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POSTCOVID-19 WAR era, reconsideration over human platelets diagnostics on earth versus space

Abstract

In these postcovid-19 periods excessive mortality and morbidity rates became a fact-based happening that indicate catastrophic progression of science-based limited (re)action. Different aspects of cancerogenic processes based on my invented death triangle model system are very important to reconsider for an appropriate diagnostic and consecutively Medicare and Medicaid.

Keywords: blood cells, patients, platelets, leucocytes

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Introduction

Platelets (PLTs) are very important blood cells involved in Thrombosis and Hemostasis (T&H), (Patho)physiologic growth, (Patho)physiologic anti-microbial (ir-)responsiveness, cellular and tissue's repair processes and especially play pivotal role in excessive mortality and morbidity rates, postcovid-19 infections.[1,2] PLTs triple A (re)action namely Activation, Adhesion and Aggregation play pivotal role in death triangle machinery model system that I invented 5 years ago.[2] Moreover, globally an excessive mortality and morbidity rates in longCovid- patients did assess important role of PLTs in T&H of systemic blood circulation which still exact mechanism between PLTs involvement with different COVID-19 variants in cancer patients and their excessive mortality rate on earth[1] and on space (s' Travelers) not completely elucidated yet. One of the Clinical confusing aspects is miscommunication with laboratory blood tests i.e. Complete Blood Count (CBC) reports, which in the last 5 years were not reported consistently and might such miscommunications over Pseudo-Thrombocytopenia (PT) that have resulted in bias-based treatments, postcovid-19 pandemic attacks periods since 2019.

Old fashioned definition and guidelines described PT either as a bias-based handling with synthetic anticoagulants reversible chemical effects or has been documented in patients affected by a wide variety of disorders, including autoimmune disease, chronic inflammatory disease, viral and bacterial infections, metabolic syndromes, and neoplastic diseases, as well as after allogeneic stem cell transplantation.[3] PT does not pose a hemorrhagic or thrombotic risk to the patient. A subject with PT does not require clinical or laboratory monitoring for this completely benign condition. The only clinical implication resides in its lack of recognition, which potentially could cause inappropriate platelet transfusion, unnecessary additional testing, and delays in diagnostic or therapeutic procedures.[3]

Indeed, reports describe patients with PT who have been hospitalized and subjected to unnecessary transfusions of PLTs concentrates[4] delayed recognition of PT may also determine a state of unjustified alarm in the patient, as well as precautionary behavior for fear of hemorrhage due to thrombocytopenia. In some healthy subjects, the presence of PT could be documented for more than 20 years. However, in at least half of the patients, it was determined that PT was not present in previous years but had appeared suddenly with no apparent cause.[3] Different publications reported that after PT appears, it usually remains indefinitely but, in a few cases, it is transient and disappears after some time. A potential issue arises when subjects with PT require surgical intervention under hypothermia, as occurs in heart surgery. However, Nicola Bizzaro et al.[3] and others have reported that surgery can be performed without complications in patients who undergo cardiac surgery for coronary artery bypass grafting and valve replacement.[3–5]

Another interesting aspect is that related to the possible implications of transfusion of blood obtained from a donor with PT. In the 2 cases of Sweeney et al., and in the 10 cases reported by Maslanka no problems arose in the recipients, not even the appearance of PT, indicating that the blood of healthy subjects with PT can be used in transfusion therapy.[6–9] Recall all abovementioned studies were published before COVID-19 pandemic attacks, a decade ago. Wahed and Dasgupta described that PT causes have different etiologies i.e. giant PLTs, anticoagulant-induced PT, PLT's satellitism, and cold agglutinin-induced PLT agglutination.[7] Moreover (pro)inflammatory response to the COVID-19's vaccines, during pandemic attacks and different bacterial and viral infections is not completely understood yet.

The PT may remarkably occur with giant PLTs which because of their large size, could be excluded from electronic cell counters. This scenario is of particular clinical importance in patients with rapid consumption of PLTs in the peripheral circulation as observed in disseminated intravascular coagulation (DIC), acute immune thrombocytopenic purpura, or thrombotic thrombocytopenic purpura.[6–9] To assess synthetic occurrence of PT timely, either correction in laboratory setting or an effective PLTs production by bone marrow-checkup needed. Rechecking blood smear without anticoagulants just from extra capillary blood drops from finger is a good alternative approach. In such cases one can observe many large PLTs in peripheral blood, many of which may not be identified by automated cell counters, and electronic analyzers. Besides that, an accurate PLTs count can be also obtained with a manual count using phase contrast microscopy, as well. The PLTs agglutination (weak binding and reversible) synthetic anticoagulant-induced PT is an *in-vitro* PLT-PLT (weak) binding and agglutination phenomenon generally reported previously in random specimens collected in EDTA tubes. It has been reported both in healthy subjects and in patients with various diseases (including collagen vascular disease and neoplasm), and it has an overall incidence of approximately 0.1%.[7,8]

The PLTs aggregates (not only strong binding but also irreversible) in PT is usually indicating that internally PLT-PLTs activation-

adhesion- aggregation are took place mostly not only temperature sensitive but also thromboxane release (TXA2)-dependent that could not be reversed, however. It is very important not only to differentiate between PTs with agglutinated versus aggregated samples; but also to report it to clinicians and GPs, eventually. It has been noted that in Glanzmann's thrombasthenia, a disorder characterized by the quantitative and/or qualitative abnormality of GpIIb/IIIa, PT does not occur. Interestingly, in recent years, abciximab (a GpIIb/IIIa antagonist) has been found to be associated with PT- forming.[6–8] Whether such reports clinically are important for clinicians and GPs to set priorities in their Medicare and Medicaid plans, remained not completely known procedure, remarkably.

In this POSTCOVID-19 periods, management of space travelers' cancerogenic risks is indeed important aspects as reported by Riego ML et al.[10] which could not only being associated more with chromosomal damage, and alternative transcription in human lymphocytes exposed to mixed ionizing radiation as encountered in space,[10] but also to certain factors, which I introduced in my death triangles machinery model system since 2019,[1] although the molecular exact mechanisms of the aforementioned interactions are still not completely elucidated yet.

On earth, PLTs satellitism has reported that have some features similar to synthetic / chemicals/ anticoagulant-induced PT, which is defined to have a reversible binding and agglutination characteristics. In the presence of EDTA, PLTs bind to leukocytes and form rosettes. In these postcovid-19 period which different COVID-19 mutants could be chronically escaped immune system in the circulation can also induce certain PLT-PLT and PLTs-Neutrophils agglutination (PNA), which PNA could be reconsidered as a very important biomarker of presence of different mutated COVID-19 variants in blood circulation of blood donors, in the long run. The PLT-binding to neutrophils is usually not insignificant observation, while synthetic reversible-PLTs' binding to other WBCs might be well insignificant. Automated analyzers do not identify PLTs that bind to leukocytes that resulting in PT under laboratory synthetic conditions, but PLTs satellitism, especially PNA is mediated by either pro-inflammatory processes, indicating microbial infections, or presence of certain autoantibodies against PLTs membrane' changes, which needs in detail future investigation. Different publications recently showed binding of PLTs to the neutrophil membrane, during either microbial- and/ or postcovid-19 infections are of utmost important indication to be (re)considered for either covid-19 contracted or longCovid- patients' indication (unpublished data). On space, Astronauts travelling will be exposed to mixed beams of particle radiation and photons. different exposure limits continue monitoring that correspond to defined cancer risk, which are calculated by multiplying absorbed doses by a radiation-type specific quality factor that reflects the biological effectiveness of the particle without considering possible interaction with photons.[10] A sincere question remains what would happen when some potential random Astronauts that have recessive viral infection i.e. AIDS, different COVID-19 variants,[11] during long time traveling. Which blood cells are more important to be monitored during their journey WBCs, RBCs or PLTs? Which blood cells might induce cancerogenic processes and might initiate a pro-apoptotic or T&H disorder in space travelers, from next years that space traveling become available.

Conclusion

Taken together, in these postcovid-19 periods excessive mortality and morbidity rates became a fact-based happening that indicate catastrophic progression of science-based limited (re)action. Different aspects of cancerogenic processes based on my invented death triangle model system are very important to realize from now on i.e. how the AI-chips and AI-tools can help either on Earth or in the space-associated traveling/ activities, human being under extreme (un) known condition can survive potential risks of cancer? Whether any science-based standard guideline is there for the next space travelers, to prevent cancer before, during and after space traveling for whom that being/are susceptible for cancer (ogenic processes).

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Conflicts of interest

The author declares that there is no conflict of interest.

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