The effect of red blood cell aggregation and how it can be reversed

T Lew Lim, MedicLights Research Inc., Toronto, Canada.
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Introduction

The blood profile reveals a person's state of health. If we look at a blood sample of an unhealthy subject, we will see that the red blood cells (RBCs) are sticking together (aggregating) and behaving like glue – with high resistance to flow ("high viscosity").

Under a microscope, the blood sample of an unhealthy subject would look something like in figure 1:

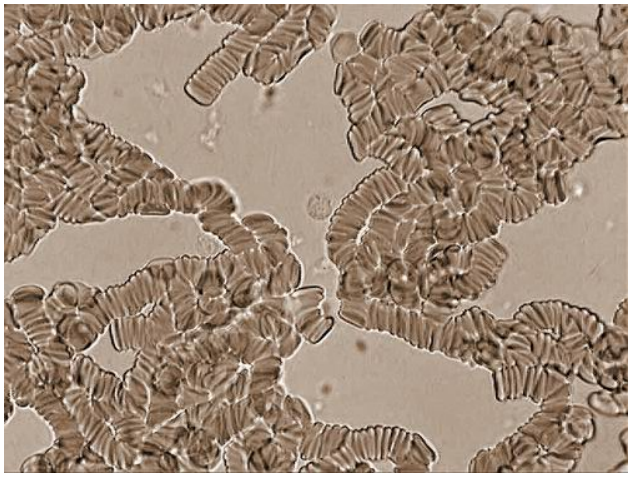

Figure 1: Blood sample of an unhealthy person

With the same person in a healthy condition, his blood sample would look entirely different, as in figure 2 below:

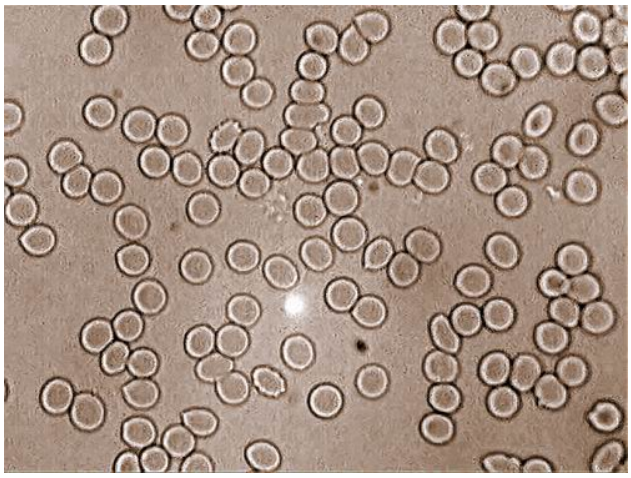

Figure 2: Blood sample of a relatively healthy person

Visually, it is clear that the main difference between the two samples is how the red blood cells (RBCs) or erythrocytes are seen to be aggregated in figure 1 and disaggregated in figure 2. It is not possible to demonstrate from a static picture like this but when the blood samples were taken, the blood in figure 2 was also observed to be less viscous.

"Blood aggregation" was coined as early as 1786 by health knowledge leaders. Occasionally it is called "blood sludging". Whatever the phenomenon is called, it has been associated with many diseases.[i]

This may surprise many: the two samples above are actually "before" and "after" pictures of blood samples taken only 30 minutes apart from the same subject after using a device like the Qi-Light by RadiantLife as shown in Figure 3 below. They are typical of many who have used the device.

Figure 3: Qi-Light device in use

Effect of RBC aggregation on blood circulation and tissue perfusion

The following is extracted from a review of evidence-based work on RBC aggregation by Baskurt and Meiselman.[ii]

Effect of RBC aggregation on microcirculatory blood flow

In studies employing intravital microscopy it has been observed that intensified RBC aggregation increases microvascular flow resistance.[iii] RBC aggregation in arterial and/or capillary microcirculation would affect the bulk viscosity of blood in larger blood vessels and the increased energy cost of disaggregation at the entrance of microcirculation.[iv]

RBC aggregation has also been found to decrease the density of functional capillaries, resulting in decreased overall microcirculatory blood flow. This is found to be especially significant under reduced arterial pressure.[v] Enhanced RBC aggregation also fills capillaries with non-flowing RBC making these capillaries dysfunctional.[vi]

Effect of RBC aggregation on venous flow resistance

The venous side of the circulatory system returns deoxygenated blood back to the heart and lungs to discharge the waste and carbon dioxide. Blood flow in the venous circulation is characterized by lower shear rates (change in velocity of blood flow parallel to vascular walls) compared to the arterial side. For this reason occurrences of RBC aggregation are more significant in the venous circulation areas.[vii]

Experiments show that normal RBC aggregation significantly contributes to venous vascular resistance in resting muscles and plays a significant role in vascular system homeostasis (stable bodily state).[viii] It has also been demonstrated that RBC aggregation affects velocity profiles in venous blood flow, especially under reduced flow rates.[ix] The energy loss caused by RBC aggregation contributes to flow resistance.

Effect of RBC aggregation on whole organ perfusion

A study isolated a rat's heart and then perfused it with a different concentration of dextran (a compound often used by researchers to simulate RBC aggregation). The results suggest that relatively low levels of RBC aggregation may reduce flow resistance in the organ, whereas greatly enhanced aggregation greatly increases resistance to blood perfusion to the organs.[x]

Effect of RBC aggregation on tissue hematocrit

At the tissue level, tissues are also adversely affected when they are not properly provided with nourishing blood. They are served by the network of capillaries; and as discussed, if the flow is impeded by RBC aggregation, their condition will be adversely impacted. There are however, specific studies that look into tissues: more specifically on hematocrit (the ratio of blood volume

occupied by RBC). Tests demonstrate that alterations in RBC aggregation induced by fibrinogen infusions affect tissue hematocritic values in rat myocardium (muscular middle layer of the wall of the heart).[xi] These experiments were also repeated using more recently developed poloxmer coating method to alter RBC aggregation. These confirm that enhanced RBC aggregation alters myocardial tissue hematocrit values.[xii]

Summary

In summary there is substantial evidence to show that RBC aggregation impedes blood flow. The presence of RBC aggregation affects microcirculatory blood flow, venous flow, whole organ perfusion, and tissue hematocrit.

The theoretical explanations for LLLT disaggregation of RBC

The anti-inflammation theory

As mentioned earlier, the presence of RBC aggregation is commonly attributed to inflammation which then stimulate the release of fibrinogen into the blood circulation system.

When inflammation is reduced through LLLT, the level of fibrinogen in the blood will also be reduced. As the result RBCs will be visibly disaggregated.

Literature suggests that LLLT reduces inflammation through healing and regeneration actions, amongst which include stabilizing the cellular membranes[xiii], enhancing ATP production and synthesis which contribute to cellular healing[xiv], vasodilatation (dilation of blood vessels)[xv], acceleration of leukocyte and lymphocyte activities to remove damaged cellular components and allowing for more rapid repair[xvi], and helps regenerate blood capillaries[xvii].

There is also a recent study suggesting that LLLT illumination "decreases the amount of inflammation and accelerates the wound healing process by changing the expression of genes responsible for the production of inflammatory cytokines." [xviii]

The photodissociation theory

Tests on rabbits established that hemoglobin is a primary photoacceptor absorbing low-intensity laser radiation of light of red and infra-red ("IR") wavelengths. The exposure of blood to this radiation causes clearly defined changes in the IR and visible absorption spectra of the blood and erythrocytes (red blood cells). These spectral changes arise as a result of partial photodissociation (breaking down of chemical compounds with light) of hemoglobin-ligand (substance to bind biomolecules) complexes in the process of absorption of laser radiation. It is suggested that photodissociation is a primary reaction that arises in blood exposed to a low-intensity laser radiation.[xix] This result is the disaggregation of aggregated RBC.

The transient local heating hypothesis

Upon contact with blood, a substantial amount of absorbed red light energy is converted to heat, which causes a local transient increase in the temperature of absorbing chromophores (molecules

responsible for their colours). This local transient heating of absorbing molecules is different from the general heating of the whole cell, tissue and organism as commonly imagined. The local transient increase in temperature causes structural changes, and trigger biochemical activity that results in the disaggregation of RBC.[xx]

The negative surface charge theory

Some researchers suggest that LLLT raises and restore the negative surface charge of the RBCs, rebalancing the play with electrolytes, and restoring their natural state of the RBCs repelling each other.[xxi]

Summary

In summary, there are several theories as to how LLLT would neutralise RBC aggregation. What matters is that LLLT is proven to succeed in disaggregating RBC.

From stasis to red blood cell aggregation

Recent research also shows that stasis in the vein is associated with RBC aggregation.[xxii] The aggregation is found to be due to the presence of macromolecular proteins such as fibrinogen and globulin.[xxiii] Fibrinogen is the protein which is sensitive to inflammation and helps with the blood clotting function. It is a dominant factor in RBC aggregation.[xxiv] Since inflammation is caused by an illness or disorder, one can attribute the presence of blood aggregation to the presence of an illness or a disorder. Therefore when RBC aggregation occurs, it means that the subject is at least not in perfect health.

Red blood cell aggregation to shear stress

Blood has a non-Newtonian fluid behaviour. Once it gets moving and builds momentum, it flows fast. Unlike water, which exhibits much more Newtonian properties, blood moves sluggishly at low speeds and is more liquid at fast speeds. When the heart is resting between beats (diastole) it becomes more viscous and then when the heart forces blood out (systole), with the higher shear stress blood becomes less viscous.

It is important to recognise that the presence of high RBC aggregation (along with hematocrit and plasma viscosity) is a factor contributing to low shear stress. Low shear stress leads to high whole blood viscosity.[xxv] Eventually, elevated blood viscosity and RBC aggregation are important factors in affecting blood circulation, cardiovascular diseases and a number of diseases,

as found in the cumulative work of renowned haemarheology (blood flow) researcher, Dintenfas.[xxvi]

Low shear stress regions along the arterial walls is associated with the occurrence of lesions.[xxvii] This leads to deposit of plaque in these areas. When the plaque builds up, blood flow is further impeded, leading to turbulence and more plaque deposition. When the plaque deposition reaches a critical stage, it often causes vascular occlusive (blockage) disorder[xxviii], and thrombosis (blood clot) both in arterial and venous systems. This is the prelude to cardiovascular events such as heart attacks and strokes. Before that happens, the subject exhibits classic symptoms indicated in TCM with blood stasis – such as reduced immunity, increased tiredness and so on, making the subject vulnerable to diseases.

Effect of RBC aggregation on blood circulation and tissue perfusion

There is considerable amount of research conducted by a core group of researchers on the effect of RBC aggregation on blood circulation and tissue perfusion. Among the leading researchers are Baskurt and Meiselman. It is apparent that this body of research is growing with evidence that the interplay of these elements hold important keys that are central to a person's well being.[xxix] A more detailed extract of their review are presented in **Appendix 2**.

In summary there is substantial evidence to show that RBC aggregation impedes blood flow. The presence of RBC aggregation affects microcirculatory blood flow, venous flow, whole organ perfusion, and tissue hematocrit. Freedom from RBC would be a very desirable health condition.

Red blood cell aggregation is an independent risk factor

Studies also show that the presence of RBC aggregation is an independent risk factor for vascular occlusive disorder[xxx] and thrombosis both in arterial and venous systems[xxxi]. With these properties, RBC aggregation would also be identifiable with poor circulation in the small capillaries. These studies show that RBC aggregation is also related to blockages in the larger vascular network, caused by the formation of plaques.

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