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Study of Effect of Variables on Platelet Stickiness in Subjects of Central India

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ABSTRACT

Aim: *To study the effect of various variables eg. S. Cholesterol, stress, physical activity and Socioeconomic status on platelet stickiness in subjects of Gwalior region in Central India. These tests were done to investigate the physiological norms to evaluate the possible role of various factors affecting the parameter of platelet stickiness as high residual reactivity of platelet moves forward thromboembolic complications.*

Material Methods: *The present study made in 200 apparantly healthy subjects including 160 males and 40 females, belonging to 16-40 years, were selected randomly. After noting careful history, thorough clinical examnation was done and recording of pulse and blood pressure were taken .Stess was assessed by Taylor's Personality scale of manifest anxiety. Serum Cholesterol was estimated by Sackett's method for total cholesterol. Platelet Stickiness was estimated by Mac Donald's Modification of Payling Wright's Technique. Platelet count was done by Rees & Ecker technique.*

Observation & Result: *The value of platelet stickiness was observed to be 10-50% with an average of 21.6%±8.6%. Values were significantly lower in females. No significant correlation was found between age, weight and platelet stickiness. Physical activity significantly lowered platelet stickiness as compared to values in sedentary group. It was also higher in non-vegetarian than in vegetarian group. Also in those consuming saturated fat in diet, it was found to be higher. Smoking also has been shown to increase platelet stickiness, thus making smoking one of the causative and risk factors for thromboembolic diseases.*

In addition, mental stress of examination raised the values of platelet stickiness significantly, thus, proving stress to be one of the contributing factor in worsening the prognosis of the illness and in appearance of clinical symptoms often following emotional stress. However, platelet stickiness values rise with rise in blood pressure figures.

Keywords: *platelet stickinessthromboembolic phenomenon Rees & eckertechnique.*

INTRODUCTION

Recently, attention has been focussed on the point of abnormal platelet behaviour (platelet stickiness) being responsible for the promotion of intravascular thrombosis and it is believed to be the starting factor in the initiation of atherosclerosis.

McDonald & Edgill (1955) found that in ischaemic heart disease platelet stickiness was

markedly and significantly increased. Horlick (1961) found that atherosclerotic subject have higher adhesive platelet index than normal. Since certain relaxation technique and yogic practice can be used beneficially in the treatment of hypertension and IHD, this made us to decide to investigate the relationship of mental and emotional stress of examination of students on platelet stickiness as well in medical students.

Historical background

The discovery of platelet is attributed to Donne who describe the presence of a third element in blood in addition to red and white blood cell which also came to be known as 'globulins of chyle'.

Schultze (1865) observed coalescence of platelets into granular masses and noted their relationship to fibrin, in formation of the early stage of blood coagulation. Osler (1874) demonstrated in experimental animal that platelet remains discrete within circulation, but form granular masses when blood is shed. Hayem (1878) established platelet as a separate cell entity and recognized its part in blood coagulation. Bizzozero noted the adherence of platelets at sites of experimentally induced vessel wall damage. Ebath and Schimmelbusch described the morphological and physical changes occurring in platelets with their adhesion to foreign surfaces and described the changes in the early stages and the process as "**viscous morphosis**".

Structure - Platelets are disc shaped objects, 2-3 μ in diameter. In ultrathin section using glutanald-ehyde fixation they appear elliptical rod like or disc shaped and are some 1.5-4 μ in length and 0.5-2 μ in breadth. The cytoplsmic matrix is enclosed in the cell membrane and contains various organelles. The membrane is a triple layered structure 70-90 A^0 which is composed of two electron dense layers, resembling morphologically the membranes of other cells.

Aynand and Deetjan showed that agents which inhibit clotting also suppress or delay platelet adhesiveness, agglutination and lysis, retard thrombus formation. and may impair hemostasis.

The arrest of hemorrhage at the site of vascular injury is brought basically by the formation of a plug i.e. packed platelet aggregate.

There are 3 phases in the formation of haemostatic plug:

1. Platelets adhere to connective tissue collagen exposed by vascular injury within 2-3 sec after injury.
2. Contact with collagen stimulates platelets to release ADP which in turn causes further platelet aggregation. 5 HT, histamine and thrombin may contribute to this process.
3. Consolidation of the platelet plug involved in the formation of fibrin by the coagulation mechanism.

The primary action of thrombin is to induce fibrin formation but it also promotes platelet aggregation by the release of ADP, thus contributing to the formation of platelet plug. Electron microscopic studies have revealed that the platelets adhere to gaps between endothelial cells in injured vessels . Thrombi may however, also form without detectable primary endothelial lesion.

It has been stressed that the role of platelets in a haemostatic plug is a two stage phenomenon. 1st is the initial reversible aggregation and the next step is the subsequent fusion of aggregate platelets into a more or less amorphous, irreversible blended mass. Blood flow is an important factor in thrombosis. Disturbance of flow may be involved in the aetiology of the thrombi and may have a profound effect on the subsequent growth of a thrombus, once the process has been initiated. ADP was thought to be the final agent responsible for platelet aggregation.

Increased platelet adhesiveness was found to be an almost constant accompaniment of cellular destruction anywhere in the body and to be generally, but not always associated with a rise in the total platelet count.

Helen Payling Wright (1941) described a technique whereby the adhesiveness of platelets to

a glass surface was measured which came to be known as "platelet adhesiveness". Platelet adhesiveness is described as property of platelets to adhere to a foreign substance, aggregation is the property of platelets to adhere to each other and agglutination is the property of platelets to clump in response to an antigen antibody reaction.

Platelet stickiness has been reported to vary from 15 to 35 percent (Ham & Shake 1967, Seymey 1964, Brulges et al 1965, Hajela 1973 and Sharma 1975). A number of potent stimulants of platelet adhesion and aggregation are known.

1. ADP

Macmillan using the nephelometric method of Bonn and Cross described two phases of platelet aggregation in citrated platelet rich plasma in response to ADP.

1. The first phase is probably the direct result of the addition of the aggregating agent.
2. The second response which is irreversible is associated with the release of ADP like activity into the plasma from the platelets.

2. Other factors

Platelet aggregation may be modified by a number of factors besides ADP concentration – like temperature, stirring speed, calcium concentration and various plasma factors.

3. Adrenaline

4. Noradrenaline, and

5. 5 HT – present in platelet have also been shown to produce platelet aggregation.

6. Thrombin - It is well established that thrombin causes platelet aggregation.

7. Collagen– Collagen induced platelet aggregation is irreversible, triggered by the release of ADP by platelets on contact with collagen. It is independent of divalent cation and is not inhibited by adenosine or AMP. Partially degraded collagen retains the ability to cause platelet aggregation. ADP participated in a binding reaction with Ca ion and plasma protein probably Von Willebrand's factor resulting in a complex intraplatelet bridge.

Factors influencing platelet stickiness

(1). Age - McDonald (1962) reported no definite relationship between age and platelet stickiness.

(2). Sex - Pandey (1969) reported that male had higher platelet stickiness as compared to females.

(3). Diet - McDonald & Edgill showed that rich fruit diet, low in fat as well as in proteins could reduce platelet stickiness. In 1964, Gupta and Rai observed no. significant change in platelet count by any of the fats commonly used, but platelet stickiness was increased by ghee and hydrogenated ground nut oil.

Khatwani et al (1971) also observed higher platelet stickiness in non vegetarian group.

(4) Exercise - Finkel and Cumming observed the effect of acute exercise on platelet stickiness at two different temperature and showed that it increased at 25^{o}C as compared to 20^{o}C. Also, strenous exercise was found to be associated with increased aggregation.

(5) Diurnal variation - Increased platelet aggregation occurs towards the end of the day. Recent data suggest that inhibition of PA (platelet aggregation) by drugs reduces the frequency of transient ischaemic attack, ischaemic strokes, death from strokes and non-stroke causes, which are closely related to vascular stenosis due to atherosclerosis and thrombosis (Harrison). The non-steroidal anti-inflammatory drugs, steroids and penicillin G and related antibiotics strongly inhibit platelet adherence to damage vessel wall, ADP induced aggregation and effect of collagen and thrombin on platelets.

(6) Humoral factors - Variation in PCV profoundly affects the result adhesiveness being high when PCV is high and vice versa. It was found that within the range, lower fibrinogen levels are associated with faster platelet aggregation.

AIM

1. To determine the norms of platelet stickiness in healthy subjects n this part of country with its characteristic diet and life style consequent upon. Somewhat

primitive socioeconomic factors existing here. Pace of life is also different in this part of city from the competitive time bound pattern observed in metropolitan cities.

2. To study the effect of various other variables e.g. physical activity and stress besides socioeconomic status on platelet stickiness in subjects of this region.

Ethics - The procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000.

MATERIAL AND METHODS

The present study has been undertaken on 180 apparently healthy subjects including medical students, doctors, laboratory staff between the age of 16-40 years of either sex. Subjects were selected in random manner. Each case was subjected to a clinical examination to exclude the possibility of any disorder.

History regarding socio-economic status, physical activity and dietary habits were taken. Routine physical examination was conducted and measurement of platelet stickiness was done.

These methods were also compared to other methods and observations were found comparable with them. The technique was standardised and personal error was brought down to minimum.

Before undertaking the test the subjects were made to rest at least 15-20 min to exclude physiological effects of physical strain and apprehension on platelet stickiness.

Collection of blood

Under aseptic condition 5 cc of blood from the medical cubital vein was taken by a 5 cc sterilized and siliconised syringe with a base needle of no. 20 G.

All the persons taken for study were healthy, had no evidence of any disease, had a sense of physical, social and mental well being and performed their routine work without difficulty. In view of certain physiological conditions affecting platelet stickiness following additional precautions were taken while selecting normal cases.

Inclusion and exclusion criterias –

Only non-obese persons were selected for study. A person was taken to be obese when he weighted 20% or more than the ideal weight for his height according to table and published by LIC.

Strenous exercise was avoided and they were asked to relax in lying position half on hour before collection of sample.

Persons were categorised into smokers and non-smokers according to their smoking habits.

Only fasting samples were collected.

Blood samples were collected in the morning hours between 7:00 a.m. to 8:30 a.m. so that the effect of diurnal variation was excluded.

Sample was taken by a clean venepuncture of the vein and every care was taken to avoid air bubbles from entering the syringe. Counting was done within 2 hrs of collection.

Persons were divided into sedentary and active workers according to their occupational and working habits.

1. Sedentary

(a) No regular physical exercise - only sedentary or light activity in 24 hrs. Activities like - sitting, gossiping, driving car, paper work and personal care work is only done.

(b) Active exercise in the past - e.g. postman, bus conductor, retired and unemployed persons.

2. (a) Regular physical activity - like walking, shopping, morning exercises, gardening.

(b) Hard manual work - Vigorous physical activity, digging, playing sports, running, swimming etc.

Normally subjects were divided into following age groups:-

Group I	-	31-40 years
II	-	41-50 years
III	-	51-60 years
IV	-	61 years & above

Platelet count was done by **Rees & Ecker's techniques** using R & E reagents while platelet

stickiness was established by **McDonald's modif-ication of Helem, Payling Wrights technique.**

S. cholesterol was estimated by **Sackett's method for total cholesterol.**

In total 600 estimations were done in entire series.

MATERIALS

Necessary Reagents & Equipments

Bottle of Rees & Ecker reagent, filter paper, funnel, Neubauer's counting chamber, red cell pipette, test tube, volumeric flasks, 25 cc, glass bulb, weighing balance, siliconised empty vials, syringes, microscope. Acetic anhydride, sulphuric acid , chloroform , alcohol, ether, colorimeter.

Method of preparing R & E reagent

3.8 gms of Na citrate was weighted and placed in a 100 cc volumetric flask containing 70 cc of distilled water. To this was added 0.1 gm of brilliant cresyl blue after weighting and 0.2 cc of 40% formaldehyde. The total 50% was made 100 cc by adding distilled water and properly mixed. The reagent was filtered and kept. Just before starting the count about 2 cc of reagent was filtered in a clean siliconised vial, to remove crystals likely to be confused with platelets.

Blood was sucked upto 0.5 mark in RBC pipette and Rees & Ecker reagent was taken upto 101 mark. The pipette was gently shaken for about 5 min. Thoroughly cleaned and dry neubauer's chamber was then charged discarding first few drops of solution in the pipette. The haemocytometer was subsequently placed undercover of a petridish for 15 minutes to allow settling. It was then observed under high power to visualised platelets as tiny, highly refractile bodies, about 1/4 to 1/6th the size of RBC. Dust particle can be differentiated by rotating the eye piece as it also moves but not the platelets. Platelets were counted in five small squares of RBC and added together.

Calculation:

Platelet count in cu mm =

$$\frac{\text{Total platelets in 5 RBCsq} \times \text{Dilation factor} \times \text{Volum}}{\text{correction factor}}$$

Estimation of platelet stickiness by McDonald modification of Helen, Paying Wright's technique

2 cc of blood from siliconised vial was transferred in a 25 cc glass bulb which was then slowly rotated for a period of 30 minute at the rate of 4 rotations/ minute. The platelet count was again made after rotation of blood and final count was expressed as percentage of prerotational (initial) count.

Number of platelets lost after rotation by sticking over the glass surface was the measure of platelet stickiness.

$$\text{Platelet stickiness in\%} = \frac{\text{Prerotational} - \text{Postrotational}}{\text{Prerotational count}} \times 100$$

Estimation of Total serum cholesterol

Principle: Serum is added to alcohol ether mixture, which precipitates the proteins and extracts cholesterol. Supernatant fluid obtained on centrifuging is evaporated. The cholesterol is taken up in chloroform and determined calorimetrically by Leibemann Burchard reaction. Stock standard solution of cholesterol was prepared by dissolving 200 mg pure cholesterol in chloroform and make upto 100 cc. Dilute 1 cc of stock standard to 25 cc of chloroform 5 cc of this solution contains 0.4 mg of cholesterol.

Technique: 12 cc of alcohol ether mixture was measured in a clean tube to which 0.2 cc of serum was added and shaken vigorously for about a minute. The tube was allowed to lie horizontally for half an hour and then centrifuged for 10-15 min to get firm deposit.

Supernatant was evaporated to dryness and the residue dissolved in 5cc of chloroform. 5 cc of standard cholesterol solution was taken in another stoppered bottle. To each 2 cc mixture of acetic anhydride and sulphuric acid solution was added and mixed properly and allowed to stand in dark at 25^{o}C for 15 min. Reading was taken at once in colorimeter transmission at 620 mg.

$$\text{Calculation of S. cholesterol} = \frac{\text{Reading of unknown}}{\text{Reading of known}} \times 0.4 \times \frac{100}{0.2}$$

(in mgm/100 cc of blood)

$$= \frac{Un}{Kn} \times 200$$

TABLE 1: Distribution of Cases As Per Serum Cholesterol.

Range	No. of cases	Mean serum cholesterol (mg %)	Mean platelet stickiness in %.
130-140 mg%	3	135	15
141-150 mg%	25	148	22.7
151-160 mg%	48	155	21.5
161-170 mg %	40	166	21.8
171-180 mg%	28	178	23.1
181-190 mg%	23	185	39.4
191-200 mg%	14	198	34.7
201-210 mg%	6	208	33.6
211-220 mg%	6	220	39
221-230 mg%	-	-	-
231-240 mg%	2	240	35
241-250 mg%	5	245	34
251-260 mg%	5	256	40

TABLE 2: Distribution of Cases As Per Platelet Stickiness

RANGE	NO. OF CASES	PLATELET STICKINESS in %
10-15 %	40	**13.3**
16-20 %	50	**17.5**
21-25 %	40	**23.5**
26-30 %	25	**28.9**
31-35 %	20	**32.5**
36-40 %	12	**37.2**
41-45 %	5	**44**
46-50 %	5	**49**
51-55 %	3	**50**

TABLE 3 : Sexwise Distribution of Platelet Stickiness.

SEX	NO.OF CASES	PLATELET ST. IN %
MALES	173	26.2+_9.2
FEMALES	27	20.2+_5.7
Entire series	-	25.5+_9

TABLE 4 : Relationship of Age Group With P.S.

AGE GROUP in years	No. of cases	Platelet stickiness in %
16-20	41	20+_9.5
21-25	40	25 +_12.5
26-30	9	35+_9.4
31-35	6	20+_10.5
36-40	4	35+_20

TABLE 5: Showing Relationship of Physical Activity With P.S.

PHYSICAL ACTIVITY	NO. OF CASES	PLATELET STICKINESS IN %
SEDENTARY GROUP	62	19.4+_6.6
ACTIVE GROUP	43	22.3+_7.3

TABLE 6 : Showing Values Of P.S. With Vegetarian And Non-Vegetarian Diet

TYPE OF DIET	NO. of CASES	PLATELET STICKINESS in %
VEGETARIAN	160	30.2+_5.8
NON-VEGETARIAN	40	44+_9.4

TABLE 7 Showing Relationship of S. Cholesterol And P.S. With Socioeconomic Status

GROUPS	NO. OF CASES	S. CHOLESTEROL	PLATELET STICKINESS in %
Mid Socioeconomic group	**160**	**180+_ 33.3**	**21.7 +_8.3**
Low Socioeconomic group	**20**	**154+_22**	**22.4 +_9**
High Socioeconomic group	**20**	**190+_ 40**	**23.5 +_8**

TABLE 8: Mental Stress And Platelet Stickiness

MENTAL STRESS	NO. OF CASES	RANGE OF PL. ST.	AVERAGE PL. ST. in %
+	36	15-40	25
++	108	20-45	35
+++	56	15-50	40

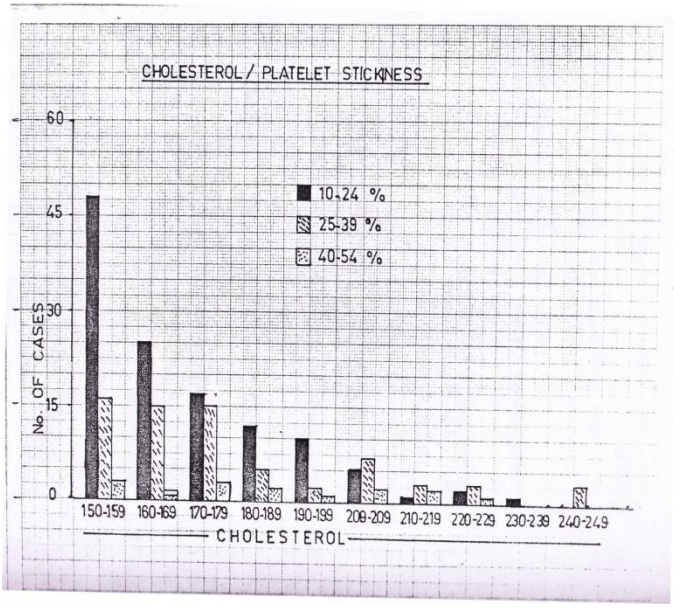

Figure: Showing distribution of cholesterol values with platelet stickiness.

OBSERVATION

From table 1 it is observed that majority of cases i.e. 68% showed values of platelet stickiness ranging from 15%-25%, 14% cases showed 30%-35%, 15% cases showed range from 35-40%. Normal range of present series corresponds favourably with values reported by McDonald & Edgill, Salzman & Brdige. They reported range from 11%-44%, 20%-47% and 18%-32% respectively.

From Table 1, it was also observed that majority of cases i.e. 69% showed a range of S. cholesterol from 151-190 mg% remaining 11% showed a range of 191-220 mg%, 6% showed 230-260 mg% and 14% showed values from 130-150 mg%. The values found in the present series were observed to be lower and thus differ from the observation of Western workers on western subjects. However, they compared favourably with Indian subjects in the studies of Sharma, Dixit, Gopalan etc and Padmavati.

From table 2 it is observed that 20% cases showed values upto 15% and 25% cases, showed values between 16%-20%, 20% cases showed values from 21-25%, 12% cases showed values from 26%-30% and 10% cases showed values from 30%-35% and the remaining 10% from 35%-50%.

Thus it was observed that cases showing higher values of platelet stickiness also showed higher values of S. cholesterol.

Detailed analysis of Table 3 showed that there is no significant difference when the mean values of platelet stickiness is compared for different age groups.

Table 4 shows the relationship of physical activity with platelet stickiness. Out of total cases, 62 belonged to sedentary group while 43 belonged to physically active. Remaining cases could be kept between the two categories but the difference in their platelet stickiness was not found to be significant.

In table 5, mean platelet stickiness was observed to be lower in vegetarians than non-vegetarians, though the difference was not statistically significant. Also, platelet stickiness did not show significant change in subjects consuming saturated or unsaturated fact.

Socioeconomic status also did not prove to influence values of platelet stickiness (Table 7) significantly

No significant rise in platelet stickiness values was observed with increased mental stress. (Table 8).

RESULT

Values of platelet stickiness observed were 10-50% with an average of 21.6%±8.6%. The values were significantly lower in females i.e. average value 16.8% with a range of 12-25%. while, males recorded an average value of 25% with a range of 15-50%±9.6%.

In subject consuming predominantly saturated fat values ranged from 20-25% with a mean of 30%±9.4%, while in subjects consuming predominantly unsaturated fat, values ranged from 10-35% with a mean value of 25%±8.6%, thus showing that platelet stickiness is more in persons consuming saturated fats.

Also platelet stickiness values were higher in those with higher blood pressure values.

No significant relationship was found with age, weights and socioeconomic status.

In sedentary group,

In sedentary group, platelet stickiness was higher ranging from 15-40% with an average value of 25%±8.2% than in active group, where the values ranged from 10-30% with an average of 15%±7.2%. Thus, conforming the role of sedentary life style as one of the contributing factor in the causation of thromboembolic diseases.

Platelet stickiness was also observed to be higher in students manifesting obvious examination mental stress (+++ score on Taylor's scale) with values ranging from 15-50% and average of 30%±10.2%. While in those with lower mental stress in ranged from 18-35% with average of 20%±7.2%.

In smokers, values ranged from 25-40% with a mean of 20%, thus indicating higher values in smokers than in non-smokers.

DISCUSSION

The present study carried out in 200 apparently healthy young individuals of both sexes belonging to different age groups i.e. 16-40 years showed the platelet stick which ranged from 10%-50% with a mean value of 21.6% and S.D. of 8.4%. It is in collaboration with the normal values given by Wintrobes & Guyton i.e. of 10-25%. Malhotra observed platelet stickiness in two groups of Indian population and reported the mean value for south & North Indians as 22% and 21% respectively. Our values compare favourably with the observations of other workers as well.

In our study, 6.3% cases showed a range of stickiness from 31-35% and 6% from 36%-50%. This is in conformity with the value reported by Mathur et al (1976). From table no. 3 it was found out that mean platelet stickiness in males was 26.2%±9.2% while in females its mean was 20.2%±5.7%. Thus, it was reported to be higher in males as compared to females. No definite relationship with age was found through our study as all the cases were below 40 years of age adults.

Variation with physical activity

Subjects were divided into two broad groups - Group I - Sedentary group and Group II - Active group. In group I (31% cases), platelet stickiness was found to vary between 15-40% with an average of 2% while in group II, it ranged from 10-30% with an average of about 15%. Thus, it was clearly higher in sedentary group, thus, declaring sedentary life style to be one of the risk factors heralding potential coronary and cerebral thromboembolic crisis.

Type of fat consumed - In subject, consuming mainly unsaturated fat, values ranged from 10-35%, with a mean of 25%±8.6% while in subjects consuming saturated fat the values ranged from 20-50% with a mean of 30%±9.4%. This showing that platelet stickiness has a definite correlation with the type of fat consumed showing higher values in subjects taking saturated fat and increasing coronary proneness and atherogenesis. Our findings are in consonance with the findings of Khatwani et al (1968.

Type of diet: Values of platelet stickiness ranged from 25-50% with a mean of 34%±9.4% in those taking non-vegetarian diet. While in vegetarians it ranged from 10-30% with a mean of 15%. Our findings confirms with Dixit (1976) who also reported higher values in non vegetations.

Smoking: Platelet stickiness was higher in smokers than in non-smokers. The values in regular smokers were averaged at 30% while it was 20% in non-smokers. Thus, smoking probably has a marked effect of increasing platelet stickiness in individuals.

Socio-economic groups:

Mean value of P.S. was found to be 25% in high socioeconomic group. While it was only 20% in lower socioeconomic group which may be because of high fat content in the diet and less stressful as well as less active life.

Mental stress: As the values were found to be higher, mean 35%, in high mental stress group, as compared to low stress group (having low anxiety score according to Taylors score) with a mean of 25% platelet stickiness rises with high stress in life, giving coronary proneness.

An evaluation for common coagulation protein defects and SPS when applied to patients revealed that sticky platelet syndrome accounted for 21% of otherwise unexplained arterial events, and accounted for 13.2% of otherwise unexplained venous events (DVT with or without pulmonary embolics).

As treatment with heparin or warfarin will not alleviate the thrombotic tendency of SPS, while simple aspirin therapy will almost always correct the defect and protect the individual from second events. Thus, it is particularly important to define the presence of the defect (SPS).

Future perspective

On activation platelets are able to release or express reveal compounds such as ATP, ADP, 5HT, CXCL12, CD34, P-selectin or thromboxanes which may be assessed by using different methodologies such as immunological assay, liquid chromatography or flow cytometry. The major drawback of the application of these assays in clinical practice is the scarcity and high variability of clinical and laboratory data and the absence of clear guidelines for the correct use of such tests. For the study of global or single step of platelet function, new or renewed platelet assays with high sensitivity and specificity are considered necessary especially for routine laboratory analysis. In future, it is desirable that specific, standardised, more rapid and easy tests whose clinical value has been well defined are available.

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