Introduction

Today thrombosis is still one of the main causes of affecting mortality and morbidity rate either in-hospital or out of hospitals [1-3]. Based on the last Century’s different global research data could be claimed that the main cause (s) of high mortality and morbidity rate is ‘death triangle’ machineries consisting of Cancer-Microorganisms-Platelets (CMP) [4].

Toxins are toxic substances, which can increase morbidity and mortality rate as well. Microorganisms’ toxins (Mots) are small antigens, which primarily are extremely dangerous due to:

• Their rapid propagation and aggressiveness,
• capability of RNA/ DNA damage and manipulation,
• additive and/or synergistic effects with reactive oxygen/nitrogen species, and
• still un-known mechanisms that are correlating with Platelets (PLTs) and thrombosis [1-5].

A few kinds of Mots after certain (un-)known drugs have even been linked with cancerogenous progressions [5,6]. Stomach cancer is one of the more common types of cancer. H. pylori infection is also linked with some types of lymphoma. While H. pylori infection is a major cause of stomach cancer, most people who have these bacteria in their stomachs never develop 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 released Mots is in these processes.

Different studies postulated that in one hand, using antibiotics as exogenous toxins against H. pylori infection to eradicate it results in hematologic side effects i.e. (chronic) Immune Thrombocytopenia (ITP) in some patients [5].

How PLTs and (non-)epithelial and/or (non-)endothelial cells respond to abovementioned pathological overexpression is not elucidated yet. How the CMPs relate to thrombosis and thrombo-emboli? And how they affect (un-)known thrombosis and bleeding disorders is not elucidated, as well.

Our research team have recently shown that PLTs (ir-) responsiveness is depends on three important factors:

• Activators’ type,
• Final concentration of antigens
• Biodiversity of subject’PLTs and content of PLTs during stimulation, under in-vitro and ex-vivo conditions [7].

Moreover, human PLTs’ response differently and inconsistently to the same activators during a day(weeks), which is understandable due to their dynamic and kinetic ageing in circulation and release of their content in an irreversible apoptotic manner. Subsequently, after any random treatment one get thrombosis disorders, and another bleedings disorder, however.

If we summarized all last Century’s Scientific data in a sentence for ‘God’s Sake’ and/or “patients’ Sake” we observe significant progression in technologies and tools but still Medici cannot prevent death triangle CMPs’ machinery. All three separately and/or in a synergistic combination still can increase morbidity and mortality rate of end stage patients.

Obviously, there is a leak in the appropriate care and cure Guidelines. However, Medici either miss the point or are not able to make a ‘Standard Guideline’ to prevent death. If Medici miss the point and/or ignore that! whatever the reason, it would be mission impossible! for non-Medici to solve the problem.

References


