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Editorial

Highlights About Recent Sepsis Related Studies

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Recently sepsis and septic shock are (re-)defined as the new definitions, which emphasis that sepsis as life-threatening organ dysfunction caused by a dysregulated host response to (concomitant) infection(s) [1,2]. Sepsis in human was described in various forms in about 1000 BC and for the first time defined as an infection in the blood or tissues along with fever by the Persian Philosopher Ibn Sina Zakaria Razi, who was born in the Rey city, Iran (also known as Avicenna) [3,4]. Daily, terminal patients are predominantly dying from the different causes i.e. thrombosis, bleedings disorders, cardiovascular failure, respiratory, metabolic failure with and/or without septic shocks. Sepsis incidence and mortality varied substantially across regions, with the highest burden in sub-Saharan Africa, Oceania, south Asia, east Asia, and southeast Asia [4,5]. There is scant studies published investigating sepsis and septic shock in a human model system, *in vivo*. Moreover, there are so much aspects unknown concerning either the mechanism of sepsis or inducing system of uncontrolled shot down of different organs after sever septic shocks. After sepsis was recently re-defined as the new proposal concept one could conclude that sepsis is a serious life-threatening process which induces organ dysfunction and random dysregulation of host response to infecting antigens [2-5].

Recently, Rudd KE *et al.* 2020 proposed that infection-related sepsis is reflected a main cause of mortal shot down of organs [4]. Nonetheless which main factor manages whole SRPs toward an increase in mortality and morbidity rate in hospitals is not elucidated completely. Meticulous assessments are significant to clarify and distinguish different health procedure mediations, allocation of means, and might different independent clinical management supervisions are essential in all Hospitals, world widely.

After so many sophisticated technologies still one of five death causes is Septic related processes (SRP), which could be divided in three phases I. initiation by infection with certain microorganisms i.e. different bacteria, viruses, and their toxins. Subsequently, second phase so- called propagation phase, which released toxins transmits to the multiple organs via either systemic blood circulation or lymphatic systems. Finally, termination phase, which results in life- threating shot down of different random vital organs. Moreover, initiation phase could be divided in four types or pathways, which might contaminate the host randomly, and the certain transfected microorganisms start to attack host's organs to adhere, penetrate, and establish certain signal transduction machinery for proliferation and propagation in host cells and tissues.

In theory, everyone could be being contaminated every day via 4main ways namely: 1. After medical blood transfusion and/or transplantations with infected cells and organs 2. After closed social contacts i.e. kissing, handshaking, swimming pools, sport stadium etc., 3. via air and aerosol transfections, and 4. after sexual transfections.

Though, why someone get sepsis, while another in the same room get not is not elucidated completely. Three underlying factors, which could induce sepsis regionally are 1) infection- related i.e. diarrheal disease, 2) injuries- related i.e. traffic accidents, and 3) Non-communicable diseases i.e. maternal disorders [4,5].

Hypothetically, patients and people with fragile immune systems are susceptible to get earlier septic shock than healthy individuals. Moreover, the ICU's patients, who are undergoing surgical operations undergo more risks than patients that not needed any further treatments. One might speculate that surgical ICU's patients are always at risk to get sepsis because they essentially need more intensive care from other donors i.e. blood transfusion and organs transplantation and might need different biological products, however.

Kamimura D, Bevan 2008 proposed that the XBP-1 gene splicing plays an essential role in activating the unfolded protein response (UPR) in the endoplasmic reticulum (ER) after certain oxidative stress. Transcribed XBP-1 m RNA is converted to its active form by unconventional cytoplasmic splicing mediated by inositol-requiring enzyme-1 (IRE-1) upon ER stress. They reported that acute infection could activate the IRE-1/XBP-1 pathway in effector CD8+ T cells [6]. Though, in one hand, the big gap between bench and bed research and developments, and in the other hand, complexity of infections clinically, might resulted in a miscommunication (or no communications) between Basic researchers and Clinicians.

Beside rapid diagnostic tools are obviously not specific and sensitive enough to warn (para-) Medics timely about the ongoing fatal Sepsis related processes (SRPs) propagation toward termination phase in a certain patient, extraordinarily. One might speculate why there is no animal and/or human model system available either to do research or monitor early indication of the SRPs developments, or to improve appropriate medicines, immediately.

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