Differences in cytokine production after stimulation of whole blood of septic patients with heat-killed Pseudomonas aeruginosa and Staphylococcus aureus

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BACKGROUND

Health-care associated infections represent a serious medical problem. Infectious complications in hospitalized patients are associated with high morbidity and mortality, prolonged hospital stay, increased antibiotic resistance and economic burden. These infections mainly affect critically ill patients whose immune system is often compromised by exaggerated anti-inflammatory response and inability to respond appropriately to infectious agents. The aim of our experimental study was to determine the production of selected pro-inflammatory and anti-inflammatory cytokines and chemokines after ex vivo stimulation with heat-killed Pseudomonas aeruginosa or Staphylococcus aureus.

METHODS

- Adult patients admitted to infectious diseases department with bacterial sepsis
- Ex vivo stimulation of whole blood with heat-killed bacteria
- Experiments proceeded with Pseudomonas aeruginosa (Xen41) and Staphylococcus aureus (Xen81)
- Plasma cytokine and chemokine levels quantified by flow cytometry using multiplex bead-based immunoassay
- Differences between groups of patients and healthy volunteers evaluated by Mann-Whitney U test

RESULTS

Bacterial sepsis group n = 10

<table>
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<th>Phenotype</th>
<th>Number</th>
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<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>51.5 ± 17.8</td>
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<tr>
<td>Sex, males (%)</td>
<td>5 (50%)</td>
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<tr>
<td>Hospital days (mean ± SD)</td>
<td>13.9 ± 11.4</td>
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<tr>
<td>Sepsis status</td>
<td>5 (50%)</td>
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<tr>
<td>Etiology</td>
<td>1</td>
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E.coli 5

Peptostreptococcus 2

Microbiology results

| E.coli 5 |

Phagocytic activity of blood leukocytes

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<th>Phagocytic activity of blood leukocytes</th>
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Conclusion

In our study, we observed more pronounced immunogenic potential of heat-killed Pseudomonas aeruginosa in comparison to Staphylococcus Aureus. Significant elevation of anti-inflammatory cytokines after stimulation with heat-killed P. aeruginosa suggests that P. aeruginosa may actively create an immunosuppressive niche leading to subsequent secondary infection.

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REFERENCES


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