M, Antoniadou E, Kaziani K, Psaroulis K, et al. Increase of circulating endocan over sepsis follow-up is associated with progression into organ dysfunction. Eur J Clin Microbiol Infect Dis. 2017;36(10):1749–56.
4. Orbegozo D, Rahmania L, Irazabal M, Mendoza M, Annoni F, De Backer D, et al. Endocan as an early biomarker of severity in patients with acute respiratory distress syndrome. Ann Intensive Care. 2017;7 [cited 2017 Dec 10]. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5589715/.
5. Tsangaris I, Tsantes A, Vrigkou E, Kopterides P, Pelekanou A, Zerva K, et al. Angiopoietin-2 levels as predictors of outcome in mechanically ventilated patients with acute respiratory distress syndrome. Dis Markers. 2017;2017:6758721.