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Editorial

Death Triangle Machinery, Assumptions About Possible Correlation Between Peritonitis- Platelets- Microorganism and Death Causes

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Death triangle machinery (DTM) activation cause significant increase in mortality and morbidity risks, however [1-4]. The mechanism of action is rather invented than facts-based theories, and real standard means to prevent DTM do not approved yet. There are so much key factors missing at Nano- and Microenvironment signaling that a real mechanism, which might work for everybody do not exist as well.

One of major key player in increasing In-Hospital Death Rate (IHDR) and overall-death rate (ODR) is catastrophically mixed-ups of the (un-)known microorganisms' involved in the septic shock, and cancerogenous irregular process. For instance, secondary peritonitis accounts for 1% of urgent or emergent hospital admissions and is the second leading cause of sepsis in patients in intensive care units globally [3]. Different data is showing that overall mortality is increasing to 35-40% in the different patients, who develop severe sepsis.

Despite the availability and use of advanced clinical imaging and laboratory tests, the rapid diagnosis and early management of for example peritonitis remains a challenge for physicians in emergency medicine, surgery, and critical cares of the (para-)Medici.

Ross *et al.* 2018 [3] recently postulated that bacteria spilled into the peritoneum are recognized directly by pattern recognition receptors of the innate immune system, and indirectly via molecules released from

injured mesothelial cells. The initial stage of the response depends on an influx of phagocytes (macrophages), and the production of pro-inflammatory cytokines (Cellular Immune responses) including tumor necrosis factor α, interleukin 1, and interleukin 6 [3]. However, neutrophils collaborate strictly with platelets, their expression depends on each other's signaling at cell-cell and protein-protein level, once main cause of sepsis is bacterial contamination. Moreover, bacterial destruction releases lipopolysaccharide and other cellular components that further stimulate the host pro-inflammatory response so-called Cascaded catastrophic effects. Besides, the degree of perforation or extent of contamination is unclear in secondary peritonitis, where diagnostic laparoscopy is an option [3] but not golden standard, with all due respect paradoxically.

Mesri M *et al* 2018 [2] investigation showed the main cause of surgical blunder leading to death were at first peritonitis due to intestinal perforation, and at second was thromboembolism in patients who underwent bariatric surgery in obese patients [2-5]. On the other hand, the extensive availability of qualification and quantification tools, mainly Computed Tomography (CT), Magnetic Resonance Imaging (MRI) and FACS Flow cytometry are valuable in the identification critical status of patients at risks, and with increased (microbial-)septic-shock, and septicemia [3]. Although, so much pathologic and hematooncological pathways are stagnant not elucidated that preventive approaches remain unclear alternatives.

Taken together, we are missing some links that could help us offer the best medical consultative services in the 21th Century and I am feeling guilty of to admit that.

My take home message is if one cannot diagnose appropriately, just admit it and try not to make it difficult for the patients and their families because Medici's decision may have expensive consequences for them.

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