Editorial
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Does G-CSFR Blocker Reduces Hematopoietic Dysfunction Among Trauma Associated Hemorrhagic Shock?

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Editorial

Hematopoietic Dysfunction (HD) has been proved in trauma associated hemorrhagic shock (T/HS). Prolonged mobilization of Hematopoietic Progenitor Cells (HPCs) are related with the HD and poor outcome following T/HS patients. Excessive Granulocyte Colony Stimulating Factor (G-CSF) related with this response [1-3].

G-CSF is a major regulator of granulocyte production from progenitor cells. Its receptor (G-CSFR), found on precursor cells in the bone marrow and proliferate and differentiation in to granulocyte. G-CSF induces survival, proliferation, and differentiation of granulocyte. And act as neutrophil precursors and mature neutrophils. G-CSF regulates by Janus Kinase (JAK)/ Signal Transducer and Activator of Transcription (STAT) and Ras/ Mitogen-Activated Protein Kinase (MAPK) and Phosphatidylinositol 3-Kinase (PI3K)/ protein kinase B (Akt) signal transduction pathway. G-CSF receptor also linked with the inflammation and in many pathologies and it's also effect on the mature myeloid population [4]. Previous studies reported that G-CSF induces mobilization of HPCs and inflammatory cytokine directly connected with the HD and worsen outcome in T/HS [2]. G-CSF mediated by the inflammatory cytokines such as IL-6 and TNFα. G-CSF also locally produced by liver and lung of T/HS animal's model [5]. Excessive release of neutrophils directly linked with tissue damage [6]. Production of G-CSF mRNA by bronchoepithelial cells is dramatically increased in a rat model of HS that also demonstrated lung injury. G-CSF levels may also contribute to polymorphonuclear neutrophils recruitment and activation and resultant lung injury in HS [6]. Tanaka et al. reported that elevated circulating G-CSF in trauma, burn and septic patients. G-CSF released from endothelium, macrophage and other immune cells is unclear [7]. Excessive G-CSF also associated with the severity of disease of Rheumatoid Arthritis (RA) patients [8]. Neutralizing anti-G-CSFR mAb can be prevented and rapidly reverse arthritic pain and disease [8].

The role of G-CSFR blocker is still remaining in T/HS. Scalzo-Inguanti K et al. showed that treatment with anti-G-CSFR antibody (CSL324) controlling G-CSF mediated neutrophilia using nonhuman primates [9]. Therefore, to explored the role of G-CSFR blocker following T/HS. G-CSF receptor blocker that would be neutralizing G-CSFR which will be help to reduce the mobilization of HPCs results improved the HD and outcome following T/HS.

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