



## Thrombotic Disorder Correlates with 'Death Triangle' Machinery

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Today thrombosis is still one of the main causes of affecting mortality and morbidity rate either in-and/or out of hospitals [1-3]. Recently different studies showed that the main cause(s) of high mortality and morbidity rate is still 'death triangle' machineries consisting of Cancer-Microorganisms- Platelets (CMPs).

Cancer treatments' side effects results in an unintentional cancer-related thrombosis (CAT). From 153 years ago was CAT indicated as an important death cause [4]. From different data published in both Europe and the USA become obvious that cancer patients have 2-up to 20-fold higher risk of suffering venous thromboembolism (VTE) and deep venous thrombosis (DVT) than other patients [5].

Microorganisms' toxins (Mots) are small antigens, which primarily are extremely dangerous due to:

1. Their rapid propagation and aggressiveness,
2. Capability of RNA/DNA damage and manipulation,
3. Additive and/or synergistic effects with reactive oxygen/nitrogen species,
4. Still un-known mechanisms that are correlating with platelets (PLTs) dysfunctions [1-5].

A few kinds of Mots after certain unknown drugs have even been linked with cancer 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. Different studies postulated that in one hand, using antibiotics and different drugs as exogenous toxins against *H. pylori* infection to eradicate it results in hematologic side effects i.e. (chronic) Immune thrombocytopenia (ITP) and in others thrombosis in some patients [5-7].

How PLTs and (non-)epithelial and/or (non-)endothelial cells respond to abovementioned pathological overexpression is not elucidated yet. One can speculate that might increase concentration of toxins in blood circulation induce premature release of adhesion molecules and/or prothrombotic proteins from PLTs' granula. Recent studies showed that PLTs (ir-)responsiveness depends on three important factors 1. activators' type, 2. final concentration of antigens 3. biodiversity of subject' PLTs and content of PLTs during stimulation, under in-vitro and ex-vivo conditions. 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 [1-3]. Subsequently, after any random treatment one get thrombosis disorders, and another bleedings disorder, however.

Apparently, there are still important mis guidelines resulting in side effects, and an intentional increase in the morbidity and mortality rate in-hospitals, however. On the other hand, Medici obviously' miss the point' continuously to make an appropriate standard guideline to tackle side effects of treatment, timely. Future research and developments needed to unravel what exactly is the correlation between CMPs, and how they affect morbidity and mortality rate significantly.

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