CPQ Medicine (2019) 7:2
Editorial

Prevention Strategies of Metastatic Cancers by Understanding Key Role of the PLTs Microparticle and Exosomes

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Received: 18 June 2019

Published: 21 June 2019

Keywords: *Metastatic Cancer; Microparticle; Exosomes*

Prevention of the Metastatic of Malignant Cancer Tissue and Cells Proliferation (MMCTCP) is a lifesaving approach for any cancer patient. Although, the possible mechanism is not elucidate completely yet.

Song Z *et al.* have suggested that liquid exosomes can be used as biomarkers for the diagnosis of Lung Cancer (LC). Cells release membrane vesicles in their surrounding medium either constitutively or in response to activating signals [1-5]. Two main types of extracellular vesicles (EVs) are commonly distinguished based on their I. mechanism of formation, II. membrane composition and III. size. The biological EVs shed from the plasma membrane, often called microvesicles (MVs), expose phosphatidylserine (PS) and range in size from 100nm to 1µm, while EVs originating from endosomal multi-vesicular bodies, called exosomes (EXos), contain tetraspanin proteins, including CD63, and range in size from 50 to 100nm [1,3,5]. How MVs and Exos are involved in manipulation of the Cancer-Related mortality and morbidity is not elucidated as well.

Brisson *et al* (3) show that EVs activated by these three agonists present a similar size distribution, and also that 60% of the EVs from TRAP or CRP-XL activation expose the glycoprotein (Gp) IIb/IIIa integrin, also called CD41, a majority of them exposing also PS [2]. Recall, expression of CD41 is well accepted that CD41 is detected during Megakaryocytes (MK) differentiation at a stage of a late MK progenitor. The CD41, is the platelet receptor for fibrinogen and several other extracellular matrix molecules. Recent evidence suggests that its expression is much wider in the hematopoietic system than was previously thought [6].

The PLTs' microparticles are small extracellular vesicles abundant in blood. Plts' MVs have physiologic function in blood circulation. The mechanism of PLT MVs participation in specific disease entities such as rheumatoid arthritis has been elucidated [3], which after pathological processes might affect metastasis of malignant cells [1,5,6]. More recent studies have demonstrated that PLT MVs function as a transport and delivery system for bioactive molecules, participating in hemostasis and thrombosis, inflammation, malignancy infection transfer, angiogenesis, and immunity [3]. New evidence indicates that the majority of CD41+ MVs circulating in healthy individuals derive directly from MKs. The CD41+ Mvs also form from activated PLTs upon loss of cytoskeleton-membrane adhesion, which occurs in a multitude of disease states characterized by elevated PLT microparticle levels [3-6]. Circulating MVs originating from MKs are differed from those derived from activated PLTs by the expression of CD62P, LAMP-1, and immunoreceptor-based activation motif receptors. Under physiologic flow, Mvs bud off from long membrane strands formed by activated PLTs. The PLT MVs are commonly characterized by the expression of surface PLT antigens and PS flip flop. Actually, only a fraction of PLT Mvs harbor PS, and a distinct subset contains respiratory-competent mitochondria [3-6]. Those PLTs that flip flop their PS are larger in size, have an increased circularity index, and have a reduced internal complexity compared with non-PS-exposing ones. Recall, activated PLTs express CD62P and αIIbβ3 in an inactive conformation on the surface. While their functions have yet to be fully elucidated, the heterogeneity of PLT-associated and released EVs suggests that they may contribute to different aspects of procoagulation and Cancer-Associated-Thrombosis (CAT) [3-6]. Understanding exact mechanism of MVs and EXOs signaling might help earlier prevent MMCTCPs. Recent data from six Countries in European countries have shown that managing Thrombosis episodes in a certain cancer patient is one the best ways to prolong life expectation, and enhance healthy life style. Further investigation will show whether thrombocytes management could be used to prevent mortality rate of other diseases as well.

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