ABSTRACT

The purpose of this study is intended to explain a shock septic mechanism in IC and NIC. This study contains two phases of observational analysis. First phase applies cross sectional design to differentiate immune response for IC and NIC in non-sepsis condition. Second phase applies Cohort design to differentiate immune response for IC and NIC in sepsis and septic shock condition. First and second phases of this study use a separate sample. The variables of immune response are determined by analyzing IgG, C3, C4, IL-10, IFN-γ, TNF-α and IL-1β. A multivariate analysis (Manova) is applied in this study for statistical analysis.

Result of this study, there was a significant difference ($p<0.05$) in immune response toward an IC and NIC non-sepsis condition marked by increasing on IL-10, TNF-α, IgG and decreasing on C3 variable. However, there were no significant ($p>0.05$) different on the result for IC and NIC in sepsis condition. No significant different also found for IC and NIC in septic shock condition. Then it is concluded that IC and NIC are not different immune response. Thus, IC and NIC were treated as a one group of experiment. The result for IC-NIC in septic shock and IC-NIC in non-sepstic shock showed a significant difference ($p<0.05$).

Furthermore, due to immune response differences above, the study was continued with discriminant analysis (discriminator). For IC-NIC septic shock and IC-NIC sepsis, there were four variables as discriminators, IL-10, IL-1β, IgG and C3.

To explain the immunopathobiogenesis, a narration of conceptual framework has been done using discriminant pattern. Based on such method, it can be explained that the increase of IL1β contribution was caused by immunogene stimulation to macrophage (APC). Furthermore, APC stimulated Th2, inducing the increase of IL-10 contribution that accelerating B lymphocyte maturity to become IgG-producing plasma cells. Together with toxin, IgG formed a complex binding that precipitated on the wall of endothelial cells in blood vessels, and activated C3 complement, leading to endothelial destruction through a process called antibody dependent cellular cytotoxicity (ADCC). The increase of IL1β stimulated endothelial cells to induce ICAM-1. As an adhesive substance, ICAM-1 would be bound with neutrophyl, leading to endothelial destruction in blood vessels through DTH process. Both processes caused leakage in the wall of blood vessel, producing the occurrence of septic shock.

In conclusion, the immunopathobiogenesis of septic shock can be caused by the mechanisms of ADCC and DTH.

Keywords: Immunocompromise (IC), Non-Immunocompromise (NIC), Sepsis, Septic Shock, ADCC, DTH