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Research Article

Comparative Effects of Ascorbic Acid and Aspirin on Platelet Count and Aggregation in Albino Wistar Rats

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Abstract

The effect of oral vitamin C and aspirin supplementation on platelet count and aggregation in rats was studied. Twenty-one (21) albino Wistar rats weighing between 130 - 160g were used. They were grouped into three groups of seven (7) rats each. The first group served as control. The second group was administered with vitamin C at a dose of 200 mg/kg body weight, while the third group was administered with aspirin (300 mg/kg body weight) orally for 28 days. At the end of the 28 days, platelet count was done using standard procedure while platelet aggregation was done by determining platelet count ratio in EDTA and EDTA formation saline. The value of platelet count and platelet aggregation ratio in the control rats was 544 ± 27.0 x 103 cells/mm3 and 1.03 ± 0.02 x 103 cells/mm3 (Mean ± SEM) respectively. The platelet count in rats given vitamin C and Aspirin were 416 ± 24.4 x 103 cell/mm3 (Mean ± SEM), 426 ± 22.8 x 103 cell/mm3 (Mean ± SEM) respectively, while the platelet aggregation were 0.960 ± 0.02 x 103 and 0.915 ± 0.02 x 103 respectively. Platelet counts in vitamin C and Aspirin treated groups were significantly reduced when compared with the control group, but there was no significant difference between the counts in the groups given vitamin C and Aspirin. There was no significant difference in platelet aggregation in the groups administered with vitamin C and Aspirin when compared with the control group, and when compared with each other. It could therefore be concluded that Vitamin C and Aspirin reduce platelet count but do not affect platelet aggregation.

Keywords: *Ascorbic Acid; Aspirin; Platelet Counts; Platelet Aggregation*

Introduction

Platelets are the blood cells responsible for clotting. They also referred to as thrombocytes. Abnormally low platelet or thrombocytopenia can occur due to reduced production of the cells by the bone marrow, or due to their increased breakdown in the blood stream, spleen or liver.

Platelet to platelet clumping is termed aggregation and this occurs as a result of increasing adhesiveness. In-vitro platelet adhesion strictly signifies the attachment of platelets to a foreign surface; it is subsequent platelet attachment that is called aggregation [1].

Platelet aggregation involves alteration in the normal discoid shape of the platelet to a spherical shape and extrusion of pseudopodia from the surfaces of the platelets. Reversible aggregation occurs after this initial shape changes, but this becomes irreversible after the platelets release their granules, which contain adenosine diphosphate (ADP), serotonin, prostaglandins and thromboxane.

Vitamin C or ascorbic acid is on essential nutrient for humans, a large number of higher primate species, and small number of other mammalian species (notably guinea pigs and bats), few species of birds, and some fish. Ascorbate (an ion of ascorbic acid) is required for range of essential metabolic reactions in all animals and plant. It is widely known that a deficiency in this vitamin causes scurvy in humans. It is also widely used as a food additive. In living organisms, ascorbate is an anti-oxidant, and serves to protect the body against oxidative stress and is a co-factor in several vital enzymatic reactions [2].

Origin of vitamin C

Vitamin C is an important nutrient in the human diet obtained from vegetable and fruits.

Sources of Vitamin C

Many fruits and vegetables including citrus fruits and berries are rich in vitamin C [3].

Plants are generally a good source of vitamin C though the amount in food of plant origin depends on the precise variety of the plant, the soil, the climate in which it grow, the length of time since it was picked, the storage conditions and the method of propagation. Some of the plants are analyzed fresh while others are analyzed dried [4]. In the western human diet, the muscle provides majority of meat consumed and vitamin C is most present in the liver and least present in the muscles, but studies have come to a conclusion that animal products are not a reliable source of vitamin C rather it is abundant in mother's milk while it occurs low amount in raw cow milk [5]. Other sources of vitamin C include Rose hip, broccoli, tomatoes, berries, papaya, lemons, and Brussels sprouts which are also good sources [6]. The vitamin C supplements are another common sources of the vitamin C. Individuals at risk for vitamin C depletion such as smokers, women who take birth control pills and those with unhealthy dietary habits may benefit from a daily vitamin C supplement [7].

Biosynthesis of vitamin C

Vitamin C is synthesized in two modes; the natural and the artificial modes. The natural mode has to do with the production of plant and animal species, while the artificial mode has to do with fermentation process [8].

Vitamin C (ascorbic) synthesis in the natural mode is a glycogenolysis dependent process (i.e. the glucose needed to produce vitamin C is extracted from glycogen). Natural synthesis of vitamin C in mammals is carried out in the liver, while in birds and reptiles the synthesis is carried out in the kidney [9]. In some micro-organisms such as the yeast (*Saccharomyces cerevisiae*) the biosynthesis is carried out via a simple sugar [10].

A vast majority of animals and plants are able to synthesize their own vitamin C through an enzyme driven steps which the L-gulonolactone oxidase enzyme in the human live is the last enzyme in a series of four enzymes driven steps, which converts blood sugar, glucose into ascorbate in the mammalian liver. This liver metabolizes ascorbate and is produced in an unstressed goat (which is a typical example of vitamin C producing animal), for instance, at the rate of about 13,000 mg per day, per 150 pounds of body weight. A mammalian feedback mechanism increases this daily ascorbate production in many folds even under stress [11].

There are also animals that have lost the ability to synthesize vitamin C. An examples of such is the simans, guinea pigs and many major families of bats [11]. They lost the ability to synthesize vitamin because they lack the L-gulonolactone oxidase (GLO) enzymes required in the last step of vitamin C synthesis and also because they have a defective form of the gene for the enzyme, some of these species are able to make do with the lower levels available from this diet by recycling oxidised vitamin C [12].

The artificial mode of vitamin C is produced by two main routes: The reinchstein process, developed in 1930s which was a single pre-fermentation process followed by a purely chemical process [12]. These two major processes were originally developed in China in 1960s, these processes used an additional fermentation to replace part of the later chemical stages. Both processes yield approximately 60% vitamin from the glucose feed [12].

Types of vitamin C

Vitamin C supplements are of different forms, this variety enables an individual make a choice of a particular supplement that is best suited to his/her own need, lifestyle and budget. The vitamin C supplement is therefore, of five different forms.

Ascorbic acid

Ascorbic acid is a pure vitamin C which has a sharp taste. Ascorbic acid is a cheap and cheerful way of taking vitamin C, but it most likely causes gastric irritation in those with sensitive stomach.

Calcium ascorbate

This is an ascorbic acid that is bound to calcium providing a non-acidic form of vitamin C that is gentle on the digestive system. It also provides a supplemental form of calcium.

Magnesium ascorbate

The magnesium ascorbate is also bound to magnesium. It is also a non-acid form of vitamin C that is highly absorbable as well as gentle on the stomach. It is also a supplemental form of magnesium.

Ester C

The ester C is a patented formulation of vitamin C and the calcium ascorbate that makes its easily absorbable and non-acidic, ester C is a top range product and is highly recommended for maximum, and rapid absorption and for taking large doses. It is also the most expensive type of vitamin C.

Table or powder form

This is a powder form of vitamin C that do not contain any tabling agent, it is more rapidly absorbed and does not have to be digested first, it is rather buffered to make it more gentle on the stomach before it is digested. The vitamin C supplements are also available in a variety of forms such as pills, capsules and liquids [13].

Daily requirement of vitamin C

Sixty to seventy-five million gram (60 - 75 mg) of vitamin C per day is the recommended daily dosage for those who feel that they benefit from the vitamin. People can take up to 1000 mg safely, however, what is not absorbed and used by the body will be flushed out in urine, this is not to say that over consumption of the vitamin does not do damage before it is executed, people who take more than 1000 mg are at risk than those who take less [14].

Vitamin C requirement is also dependent on the Basal Metabolic Index (BMI), an individual must identify his/her required dose of the vitamin for quick recovery from illness and when working under stressful condition higher levels of vitamin C is suggested since it is water soluble protein [14].

Aspirin

Aspirin is in a group of drugs called salicylates. It works by reducing substances in the body that cause pain, fever and inflammation. Aspirin is used to treat mild and moderate pains, and also it reduces fever or inflammation. It is sometimes used to treat or prevent heart attacks, strokes, and angina [14].

Aspirin is a more potent inhibitor of both prostaglandin synthesis and platelet aggregation than other salicylic acid derivatives. The differences in activity between aspirin and salicylic acid are thought to be due to the acetyl group on the aspirin molecule. This acetyl group is responsible for the inactivation of cyclo-oxygenase via acetylation [15].

Aspirin, is based on an old natural remedy (willow bark tea). White willow is nature's aspirin. In fact, pharmaceutical aspirin was originally created from a chemical very similar to one found in white willow bark.

It is quite good at controlling blood clots by keeping platelets from clumping for their entire life span, about 10 days. The salicylates in foods are not as powerful and their effects are more temporary.

Although fresh foods are the best choice because they contain all kinds of important nutrients, man can get salicylates from other foods such as apricots, cantaloupes, dates, grapes, oranges, pineapple. These are food with natural salicylates. Processed foods with synthetic salicylates are ice cream, gelatin, pudding, chewing gum, syrup, candy, beverages and baked foods.

Aspirin reduced the risk of being diagnosed with a solid cancer that had already spread by 31%. For patients initially diagnosed with a local cancer, the risk of later metastasis may be linked to its effect on platelets, the clotting bodies and the blood. The role of platelets in promoting cancer spread in mice was reported more than 40 years ago. Many people take a low (25 mg) dose of aspirin each day guard against heart attacks and strokes. Experts advise against this for people who are at no special risk of heart and artery disease because of the possible long-term side effects of aspirin. The drugs, which prevent blood clotting, can increase the likelihood of internal bleeding in the stomach, intestines and brain.

In some people, such as pregnant women at risk of high blood pressure, the benefits of taking aspirin are said to outweigh the risks. However, to date, cancer has not been part of this calculation.

Source: Aspirin or acetyl salicylic acid is a derivative of salicylic acid that is a mild, nonnarcotic analgesic useful in the relief of headache, muscle and joint aches. The drugs works by inhibiting the production of prostaglandin body chemicals that are necessary for blood clotting and which also sensitize nerve endings to pain [16].

Platelets

Platelets, or thrombocytes, are small irregularly shaped a nuclear cell, 2 - 4 um in diameter, which are derived from fragmentation of precursor megakaryocytes. The average life span of a platelets is between 8 and 12 days. Normally, the platelets are spherical or rod shaped and become oval or disc shaped when inactivated [16].

Platelets are formed from bone marrow. The pluripotent stem cell gives rise to the CFU-M. This develops into megakaryocytes the cytoplasm of megakaryocyte form pseudopodium. A portion of pseudopodium is detached to form platelets, which enters circulation [16]. Production of platelets is influenced by colony stimulating factors and thrombopoietin. Colony stimulating factors are secreted by monocytes and T-lymphocytes. Thrombopoietin is a glycoprotein like erythropoietin. It is secreted by liver and kidneys [16].

The average life span of platelets is 10 days. It varies between 8 and 11 days. Platelets are destroyed by tissue macrophage system and spleen. So, splenomegaly (enlargement of spleen) decreases platelet count [16]. When having contact with any rough surface, the platelets are activated and stick to the surface. The factors, which cause adhesiveness, include collagen, thrombin, ADP, thromboxane A2, calcium ions, Von Willebrand factors, P-selectin and vitronectin [16]. Platelet to platelet clumping is termed aggregation, and this occurs as a result of increasing adhesiveness. It is subsequent platelet attachment that is called aggregation (Bloom, 1980), while agglutination is the clumping together of platelets. The agglutination of platelets occurs due to the actions of some platelets agglutinins and platelet activating factors [16].

Activators and inhibitors of platelets

Examples of activators of platelets are collagen which is exposed during damage of endothelium, Von Willebrand factors, thromboxane A2, platelets activating factor, thrombin and adenosine diphosphate (ADP). Other known substances that activate platelets are calcium ions, P-selectin cell adhesion molecule secreted from endothelial cells and convulxin - a purified protein from snake venom [16]. Examples of inhibitors of platelets are nitric oxide, clotting factors II, Ix, x, xi and xii, prostacyclin and nucleotidase which breakdown to ADP [16].

Clotting mechanism

Most of the clotting factors are proteins in the form of enzymes. Normally all the factors are inactive pro enzyme forms. Those pro enzymes must be activated into enzymes to enforce clot formation. It is carried out by series of pro enzyme - enzyme conversions. The first one of the series is converted into active enzymes that activates the second, which activates the third one; this continue till the final active enzyme thrombin is formed [16].

Various reactions involved in the conversion of proenzymes to active enzymes are explained by cascade theory. In general, clotting of blood occur in three stages; formation of prothrombin activator, conversion of prothrombin into thrombin and conversion of fibrinogen into fibrin.

Prothrombin activator is formed into two ways: Extrinsic pathway is the formation of prothrombin activator by the tissue thromboplastin.

Intrinsic pathway is the formation of prothrombin activator initiated by platelets, which are in the blood itself [16].

Tissue thromboplastin (factor III) initiates this pathway, after injury, the damage tissues release tissue thromboplastin. The thromboplastin contains proteins, phospholipids and glycoprotein, which act as proteolytic enzymes. The glycoprotein and phospholipids components of thromboplastin convert factor x into activated factor x, in the presence of factor vii. The activated factor x reacts with factor v and phospholipids components of tissue thromboplastin to form prothrombin activator. The reaction requires the presence of calcium ions [16].

During the injury, the blood vessel is ruptured. The endothelium is damaged and collagen beneath the endothelium is exposed.

When factor xii (Hageman factor) comes in contact with collagen, it is converted into activated factor xii in the presence of kallikrein and high molecular weight (HMW) kininogen.

The activated factor xii converts factor xi into activated factor xiii in the presence of HMW kininogen.

Activated factor xi activates factor ix in the presence of factor iv (calcium).

Activated factor ix activates factor x in the presence of factor vii and calcium.

When platelet comes in contact with collagen of damage blood vessel, it releases phospholipids.

Now the activated factor x (as in the case of extrinsic pathway) reacts with platelet phospholipids and factor v to form prothrombin activator. This needs presence of calcium ions.

Factor v is also activated by positive feedback effect of thrombin [16].

Blood clotting is all about thrombin formation. Once thrombin is formed, it definitely leads to clot formation.

Sequence of events in stage 2

Prothrombin is converted into thrombin by prothrombin activator in the presence of calcium. Once formed thrombin initiates the formation of further molecules of thrombin. The initial formed thrombin activates factor v. Factor v in turn accelerates the formation of both extrinsic and intrinsic prothrombin activator which is converted into thrombin. This effect of thrombin is called positive feedback effect of thrombin [16].

Thrombin converts fibrinogen into activated fibrinogen due to loss of 2 pairs of polypeptide from each fibrinogen molecule. The activated fibrinogen is called fibrin monomers. Fibrin monomer polymerizes with other monomer molecules and formed loosely arranged strands of fibrin.

Later these loss strands are modified into dense and tight fibrin threads by fibrin stabilizing factor (xiii) in the presence of calcium ions. All the thigh fibrin strands are aggregated to form a meshwork [16].

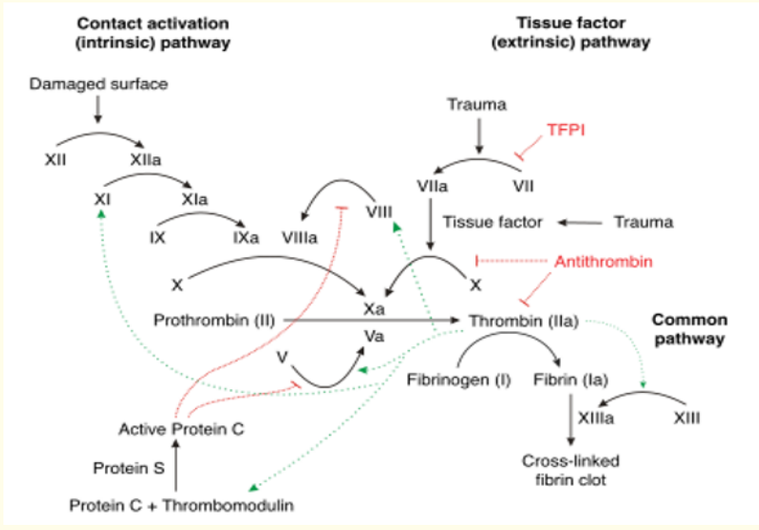

Figure 1*: Diagram showing clotting factors and mechanism.*

Platelet aggregation

Platelet aggregation is the clumping together of platelets induced by various agents (e.g. thrombin) as part of the mechanism leading to thrombus formation [17]. Therefore, platelet aggregation is clumping together of platelets of agents, such as adenosine, diphosphate, thrombin and collagen as part of a mechanism leading to the initiations and formation of a thrombus or haemostatic.

Formation of a haemostatic plug represents one of the earliest responses to vessel wall injury [18]. The development of a primary haemostatic plug represents platelet membrane receptors through which the adhesive macromolecules, von willebrand factor (VWF) and fibrinogen, anchor platelets to the vessel wall and link them each other [18].

There are two receptors pathways: classic and alternative, for the binding of VWF to platelets; the latter induced by thrombin, and adenosine diphosphate (ADP) is shared with fibrinogen [18].

Synthetic peptides, patterned after known binding of adhesive molecules, have been designed to inhibit their interactions with platelet receptors. A secondary haemostatic plug, composed of platelets enmeshed and fibrin, results from the action of thrombin, which is not only essential for formation of fibrin but also for exposure of platelet receptors for adhesive molecules and for 'activation' of factor v and viii [18].

Thrombin generation is greatly enhance through the activity of the prothrombinase complex formed on the surface of platelets, perturbed endothelial cells and leukocytes. A pivotal events is activation of factor x through the intrinsic and extrinsic coagulation pathway. Binding of factor ixa and viia to the vascular endothelial represents a localized mechanism for factor xa generation [18]. Formation of a platelet and fibrin thrombus is controlled by regulatory mechanism: prostacyclin, endogenous heparin-antithrombin iii complex, thrombomodulin-protein C - protein S system, and the fibrinolytic system. The balance of all components: vessel wall, platelets, adhesive and coagulation proteins, regulatory mechanism, determined the effectiveness of the haemostatic plug in maintaining the structural and function integrity of the circulatory system [18].

Factors that inhibit platelet aggregation include snake venom, lebetin peptides, antiplatelet aggregation and thrombocytopenia [19]. Ascorbic acid per day for 10 days reduced platelet adhesiveness and aggregation [20].

Turner (2002) reported that anti-oxidants can help prevent heart disease and cancer, reduce blood pressure and slow down the effects of aging. These naturally occurring components protects the body from harmful, excess free radicals, sweeping them up before they can cause damage. Antioxidants also reduce platelet aggregation [21].

Functions of Platelets

Platelets are rapidly deployed to sites of injury or infection, and potentially modulate inflammatory processes by interacting with leukocytes and by secreting cytokines, chemokines, and other inflammatory mediators [22].

The platelets are responsible for the formation of intrinsic prothrombin activator. This substance is responsible for the onset of blood clotting.

In the blood clot, the blood cells including platelets are entrapped in between the fibrin threads. The cytoplasm of platelets contains the contractile proteins namely actin, myosin and thrombosthenin. The contractile proteins are responsible for clot retraction [16].

Platelets accelerate the processes of homeostasis by three ways:

1. Platelets secrete serotonin which causes the constriction of blood vessels.
2. Due to the adhesive property, the platelet seals the damage in blood vessel like capillaries.
3. By formation of temporary plug, also platelet seals the damage in blood vessels.

The platelet derived growth factor (PDGF) formed in cytoplasm of platelets is useful for the repair of the endothelium and other structure of the ruptured blood vessels.

By the process of agglutination, platelets encircle the foreign bodies and destroy them by phagocytosis [16].

Justification of the study

Vitamin C and Aspirin have been found generally to be more potent in the treatment of scurvy and inflammation respectively but their effects on platelet aggregation and platelet count has not been widely studied. Hence, this study therefore seeks in part to investigate this.

Conflicts of Interest

Apparatus

Glucometer (Accu - Chek advantage, Roche Diagnostics GMBH Germany), Microscope (Olympus, UK), pH meter (JENWAY, Model 3505, PH/Mv/Temperature meter), centrifuge, Neubauer counting chamber, methylated spirit, sample bottles, syringes, burette and conical flasks.

Experimental animals

Albino wistar rats weighing between 120g - 180g were used for the study. the rats were comparable at the start of the experiment. They were obtained and kept in the animal house of the Department Physiology, University of Calabar at normal room temperature 29 ± 2°C. The animals were provided with food and water.

Administration of vitamin C and Aspirin

A stock solution of 50 mg/ml of vitamin C and aspirin was prepared separately. Vitamin C was administered at a dose of 200 mg/kg body weight daily. This was done by administering 0.004 ml of the stock solution per-gram body weight, while aspirin was administered at a dose of 300 mg/kg body weight orally. This was done by administering 0.006 ml of the stock solution per gram body weight for twenty eight (28) days.

Collection of blood sample and harvesting of platelet rich plasma

All animals were placed in an oxygen chamber one after the other with a cotton wool soaked in chloroform. The animal was removed from the oxygen chamber, placed in a supine position as the linear Alba was dissected using a very small sharp pair of scissors. After the dissection, the heart was carefully located and blood was then drawn into the syringes.

Blood sample was drawn from each animal into a plain sample bottle. From here, 0.25 ml each was added into 4.5 ml of buffered EDTA solution and 4.5 ml of buffered EDTA/formation solution respectively. And then the blood was thoroughly mixed with the 4.5 ml of buffered EDTA/formation solution respectively.

These volumes give 1:20 dilution while the formalin fixes any platelet aggregation and cause it to sink to the bottom during spinning [23]. The samples were centrifuged at 100g for 10 minutes using MSE centrifuge to obtain platelet-rich plasma (PRP).

Platelet counting

Platelets in the platelet rich plasma samples were counted using the light optical microscope and a haemocytometer. The counting chamber was the improved Neubauer and the power magnification used was 40x. The haemocytometer was covered with a cover-slip tightly to show the characteristics Newton's ring. The platelet rich plasma was run into the groove on the haemocytometer using the RBC pipette and allowed to stand for ten minutes so that the cells might be evenly distributed.

The counting was done in a total of five large squares (four corner squares and a central square) to minimize standard error. Each large square contained sixteen small squares, thus platelets were counted in eighty small squares. To avoid counting a cell twice, the square and those on, or touching the boundary lines on top and left hand side of the squares were counted while those touching the bottom and right hand side the squares were disregarded. All counting was done within two hours after centrifuging the samples.

Conflicts of Interest

Counting was done in the same way as red blood cell.

Number of cells counted in 80 small squares = n

Area = 80 x 1/400 = 5 mm^2

Depth = 1/10 mm

Volume = Area x depth = 1/400 x 80 = 1/10 = 1/50 mm^3

Volume correction factor (VCF) = $\frac{\text{volume required}}{\text{Volume used}}$

= 1/1/50 = 50 mm^3

Diluting factor (DF) = 20

Total number of platelets = 50 x 20 x n = 1000 n/mm^3

Drugs and Chemicals

The following drugs were used during the experiment; vitamin C and aspirin which were obtained from University of Calabar Teaching Hospital. Also used were the following chemicals; Disodium chloride, ethylene diamine tetra - acetate (ESTE), formalin, citric acid, chloroform and sodium citrate which were obtained from M and B chemical Co, Dagenham, England. Urethane was obtained from sigma chemical Co, Poole, UK.

They were of analytical grade. Vitamin C was dissolved in distilled water while aspirin was dissolved in ethanol and distilled water.

Statistical analysis

The results are presented as mean ± standard error of mean. Analysis of vitamin was used to compare values obtained from the experiment. Significant level was put as $P < 0.05$. Post hoc Bonferroni test was used where F-ratio was significant.

Results

Platelets count in control rat and rats administered with vitamin C and aspirin

The platelets count and platelet aggregation ratio in the control and rats administered with vitamin C and aspirin are shown in table 1. The mean platelets count in the control rats was 544 ± 27.0 x 10^3 cells/mm^3 (mean ± SEM) and the mean platelet aggregation ratio was 1.03 ± 0.03 x 10^3 cells/mm^3 (mean ± SEM).

Platelet Count			
	Control	**Vitamin C**	**Aspirin**
	602.00 609.00 526.00 468.00 515.00	496.00 366.00 364.00 421.00 432.00	400.00 367.00 500.00 450.00 413.00
Mean Std. Dev Std. Error	544 60.3 27.0	416 54.5 24.4	426 50.9 22.8
Platelet Aggregation Ratio			
	Control	**Vitamin C**	**Aspirin**
	0.98 1.04 1.00 1.10	0.60 1.09 1.19 0.96	1.30 0.72 0.66 0.98
Mean Std. Dev Std. Error	1.03 0.0529 0.0265	0.960 0.258 0.129	0.915 0.292 0.146

Table 1: *Platelet Count and Platelet Aggregation Ratio.*

The mean platelet count in rats administered with vitamin C and aspirin were 416 ± 24.4 x 10^3 cells/mm^3 (mean § SEM), 426 ± 22.8 x 10^3 cells/mm^3 cell (mean ± SEM) respectively. Platelet count in rats administered with vitamin C and aspirin was significantly ($p > 0.05$) lower when compared with the control. However, platelets count in rats treated with vitamin C was not significantly different from aspirin administered rats.

The mean platelets aggregation ratio of the control was 1.03 ± 0.02, 0.96 ± 0.12 in rats treated with vitamin C and 0.91 ± 0.12 in rats treated with aspirin. There was no statistical difference between the treated groups when compared with control.

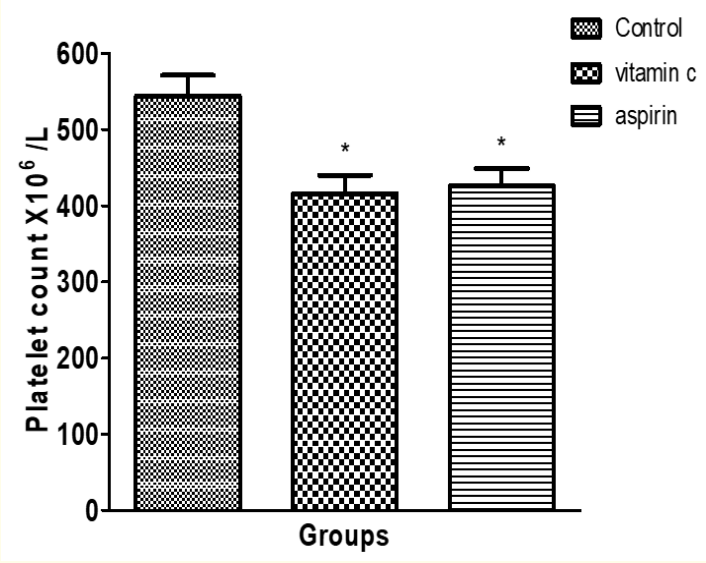

***Figure 2**: A bar chart showing platelet counts in each group.*

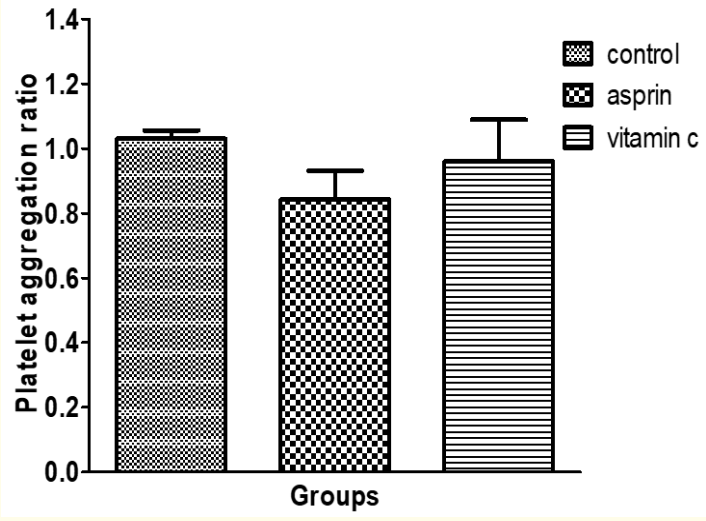

***Figure 3**: A bar chart showing platelet aggregation in each group.*

Discussion

The present study showed no platelet aggregation with aspirin, though the count was decreased. This may be attributed to effect of aspirin on platelet and count.

There is substantial evidence by Lawrence., *et al.* [24] that the use of aspirin is associated with increased urinary excretion and reduced blood levels of folate, particularly in rheumatoid patients and those with arthritis. There is also evidence that supplementation of this drug at high doses can result in hypokalemia [25] and that alcohol absorption can be enhanced by taking aspirin. Aspirin, as an inhibitor of platelet aggregation, may be of benefit in ischemic heart disease. However, aspirin blocks not only platelet aggregation but also synthesis of prostacyclin, a vasodilator and platelet deaggregator. The relative sensitivity of prostaglandin-mediated coronary vasodilation and platelet aggregation to inhibition by aspirin remains uncertain.

Research shows that aspirin reduces the ability of platelets to form clumps or clots, thus, making the platelets less sticky and it work by inhibiting the production of prostaglandins, body chemicals that are necessary for blood clotting [26]. But low-dose aspirin inhibits the enzymes Cox-1, which produces thromboxane A-2, necessary for platelet aggregation [26].

Conclusion

Vitamin C and Aspirin reduce platelet count, but do not significantly affect platelet aggregation. Both drugs may have cardio-protective effect because of the antioxidant activities of vitamin C and anti-platelet activities of aspirin which reduces the risk of clots formation in the blood vessels.

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