

Climaxes of Relationship between Cancer, Microorganisms and Platelets

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From different global research data could be argued that the main cause(s) of high mortality and morbidity rate is (are) still 'death triangle' machineries consisting of cancer- microorganisms-platelets as main critical (in-)direct cause(s).

Human platelets (PLTs) are enucleated blood cells derived from bone marrow under control of Thrombopoietin hormone, which circulate and have a shelf life between 9 up to 18 days [1]. PLTs obviously consist of many kinds of adhesive molecules i.e. PCAM, P-selectin exclusively. Some (un-)published proteomics data has claimed that between 40 - 60% of PLTs be made up of adhesive molecules. Isolated PLTs concentrates (PCs) produced in the blood bank could be used to safe life of many patients via terminating their bleeding disorders and restoring haemostasis especially cancer patients and patients, who are suffering from PLTs function defects [2].

Besides, microorganisms are small antigens, which primarily do not cause harm to human, but some kinds are extremely dangerous due to 1. rapid proliferation and differentiation, 2. aggressiveness of their toxins, 3. capability of RNA/ DNA manipulation, 4. additive and/or synergistic collaboration with another microorganisms, where might cause an increase in mortality and morbidity rate of subjects, dramatically. Bacteria are very small living entity that are made up of only one cell. Most types of bacteria aren't harmful, but some can infect people and cause diseases. A few have even been linked with cancer. Stomach cancer is one of the more common types of cancer worldwide. Long-term infection of the stomach with *Helicobacter pylori* (*H. pylori*) can cause ulcers. It can also inflame and damage the inner layer of the stomach. Some of these changes could lead to cancer over time, especially cancer in the lower part of the stomach [3,4]. *H. pylori* infection is also linked with some types of lymphoma of the stomach. While *H. pylori* infec-

tion is a major cause of stomach cancer, most people who have these bacteria in their stomachs never develop stomach cancer. There are some evidences, which indicate people with *H. pylori* might have a lowered risk of some other types of cancer, although it is unclear exactly what role the bacteria play in this process.

Antibiotics and other drugs could be used to cure different kinds of infections. However, testing for, and treating *H. pylori* infection is recommended after removal of an early stomach cancer. [3,4] Different studies postulated that in one hand, using antibiotics against *H. pylori* infection to eradicate it results in hematologic side effects i.e. (chronic) Immune thrombocytopenia (ITP) in some patients [3,4]. Recall, chronic ITP could lead into bleeding disorders and even death. On the other hand, the lymphatic vessels express not only platelet endothelial adhesion molecule-1 (PECAM-1) but also the intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) in inflamed and uninflamed human small intestine and tongue, and lymphatic endothelium enhances VCAM-1 and ICAM1 production with tumor necrosis factor-alpha (Sawa., *et al.* 1999, 2007; Ebata., *et al.* 2001).

It is well established that VCAM-1 and ICAM-1 induction needs a signaling pathway that involves protein kinase C and p38mitogenactivated protein kinases-mediated activation of transcription factors nuclear factor-kappa B and activator protein 1 (Voraberger., *et al.* 1991). Overexpression of the adhesion molecules in the systemic and lymphatic circulating system are not considered as physiologic process. Adhesion molecules might increase risk of agglutination, aggregation of blood and lymphatic cells, (ir-)reversibly. How (non-)epithelial and/or (non-)endothelial cells respond to abovementioned pathological overexpression is not elucidated yet.

Taken together, how cancer-platelets-microorganisms (CPM) so-called 'death triangle' relate to each other, and how they affect (un-) known processes, is not elucidated yet. Which processes are either underestimated or overestimated is not clarified as well. Furthermore, what 'one' is observing about the CPM relationship is continuously changing concerning 1. the way of prognosis, 2. Diagnosis 3. Pretreatments and treatments, 4. cure and care plans. While none of the (non-)standardized guidelines works; alternative new and mouthful treatments also could not offer significant progressions (with all due respect) in lowering Mortality and Morbidity Rate of Diagnosed 5 years-Cancer Patient Survival Chance (MMRD5YCPSC). The MMRD5YCPSC phenomena still remains a mystery for Medici, after a Century, however. I hope that we as Medici recognize that in 21st Century that no mistakes/side effects of treatments are allowed anymore. We got millennia Medical Field/Fiction experiences and almost 100 years Scientifically evidence-based ones [5].

Bibliography

1. Harker LA. "Platelets survival time: its measurement and use". *Progress in Hemostasis and Thrombosis* 4 (1978): 321-347.
2. Panlilio AL and RF Reiss. "Therapeutic Plateletpheresis in Thrombocythemia". *Transfusion* 19.2 (1979):147-152.
3. Abdollahi A1., *et al.* "Is Helicobacter pylori infection a risk factor for idiopathic thrombocytopenic purpura in children?". *Annals of African Medicine* 14.4 (2015): 177-181.
4. Rahman MM and McFadden G. "Modulation of tumor necrosis factor by microbial pathogens". *PLoS Pathogen* 2.2 (2006): e4.
5. Scopel-Guerra A., *et al.* "The Impact of Helicobacter pylori Urease upon Platelets and Consequent Contributions to Inflammation". *Front Microbiology* 12.8 (2017): 2447.

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