e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2025; 16(12); 52-56

Original Research Article

A Cross Sectional Study to Show the Relationship between Von Willebrand Factor and Platelet Count among Different Age Groups in Guwahati City

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Received: 25-09-2025 / Revised: 23-10-2025 / Accepted: 26-11-2025
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Conflict of interest: Nil

Abstract:

Introduction: Cardiovascular disease is one of the major causes of mortality in the world. India has also shown an increase in the incidence of mortality related to cardiovascular disease. The establishment of Von Willebrand factor (vWF) as a new risk factor for cardiovascular disease is one of the initiatives towards its prevention and moreover, it has been widely studied in the western countries.

Aims and Objectives: Distribution of Von Willebrand factor and platelet count and its correlation among different age groups in Guwahati city.

Materials and Methods: This cross sectional study was carried out on 200 Healthy adult individuals over 1 year duration. The estimation of vWF concentration was done in MINIVIDAS machine and Platelet count was enumerated by direct method of platelet estimation. The study protocol was presented to the institutional ethics committee for approval and was approved. Informed consent was obtained from all participants.

Results: the mean vWF levels with standard deviation increased with the increasing age with subjects for the age group of >50 years measured the highest (86.38±17.64IU/dl) as compared to age group 41-50 years (77.57±15.98 IU/dl); age group 31-40 years (77.63±17.38 IU/dl) and age group 21-30 years (70.10±9.75IU/dl). The Platelet count for age group 21-30 years was the highest (1.88±0.25lacs/cu.mm) as compared to the age group of 31-40 years (1.83±0.21 lacs/cu.mm); age group 41-50 years (1.80±0.21 lacs/cu.mm) and the age group of subjects >50 years (1.75±0.27 lacs/cu.mm).

Conclusion: Our study showed a significant statistical difference between vWF and Platelet count among different age groups. As it is an established fact that increased vWF levels are associated with incidence of arterial and venous thromboembolism vWF levels may become an important parameter to be recognized as a new risk factor in the development of the cardiovascular disease.

Keywords: Von willebr and Factor Platelet count.

Introduction

The global burden of disease estimated that 24.8% of all deaths in India in 2010 were attributable to cardiovascular disease which showed an increase incidence to 27.03% in 2015. [1]

This increasing trend suggests that new risk factors must be identified along with formulating prevention strategies for the traditional risk factors. There are several studies that show the relationship between Von Willebrand factor and thrombosis. [2,3] Von Willebrand factor (vWF) is a large, multimeric glycoprotein found in blood plasma, subendothelial connective tissue, and platelet α-granules. vWF performs two essential functions in hemostasis: it acts as a mediator in the adhesion of platelets to subendothelial connective tissue, and it binds blood clotting Factor VIII. [4] When the vascular endothelium gets disrupted the platelets adhere to the exposed subendothelium and at high shear wall rates present in the small blood vessels, this platelet adhesion requires the help of von Willebrand factor synthesized in the vascular endothelial cells.

After endothelial cell disruption, plasma vWF must also come down onto the subendothelium through binding to a yet unidentified subendothelial matrix material.

This alters the plasma vWF so that it can then also bind to a binding site present on the surface membrane of the unstimulated platelet and so link the platelet to the subendothelium.

Megakaryocytes also synthesize vWF which is a normal constituent of the platelet alpha granule and

is secreted along with other granule constituents during platelet activation. [5]

Aim and Objective: To study relationship between Von Willebrand factor and Platelet count among different age groups in Guwahati city

Materials and Method

The study was carried out on 200 healthy individuals of Guwahati who met the inclusion criteria outlined below and were willing to participate in the study.

An informed consent was obtained from each patient prior to participation in the study

Inclusion Criteria:

1. Healthy adult subjects with no history of bleeding disorders and coagulation defects
2. Normotensive and Non-diabetic subjects.
3. Subjects with no history of other major illness.
4. Not on any medication.

Exclusion Criteria:

1. Subjects with known history of bleeding disorders like hemophilia, purpura.
2. Subjects with history of clotting factor deficiencies (I, II, V, VIII, IX, X)
3. Subjects with history of hypertension and diabetes
4. Subjects with recent history of taking NSAID like aspirin
5. Subjects with history of any operations within one year.
6. Pregnancy

Ethical clearance was obtained from institutional ethical committee.

The estimation of vWF concentration was done in MINIVIDAS machine- a compact automated immunoassay system based on the Enzyme Linked Fluorescent Assay (ELFA) principles. It is completely self-contained with an analytical module with 12 test positions / 2 independent 6 – test compartments along with an integrated monitor, keyboard and printer. It also has automated barcode identification. Platelet count was enumerated by Direct method of platelet estimation. [6] The data was presented as mean ± standard deviation**.** Statistical analysis was done using the Analysis of variance (ANOVA) and Pearson coefficient correlation in the SPSS version 20 and Microsoft Excel software. P-value of < 0.05 was considered statistically significant respectively.

Results

A total of 200 subjects were taken for the study after proper history- taking and clinical examination.

Table 1: Sample distribution of Subjects according to different age groups

Age group(years)	No. of subjects	Percentage
21-30	50	25
31-40	59	29.5
41-50	54	27
>50	37	18.5

Table 2: Mean distribution of vWF levels and Platelet count among different age groups

Age groups (years)	vWF(IU/dl) (Mean±std)	Platelet count(lacs/cu.mm) (Mean±std)
21-30	70.10±9.75	1.88±0.25
31-40	77.63±17.38	1.83±0.21
41-50	77.57±15.98	1.80±0.21
>50	86.38±17.64	1.75±0.27

Table 3: Analysis of variance of vWF levels and Platelet count among different age groups

Parameters	21-30 Years	31-40 Years	41-50 Years	>50 Years	p-value
vWF (mean±std in IU/dl)	70.10±9.75	77.63±17.38	77.57±15.98	86.38±17.64	<0.01*
Platelet count (mean±std in lacs/cu.mm)	1.88±0.25	1.83±0.21	1.80±0.21	1.75±0.27	0.1

***p-value<0.05 as significant value**

Table 4: Pearson Correlation of vWF levels and Platelet count among different age groups

Age group(years)	r-value	p-value
21-30	-0.43	<0.01*
31-40	-0.20	0.07
41-50	-0.53	<0.01*
>50	0.24	0.08

***p-value<0.05 as significant value**

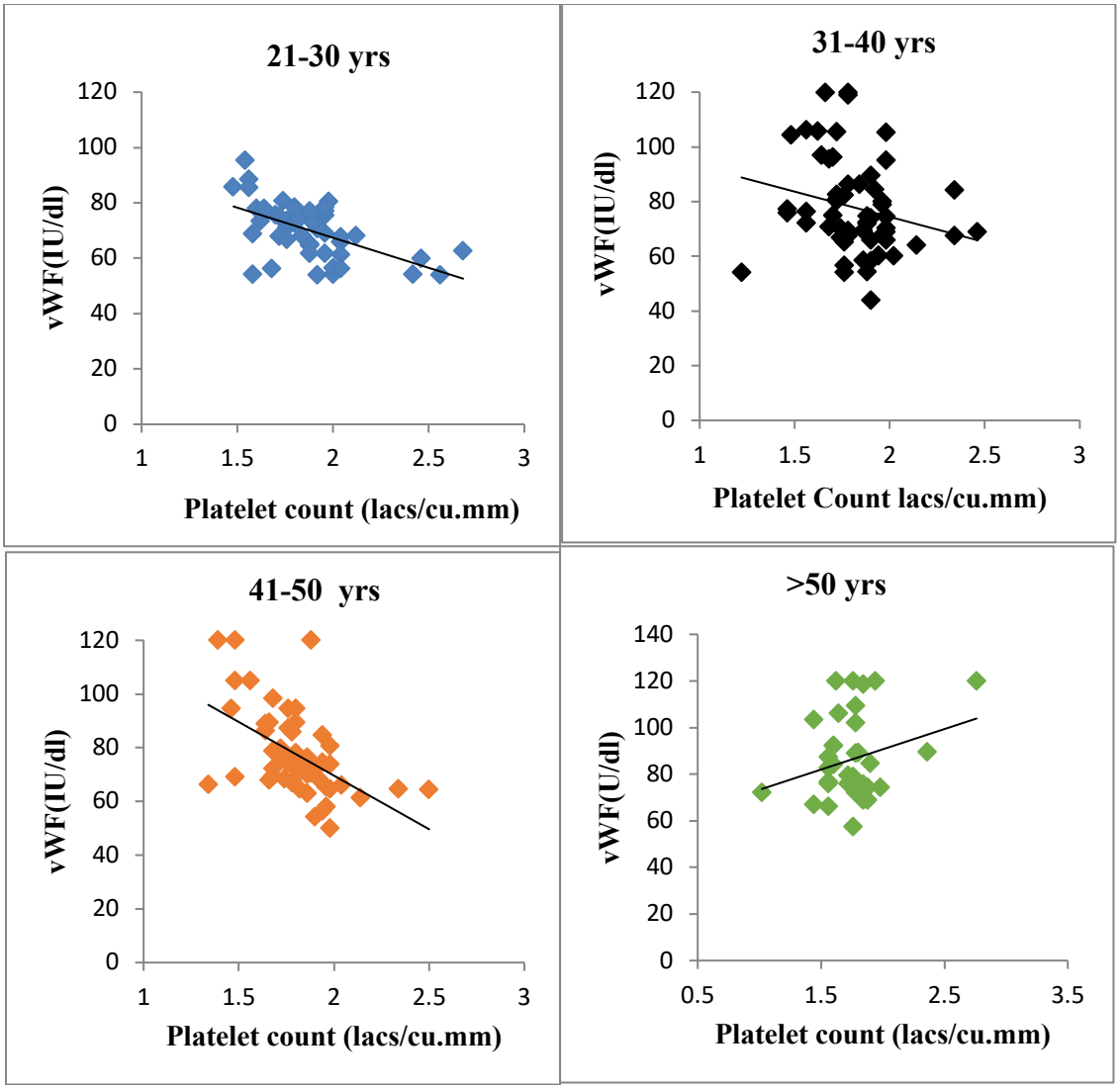

Figure 1: Scatter diagram showing correlation of vWF and Platelet count among different age groups

Interpretation: vWF concentrations increased progressively with age. Individuals above 50 years exhibited the highest mean vWF level (86.38 ± 17.64 IU/dL), while the youngest group (21–30 years) showed the lowest mean (70.10 ± 9.75 IU/dL).

In contrast, platelet counts were highest among the youngest participants (1.88 ± 0.25 lacs/cu.mm) and gradually decreased with age, reaching the lowest mean value in the >50 group (1.75 ± 0.27 lacs/cu.mm).

ANOVA demonstrated a statistically significant difference in vWF levels across age groups ($p < 0.01$). Platelet counts showed a decreasing trend but did not reach statistical significance ($p = 0.1$).

Discussion

The purpose of the study was to establish vWF in association with Platelet count as a newer risk factor in the development of cardiovascular disease and to correlate the relationship of vWF and Platelet count across the progression of age. vWF is a hyper-adhesive glycoprotein that circulates in plasma as heterogenous multimeric complex. [7] The contribution of vWF to platelet adhesion and aggregation is crucial at high and very high shear rates. At very high shear rates above 10.000/sec, aggregation is exclusively mediated by vWF-GpIb-XIV engagement. [8] In the presence of arterial plaque, the stenosed vessels produces a hemodynamic microenvironment that interferes with the physiological shear-flow and elongational flow zones are generated immediately before and after the stenosis. vWF elongates maximally and achieves the highest degree of activation in elongational flow zones, that contributes invariably to post-stenotic thrombus formation. [9] The platelet cell membrane surface contains a coat of glycoproteins that adheres only to the injured areas of the vessel wall, especially to the injured endothelial cells and has more affinity towards any exposed collagen from deep within the vessel wall which is aided by the vWF released in the area of vascular injury. [10] In our study we found that the vWF levels increased significantly with increasing

age (p value< 0.05) and a negative correlation was observed between vWF and Platelet count among all the age groups except the individuals from age group of > 50 years. Similar results were also demonstrated by other studies. [11,12,13,14] This variation might be because the plasma ADAMTS [13] (a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13) levels gets reduced as age progresses and the ratio of vWF: ADAMTS [13] activity increases with age suggesting that increased vWF might be a mediator of prothrombotic state in elderly individuals. [15] vWF after its secretion from the endothelial cells comes in contact with ADAMTS [13]. Formation of vWF strings occurs as soon as there is agonist-stimulated secretion of vWF. [16] Under high shear forces, there is elongation of these strings along the vascular wall that exposes the platelet binding site in the vWF A1 domain and the vWF A2 domain stretches so that the cleavage site for ADAMTS [13] becomes available. [17] On clear visualization of the ADAMTS [13] cleavage site it has been seen that the cleavage occurs selectively at vWF elongated sites. [18]

After the cleavage, vWF is released into the circulation and vWF changes from a stretched into a globular form due to a drop in shear forces, wherein the A2 domain present in the ADAMTS [13] cleavage site becomes inaccessible. [19] The increase in vWF levels has been associated with increased risk of developing arterial and venous thrombosis which was evident in several studies. [2,20-28] The high shear forces produced in cases of stenosis of large arteries due to atherosclerotic plaques increases the vWF secretion by vascular endothelium thereby increasing the platelet adhesion and aggregation activity at the site of damaged arterial walls and leading to thrombus formation. [29] The inverse correlation seen in younger and mid-aged adults also suggests that elevated vWF may be associated with subtle increases in platelet consumption or engagement in hemostatic processes, even in healthy individuals

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