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Research Article

POSTCOVID-19 War era, Platelets Dysfunction in different Diseases and their Medicare and Medicaid are Underestimated

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Abstract

PLTs dysfunction is increased during increased mortality and morbidity processes, in vivo however. During COVID-19 pandemic attacks what were the main cause(s) of PLTs dysfunction is also not elucidated completely.

In this paper is focused on different kinds of acute and chronically diseases, and consecutively, PLTs dysfunction after COVID-19 variants infection, as main infectious mutants (antigens) as concrete example, on one hand. On the other hand, focusing on the hematologic disorders like heparin induced thrombosis (HIT), PF4 disorders as not infectious disease, to better highlight PLTs (dys)functioning, that are not still clarified entirely.

Keywords: POSTCOVID-19 era, COVID-19, Infection, Human, Platelets, Cardiovascular Diseases, Cancer, Platelets Function, Medicare, Medicaid

If human platelets (PLTs) do normally function, then their involvement in Thrombosis and Haemostasis is a lifesaving process. There are more than 20 functions described for PLTs up to 2023. As described previously about PLTs' physiology and triple A's functions (Activation, Adhesion, Aggregation), PLTs have very delicate but explosive (ir) responsiveness / (re-)actions/ hypo-hyperactivity features that could be manipulated by metabolic suppression [1]. Recall, PLTs' cell-cell and cell-proteins kinetic and dynamic are temperature- metabolic- pharmacotoxicologic- and activator dependent functions, in vitro, ex-vivo and under the blood bank's related condition. How? And why in vivo they show different hypo- and hyperactivities is not completely elucidated yet (2023).

PLTs dysfunction is increased during increased mortality and morbidity processes, in vivo however. During COVID-19 pandemic attacks what were the main cause(s) of PLTs dysfunction is also not elucidated completely. One is observing different clinical studies describing not only infected subject that showed Pathophysiological dysfunction of PLTs could be initiated by either external or internal antigens, but also hematologic disorders, physical changes of hard and soft tissues, and at last but not least vaccines as an unknown factors, affected PLTs dysfunction in infected COVID-19 patients(4-6).

PLTS dysfunction in different chronical diseases (internal inducers) and their associated Medicare and Medicaid (external inducers) of patients, are not also elucidated completely. For instance, during COVID-19 infections that were caused by different variants of coronaviruses (and associated vaccines 'side effects), nobody still defined exact mechanism of PLTs dysfunction, eventually. Hence, to highlight more about potential cause(s) of PLTs dysfunction, diseases could be divided into two main kinds 1.infectious and 2.not infectious concerning PLTs dysfunction.

In this paper is focused on different kinds of acute and chronically diseases, and consecutively, PLTs dysfunction after COVID-19 variants infection, as main infectious mutants (antigens) as concrete example, on one hand. On the other hand, focusing on the hematologic disorders like heparin induced thrombosis (HIT), PF4 disorders as not infectious disease, to better highlight PLTs (dys) functioning, that are not still clarified entirely.

PLTs dysfunction during/after infectious diseases

Microorganisms' attack and entrance to the body takes place in different kinds of body openings 1. Vast tissue i.e. skin 2. Fluid soft tissue i.e blood 3. Gas exchange organs i.e aerosol Gastrointestinal track systems, lungs and skin openings.

Physiologically, all body systems are protected by local specialized immune systems. The main blood (white blood cells, WBCs) and lymphatic (T- and B) cells involved in first and secondary immune system responses are abovementioned

cells' responses. Any pathological (ir-) responsiveness of immune system causes prolonged pathologic and chronic diseases, which might be progressively result in an increased mortality and morbidity rates, eventually. Simultaneously, PLTs play a pivotal role not only in Thrombosis and Haemostasis (TH), blood clotting, cancerogenic metastatic processes but also in mortal consequences of either different immune system failure, severe abused antibiotics or anticoagulants to manage certain procoagulant, prothrombosis, and/or anticoagulant/ antiplatelets processes. Nonetheless, PLTs were affected by COVID-19 variants (-vaccines), especially PF4 (6) which is resulting in different uncontrolled and severe clinical episodes, and an increase in morbidity and mortality rates (3-6). Moreover, suggested that Vaccine-induced immune thrombotic thrombocytopenia (VITT) has a rare adverse effect of COVID-19 adenoviral vector vaccines (3-6), remarkably [2]. With using a functional platelet activation assay (serotonin release assay; SRA), they showed that VITT and HIT samples had unique patterns of PLTs reactivity in vitro. Their study did offer an explanation for VITTmediated PLTs activation (3). Although still is not completely elucidated why in some COVID-19 'patients PLTs dysfunction that did show both disorders i.e. not only (hyper) reactivity/Thrombosis but also hypoactivity; causing fatal severe bleedings disorders.

Marie Scully et al. 2021 reported in their study results(Quote)…In the absence of previous prothrombotic medical conditions, 22x patients presented with acute thrombocytopenia and thrombosis, primarily cerebral venous thrombosis, and 1x patient presented with isolated thrombocytopenia, and a hemorrhagic phenotype [3]. No evidence of thrombophilia or causative precipitants was identified (6). Testing for antibodies to platelet factor 4 (PF4) was positive in 22x patients (with 1x equivocal result), and negative in 1x patient. On the basis of the pathophysiological features observed in their patients, they did recommend that treatment with platelet transfusions be avoided, because of the risk of progression in (pro-) thrombotic symptoms, and that the administration of a non-heparin anticoagulant agent, and intravenous immune globulin be considered for the first occurrence of these symptoms. Suggested that a pathogenic PF4-dependent syndrome, unrelated to the use of heparin therapy, can occur after the administration of the ChAdOx1 nCoV-19 vaccine (6) [3]. Rapid identification of this rare syndrome is important because of the therapeutic implications (6). The still remaining sincere question did remain why "externally modified certain vaccines produced between 2019 and 2023" did initiate random PLTs dysfunctions, however? One is speculating that might weakened/ death viruses, processed in certain vaccines were could initiate PLTs (re)activation, and induce prothrombotic disorders, (UN) intentionally.

Hence, future detail investigation might unravel how certain vaccines did kill their subjects between 2019-2023. Previously our research study teams have already described death triangle machinery, and how might certain microorganisms change PLTs (dys) function after infecting certain patient. Moreover, based on literature studies and own pilot internal research studies one did observe significant changes in the RBCs and WBCs composition [4]. Now by different study groups is significantly shown that there are different primary, secondary and even tertiary signal transduction take place between microorganism and PLTs, prior to transforming patient's condition from a reversible /curable condition into irreversible mortal situation.

PLTs dysfunction during/after not infectious diseases

There are significant amount of studies in PUBMED, which have shown that external and internal inducers i.e. Accidents, diabetic and metabolic syndromes, pharmacotoxitologic agents, and chronic diseases have significant effect on increase in blood composition and consequently, platelets hypo- hyperactivities. Why? And how in a patient's PLTs dysfunction results in hypo- and in almost the same condition in another subject researchers observed hyperactivity of PLTs, is not elucidated as well. As previously reported, one is observing a significant increase in POST-ICU treatments concerning PLTs dysfunction and inducing bleeding disorders (UN) intentionally (1). PLTs are normally form blood clots, but in lung fibrosis they become also involved in immune cell functions that end up attacking healthy cells and damaging the lung. While the immune system is supposed to protect us from viruses and bacteria, in patients with lung fibrosis it harms their own body. Furthermore, pulmonary fibrosis is a medical condition characterized by the gradual scarring of lung tissue. In individuals with pulmonary fibrosis, the normal lung tissue becomes thickened, stiff, and scarred over time. This scarring, also known as fibrosis, makes it difficult for the lungs to function properly and can lead to breathing difficulties. In the last 4 years, accelerated mortality and morbidity rates in different cardiovascular patients are remarkable. What would be the accelerating cause(s) that has not still revealed yet? Was/ is there any association between COVID-19 variants- PLTs- lung fibrosis and accelerated mortality in chronic patients, recently (NOV-2023).

Postulated that preeclampsia can lead to significant adverse outcomes, including fetal and intrauterine growth restriction, severe maternal hypertension, and endorgan dysfunction that may result in preterm delivery, placental abruption, and maternal and perinatal death [5]. The cause of preeclampsia is still unclear, but there is evidence that platelets may be pivotal mediators of its complications, linking inflammation and thrombosis with endothelial and vascular dysfunction. Procoagulant PLTs' factors are increased in patients with preeclampsia, such as plasma levels of transforming growth factor-β (TGF-β) and platelet factor-4. Such a degree of procoagulation is not present in a healthy pregnancy [6, 7].

There are still remaining questions which stills did not get appropriate attention i.e. how? And why PLTs (dys) function is remaining underestimated and unknown processes (NOV-2023)? One is observing that might be because of 1.lack of know-how to measure platelets' function simultaneously in vivo and ex-vivo; and 2. Basic and clinical researcher (un-)intentionally pay no/limited attention to such crucial aspects and questions.

After 7 up to 30 million died because of the COVID-19' variants and associated drugs/ vaccines abused during 2019-2023, One is observing no /limited significant research study group that (re)considering study of (co-)relation between COVID-19 variants and significant changes in PLTS' triple A's (dys)functions and associated blood cells' changes. Taken together, after 4 years intensive Research studies still there is not complete information about what did happen between 2018-2023, which might play pivotal role in the main mechanism of action, and were induced infected patients' situation to an extend that can initiate hypo- hyperactivities of own PLTs, randomly. The near future investigation should focus on personalized medicine, associated diagnostics and basic studies to unravel how and why one patient gets hypoactivity / (ir)responsiveness of PLTs.

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