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Evolution of cytokines/chemokines in cases with severe nosocomial pneumonia and distinct etiologies in mechanical ventilated patients

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Abstract

Aim: To compare the systemic cytokines/chemokines levels over time during the evolution of mechanical ventilated (MV) children hospitalized with nosocomial pneumonia (NP) with and without Pseudomonae Aerogenosa (PAi) infection.

Methods: MV children <5 y.o. hospitalized with NP were prospectively investigated in Vinnitsa regional clinical hospital. Clinical data and samples were collected to investigate 20 etiological agents and serum cytokines/chemokines levels on admission and 2 to 4 weeks later. Cases with PAi received this diagnosis irrespective of having other etiologies.

Results: 86 patients were enrolled, however 16 patients were excluded from the study. Study group comprised of 72 cases with established etiology. The median age and sampling interval was 21 (9-27) months and 19 (16-21) days, respectively. Etiology was viral-bacterial (22.2%), and bacterial (77.8%). PAi was found in 23 (26.7%) patients. Median interleukin-6 (IL-6; 9.4 [4.7-24.4] vs 18.1 [17.2-20.4]; P = .03), IL-10 (3.2 [3.1-4.5] vs 12.1 [11.8-17.4]; P = .04), and CCL2 (21.4 [12.4-24.3] vs 92.8 [68.4-118.0]; P < .001) were significantly higher in convalescent samples, whereas median CXCL10 (80.4 [35.4-172.2] vs 15.2 [0-117.4]; P < .001) was lower. Acute vs convalescent levels evolution of IL-6, IL-10, and CXCL10 did not differ among patients with or without PA. However, CCL2 decreased (24.2 [12.2-44.4] vs 20.4 [20.2-22.4]; P = .1) in patients with PAi and increased (10.1 [4.4-23.4] vs 20.8 [19.4-22.2]; P = .001) in patients without it.

Conclusion: The marked increase of CCL2 serum levels during the acute phase makes it a potential biomarker of PAi among MV children with NP.

Footnotes

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Flow-controlled ventilation (FCV) improves regional ventilation in obese patien with diabetis type 1 and 2

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Abstract

Background: In obese patients, high closing capacity and low functional residual capacity increase the risk for expiratory alveolar collapse. We hypothesized that lung aeration and respiratory mechanics improve in obese patients during FCV.

Methods: We compared FCV and volume-controlled (VCV) ventilation in 18 obese patients in a randomized crossover setting. Starting with baseline measurements, ventilation settings were kept identical except for the ventilation mode related differences (VCV: inspiration to expiration ratio 1:2 with passive expiration, FCV: inspiration to expiration ratio 1:1 with active, linearized expiration). Primary endpoint of the study was the change of end-expiratory lung volume compared to baseline ventilation. Secondary endpoints were the change of mean lung volume, respiratory mechanics and hemodynamic variables.

Results: The loss of end-expiratory lung volume and mean lung volume compared to baseline was lower during FCV compared to VCV (end-expiratory lung volume: FCV, - 144 ± 177 ml; VCV, - 323 ± 232 ml; $p < 0.001$, mean lung volume: FCV, - 103.4± 188.4 ml; VCV, - 305.8 ± 244.1 ml; $p < 0.001$) and at comparable plateau pressure (baseline, 18.3 ± 3.8; VCV, 21.4 ± 3.3; FCV, 21.1 ± 3.6 cmH2O; $p = 0.432$), mean tracheal pressure was higher (baseline, 12.9 ± 1.2; VCV, 12.8 ± 1.23; FCV, 15.1 ± 2.1 cmH2O; $p < 0.001$). All other respiratory and hemodynamic variables were comparable between the ventilation modes.

Conclusions: This study demonstrates that, compared to VCV, FCV improves regional ventilation distribution of the lung at comparable PEEP, tidal volume, Plat and ventilation frequency.