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Do Nucleic Acid–Binding Polymers (Nabps) May Be Reduce Proinflammatory Nucleic Acids Among Trauma-Associated Hemorrhagic Shock?

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Trauma-associated hemorrhagic shock (THS) is the major leading cause of death worldwide with 1.9 million deaths per year. THS accounts for 30–40% of deaths of the patients brought to the emergency room do not survive the first 24 hours [1]. Excessive releases of inflammatory cytokines are linked with dysregulation of immune system results infection, sepsis, mutiorgan failure (MOF) and death [1, 2]. In addition, elevated granulocyte colony stimulating factor, catecholamine, mobilization of hematopoietic progenitor cells from bone marrow in to peripheral blood cells and decreased expression of erythropoietin receptor are also associated with this response. Advanced care and treatment of the traumatic injured and hemorrhage patients have undergone much progress in the last decades. Resuscitation fluid, blood and its components are used for the control of haemorrhage [1, 2]. Previous studies showed fluid resuscitation also associated with the excessive amounts of intracellular organelles are released and function as endogenous damage-associated molecular patterns (DAMPs), released excessive inflammatory cytokines leads to multiple organ dysfunctions. As a results effective treatment for trauma associated hemorrhagic shocks are remains. Cell-free (cf) DNA may be released and function as DAMPs to cause organ damage in THS. Previous studies has been reported that cfDNA capability to use as a noninvasive biomarker to determine pathologic conditions in various diseases, in sepsis, traumatic injury, diabetes, autoimmune rheumatic diseases, non-infectious inflammations and tumors, as well as brain diseases [3, 4]. Increased circulating levels of cfDNA levels in post trauma injury have been associated with poor clinical outcomes, which involve the development of acute respiratory distress syndrome (ARDS), infection, sepsis, multiple organ failure and death [14]. Recently study demonstrated that, HS, serum level of cfDNA was significantly increased in the vehicle-treated mice by 87% compared to sham [4-6]. Recently studies reported that mitochondrial DNA (mt DNA) are released by DAMPs in trauma, which is similarity with bacterial mtDNA contain highly stimulatory unmethylated CpG DNA motifs that signal via toll-like receptor-9 that results excessive release of cytokines leads to inflammation [4, 6, 7].

Nucleic acid-binding polymers (NABPs) polymers act as anti-inflammatory agent and neutralize proinflammatory release by nucleic acid. In previous studies has been reported that nucleic acid-binding polymers (NABPs) such as third-generation polyamidoamine dendrimer (PAMAM-G3) are capable to neutralize cell-free DNAs and RNAs to activate that signal through toll-like receptors (e.g., TLR3, TLR7, TLR8 and TLR9). Activated TLR linked to inflammation leads to infection, sepsis, MOF (Figure 1) and death [7-8].

PAMAM-G3 is a common NABP, act as inhibitor of TLRs activation and reduced inflammatory nucleic acid. It also used for treatment of liver dysfunction, lupus, cancer metastasis, and influenza infection. PAMAM-G3 also reported as a toxic effect in vivo. Lee et al showed that the effect of PAMAM-G3 on cecal ligation and puncture (CLP)–induced severe sepsis model and the CpG-induced fatal SIRS model [7-9]. Based on the previous literature NABPs may be reduced inflammatory nucleic acid via inhibitor of TLRs activation. Need to be attention.

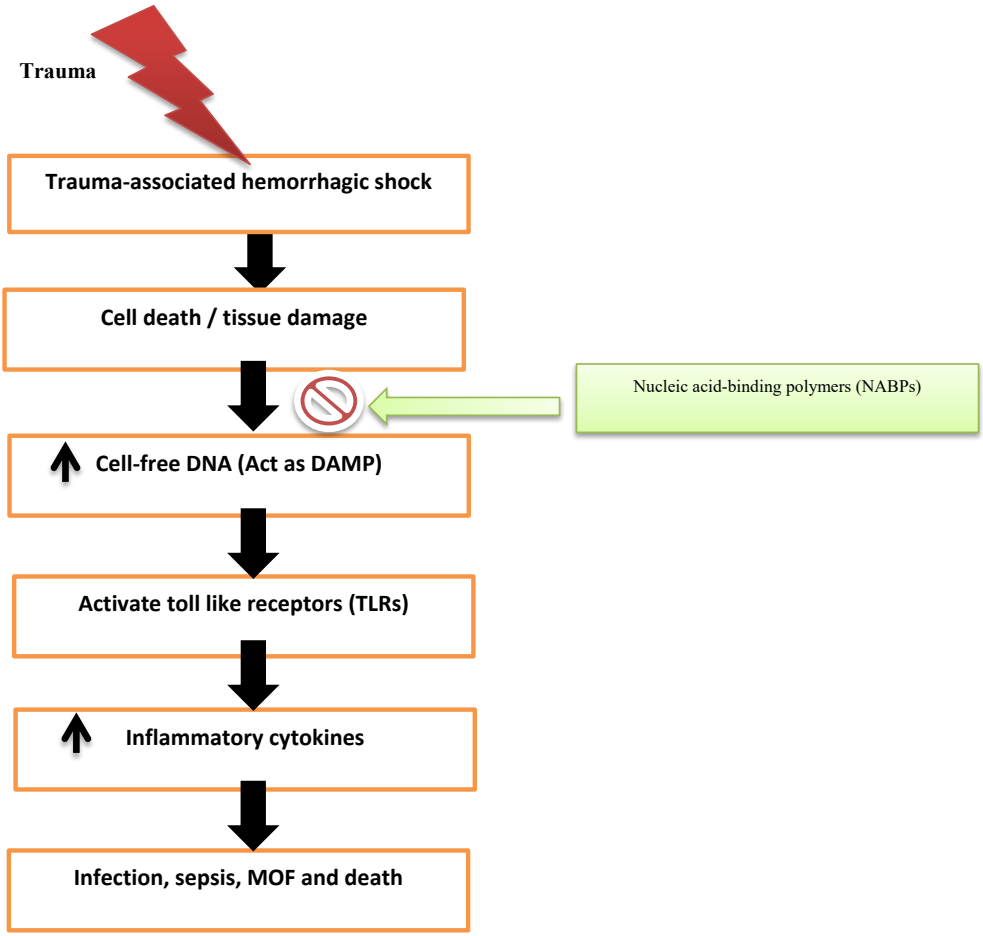

Figure 1: Mechanism of NABPs act as anti-inflammatory.

References

1. Kumar M, Bhoi S (2016) Impaired hematopoietic progenitor cells in trauma hemorrhagic shock. J Clin Orthop Trauma 7(4):282-285.
2. Huang Q, Gao S, Yao Y, Wang Y, Li J, et al. (2022) Innate immunity and immunotherapy for hemorrhagic shock. Front Immunol 3:918380.
3. Aswani A, Manson J, Itagaki K, Chiazza F, Collino M, et al. (2018) Scavenging Circulating Mitochondrial DNA as a Potential Therapeutic Option for Multiple Organ Dysfunction in Trauma Hemorrhage. Front Immunol 9:891.
4. Dawulieti J, Sun M, Zhao Y, Shao D, Yan H, et al. (2020) Treatment of severe sepsis with nanoparticulate cell-free DNA scavengers. Sci Adv 6(22):eaay7148.
5. Lee J, Sohn JW, Zhang Y, Leong KW, Pisetsky D, et al. (2011) Nucleic acid-binding polymers as anti-inflammatory agents. Proc Natl Acad Sci USA 108(34):14055-60.
6. Lee J, Jackman JG, Kwun J, Manook M, Moreno A, et al. (2017) Nucleic acid scavenging microfiber mesh inhibits trauma-induced inflammation and thrombosis. Biomaterials 120:94-102.
7. Chen HH, Yuan H, Cho H, Feng Y, Ngoy S, et al. (2017) Theranostic Nucleic Acid Binding Nanoprobe Exerts Anti-inflammatory and Cytoprotective Effects in Ischemic Injury. Theranostics 7(4):814-825.
8. Aswani A, Manson J, Itagaki K, Chiazza F, Collino M, et al. (2018) Scavenging Circulating Mitochondrial DNA as a Potential Therapeutic Option for Multiple Organ Dysfunction in Trauma Hemorrhage. Front Immunol 9:891.
9. Dawulieti J, Sun M, Zhao Y, Shao D, Yan H, et al. (2020) Treatment of severe sepsis with nanoparticulate cell-free DNA scavengers. Sci Adv 6(22):eaay7148.