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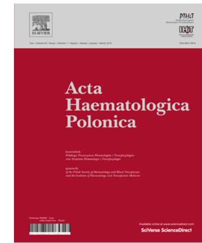

Review/ Praca poglądowa

Methods for detection of microparticles derived from blood and endothelial cells

Metody oznaczania mikrocząstek pochodzących z komórek krwi i śródbłonka

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Q2 Microvesicles are a heterogeneous group of membrane vesicles, measuring 0.1–1 μm in diameter that are released from the cell surface in response to activation or apoptosis [1–3]. In physiological conditions MVs are formed during cell maturation and aging, and increased number of circulating MVs has been associated with many pathological conditions [2, 4]. MVs are non-nucleated, cell-membrane coated vesicles containing the same surface antigens as their cell of origin [5].

Our knowledge on the morphology and function of MVs is still incomplete. This is due to the difficulties in separating MVs from other types of cells, the inability of most techniques to capture particles in a volume range of MVs, and low availability of costly and time-consuming methods for the detection of MVs [6]. The majority of studies were performed on MVs originating from platelets, endothelial cells, and monocytes [7].

Generation of microvesicles

The formation of MVs is a regulated process, leading to selective and concentrated release of cell contents to the surrounding environment. Both the parent cell and a trigger determine the number, size, and antigen composition of the released MVs [8]. Circulating MVs are released to the plasma from the surface of all types of blood cells: red blood cells, granulocytes, lymphocytes, monocytes, platelets, endothelial cells, and tumor cells. The number of MVs in the blood is the result of a dynamic balance between the release of apoptotic and activated cells, and their clearance from the circulation [8–10]. In healthy subjects, the majority of detected MVs come from platelets, whereas MVs originating from erythrocytes, leukocytes, and endothelial cells are much less abundant [7, 11]. Cell membrane is the first structure involved in the formation of the microvesicles.

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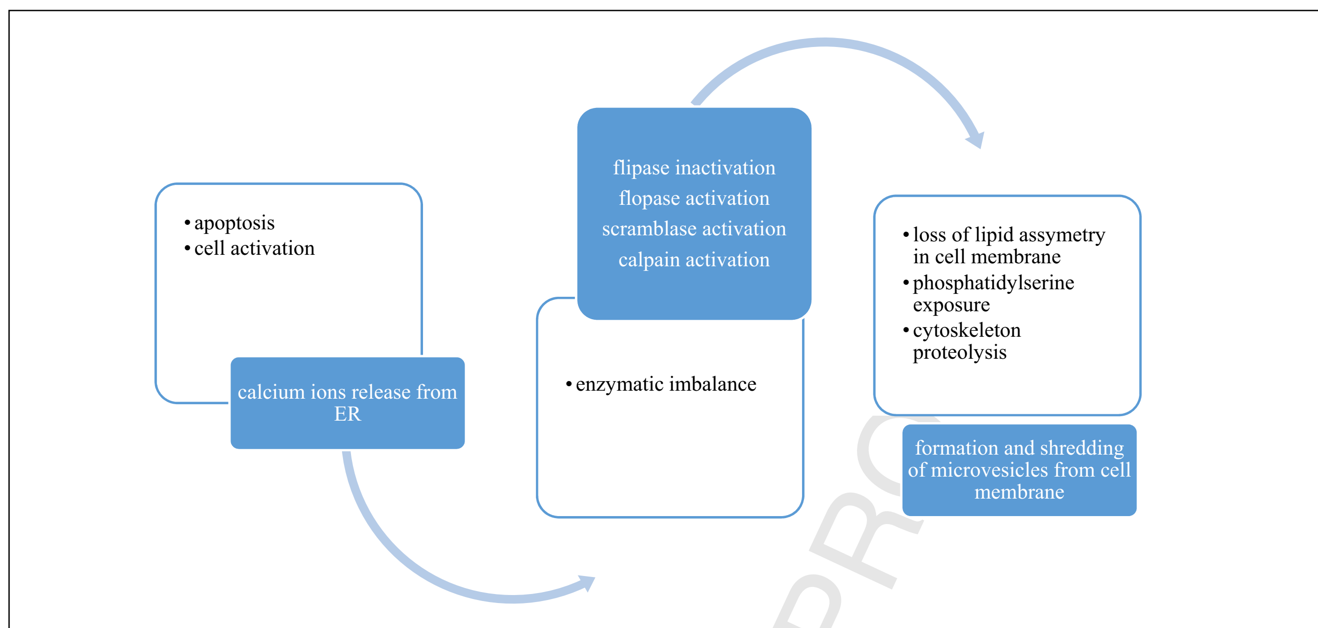

Fig. 1 – The formation of microvesicles

Under homeostatic conditions phospholipids, phosphatidylserine and phosphatidyloetanolamine, are arranged on the inner (cytoplasmic) layer of the membrane while phosphatidylcholine and sphingomyelin are localized in the outer layer. The asymmetry is a key factor for maintaining cell homeostasis and is regulated by an enzyme complex: flippase, floppase and scramblase. Flippase is responsible for keeping the phosphatidylserine and phosphatidyloetanolamine on the inner side of the membrane, while floppase keeps phosphatidylcholine and sphingomyelin on the outer side. Scramblase, responsible for the transport of phospholipids between the two monolayers of the cell membrane, is inactive in the steady state. Loss of phospholipid asymmetry and externalization of phosphatidylserine trigger the formation of MVs [7, 9, 12, 13]. Not all MVs exhibit phosphatidylserine on the surface, and their contents may vary depending on the cell of origin as well as the stimulus triggering the formation of MV [1]. The formation of MVs is depicted in Fig. 1: apoptosis and cell activation cause a sudden release of calcium ions by endoplasmic reticulum. The increased calcium ions flux results from enzymatic imbalance caused by the inactivation of the flippase, floppase and scramblase, leading to the loss of lipid asymmetry in the cell membrane and exposure of phosphatidylserine on the surface of the cell. The release of calcium activates protein proteases e.g. calpain leading to the proteolysis of cytoskeleton resulting in shedding of MVs from the surface of the cell membrane [7, 9, 12, 13].

Microvesicles are formed in response to the activation induced by various stimuli (thrombin, collagen, epinephrine, diphosphoadenosine) and ex vivo during preparation and storage of blood products for transfusion [14, 15]. Approx. 70–90% of circulating MVs originate from platelets [9, 16], 10–15% from endothelial cells [17], 4–8% from red blood cells [18], and their formation is an essential stage of red blood cell aging [19]. Basic characteristics of MVs are shown in Table I.

Table I – The characteristics of microvesicles

1.	Formation triggered	By activation or apoptosis through blebbling of cell membrane
2.	Diameter	0.1–1 μm
3.	Nucleus	No
4.	Composition	Lipids and proteins, can contain mRNA, miRNA
5.	Structure	Outer layer contains phosphatidylserine, which in normal conditions is localized in the cytoplasmic layer
6.	Antigens	Specific surface antigens derived from the parent cell
7.	Origin	Platelets, endothelial cells, erythrocytes, monocytes, lymphocytes, and granulocytes
8.	Number	Depends on the balance between release and removal from the circulation
9.	Present in physiological conditions	Increased number in various pathological conditions

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