

# PLASMA FIBRINOGEN LEVEL, PLATELET AGGREGATION AND PLATELET COUNT IN STABLE ANGINA

MOMOTAJ BEGUM<sup>1</sup>, MA HAI<sup>2</sup>, ASM LOKMAN HOSSAIN CHOWDHURY<sup>3</sup>, NOORJAHAN BEGUM<sup>4</sup>

## Abstract:

*This was a prospective study. The study was done from July 1997 to June 98. All the cases were selected from the outdoor patients of National Institute of Cardiovascular Diseases (NICVD), Controls were selected from healthy volunteers. The plasma fibrinogen level, platelet aggregation and platelet count were studied on a total number of 35 subjects with age ranged from 40-60 years of both sexes. Of these 20 were normal healthy subjects and 15 were patients with stable angina. Plasma fibrinogen level were normal as like as healthy subjects. Platelet aggregations were increased in some cases but others show normal findings. The platelet count were slightly decreased but it was within normal range. Form this study it may be observed that normal plasma fibrinogen level and increased platelet aggregation with normal platelet count may occur in patients with stable angina. The increased platelet aggregation indicate hypercoagulable states which may aggravate the condition of patients with stable angina and this is the risk factor for the further development of severe ischemia of the heart. So, the routine investigation of plasma fibrinogen level, platelet aggregation and platelet count may be helpful to utilize them as background information both for therapeutic and preventive measures in patients with stable angina.*

## Introduction :

Ischemic heart disease is an important cause of premature death in developed countries.<sup>1</sup> Angina pectoris is the symptom complex caused by transient myocardial ischemia, coronary atheroma is by far the most common cause of angina.<sup>2</sup> Stable angina is characterized left sided or central chest pain that is precipitated by exertion and relieved by rest.<sup>3</sup> Hypercoagulability of blood may be an etiological factor in the occurrence of ischemic heart disease.<sup>4</sup> An increase in plasma fibrinogen level is a risk factor for ischemic heart disease.<sup>5</sup> Platelet adhesiveness may also be increased in patients with ischemic heart disease.<sup>6</sup> Some workers observed a normal level of plasma fibrinogen in patients with stable angina.<sup>7,8</sup> But some workers observed an increase in platelet aggregation in patients with stable angina.

On the other hand, some workers observed a normal platelet aggregation in similar group of patients.<sup>9</sup> Different workers observed a normal platelet count in

patients suffering for stable angina.<sup>10</sup> Therefore, the changes in plasma fibrinogen level, platelet aggregation and platelet count in patients suffering from stable angina are still unequivocal. Stable angina is also common in our country. Early detection and treatment of state of hypercoagulability in this group of patients may also be helpful in minimizing the risk of attack and also its complications. Although a few works have been done in different countries but no such work has yet been carried out in our country. Therefore, the present study has been designed to observe the plasma fibrinogen level, platelet aggregation and platelet count in patients with stable angina and also in healthy subjects.

## Materials and Methods :

In the present study total number of subjects with age ranged from 10-60 years of both sexes were selected. Out of these 15 were patients with stable angina and 20 were healthy volunteers. All the cases were selected from the outdoor patients of National

- 
1. Assistant Professor, Department of Physiology, Dhaka Medical College.
  2. Ex-Chairman, Department of Physiology, IPGM&R and Dean, Faculty of Postgraduate Medical Science, Dhaka University, Dhaka
  3. Assistant Professor, Department of Paediatric Surgery, Dhaka Medical College Hospital, Dhaka, Bangladesh.
  4. Professor and Head of the Department of Physiology, BSMMU, Dhaka.

Institute of Cardiovascular Diseases (NICVD). Control subjects were selected from the healthy volunteers. The patients suffering from renal diseases, supraventricular tachycardia, 2<sup>nd</sup> & 3<sup>rd</sup> degree heart block and diabetes mellitus were excluded from this study. Five millilitres of venous blood were collected by disposable syringe and platelet count was done immediately by one drop of blood. Remaining venous blood transferred in a test tube containing anticoagulants for estimation of plasma fibrinogen level and platelet aggregation. Blood samples were centrifuged within 2 hours of collection and plasma was used for estimation of plasma fibrinogen level & platelet aggregation.

Estimation of plasma fibrinogen level was done by clotting method by using Hemostat Fibrinogen Kit of Human Germany of Humacot<sup>11</sup>. Platelet aggregation was done by Chrono-log optical aggregometer by using ADP<sup>12</sup>. Platelet count was done by Hemocytometer by using improved Neubauer's counting chamber<sup>13</sup>. Statistical analysis of the results were done by using unpaired student's test.

**Results :**

The mean plasma fibrinogen level were 262±5.95, 269±6.11 mg/dl, platelet aggregations were 6±1.21, 69±0.97% and platelet count were 3.28±0.06, 3.1±0.07 Lacs/mL of blood in healthy subjects and inpatients with stable angina.

Table-1, Table-II and Table-III shows that the mean plasma fibrinogen level. Platelet aggregation and platelet counts were not significantly higher (p>0.05) in stable angina than that of healthy control.

**Table-I**

*Mean (±SE) plasma fibrinogen levels in control group and in stable angina.*

| Group   | No. | Plasma fibrinogen level | P-value         |
|---------|-----|-------------------------|-----------------|
| Case    | 15  | 269±6.11                | p>0.05          |
| Control | 20  | 262±5.95                | Not significant |

**Table-II**

*Mean (±SE) plasma aggregation in control group and in stable angina.*

| Group   | No. | Platelet aggregation | P-value         |
|---------|-----|----------------------|-----------------|
| Case    | 15  | 69±0.97              | p>0.05          |
| Control | 20  | 66±1.21              | Not significant |

**Table-III**

*Mean (±SE) platelet count in control group and in stable angina.*

| Group   | No. | Platelet count Lacs/ml of blood | P-value         |
|---------|-----|---------------------------------|-----------------|
| Case    | 15  | 3.13±0.07                       | p>0.05          |
| Control | 20  | 3.28±0.06                       | Not significant |

**Discussion :**

In the present study, the mean plasma fibrinogen level was within normal range in patients with stable angina and it was almost similar to that of healthy subject. Similar findings were also reported by other workers of London. Slight increase of plasma fibrinogen level in stable angina than that healthy group may be due to increase hepatic synthesis of fibrinogen as suggested by different workers<sup>11</sup>. It is difficult to explain the mechanism involved for this slight rise of plasma fibrinogen level in this group of patients. However it has been suggested that slight rise of plasma fibrinogen in stable angina may be due to increase hepatic synthesis of fibrinogen.<sup>12</sup> Although there is slight increase of platelet aggregation in stable angina than healthy group, the mean platelet aggregation was within normal range in this study. Similar finding was also reported by other workers.<sup>8,9</sup> The slight increased platelet aggregation may be due to slight rise of plasma fibrinogen level which acts as a cofactor for increase percentage of platelet aggregation.<sup>13</sup>

The mean platelet count in stable angina was within normal range and it was almost similar to that of healthy subjects. Similar finding has also been reported by other workers.<sup>8</sup> Slight decrease in platelet count in stable angina may be due to increased consumption of platelets into platelet aggregates.<sup>13</sup>

In the present study slight higher levels of plasma fibrinogen in stable angina may be due to increase hepatic synthesis or decrease breakdown of fibrinogen. Again, plasma fibrinogen level was almost towards normal in stable angina due to gradual recovery of the disease. On the other hand, platelet aggregation was also slightly increased in stable angina although it was almost towards normal. This was may be due to slight higher level of plasma fibrinogen which acts as a cofactor for little changes of platelet aggregation in stable angina. Platelet count in this group of patients was almost similar to that of healthy subjects. But in few cases lower platelet count suggest that

increase peripheral utilization of platelet into platelet aggregates. From this study it may be observed that plasma fibrinogen level, platelet aggregation and platelet count were within normal limit but exceed the lower limit of the range. So, this may complicate the condition of patients with stable angina and these value indicate the range. So, this may complicate the condition of patients with stable angina and these value indicate the hypercoagulable state of this group of patients. As, all of these factors aggravate coagulability of blood, routine estimation of plasma fibrinogen level, platelet aggregation and platelet count may be helpful to assess the condition of the patient and thereby minimize the complications of the disease.

#### **Acknowledgement :**

I would like to extend my deep gratitude to Prof. Liakat Ali, M. Phil, PhD. Prof. of Biochemistry and cell Biology coordinator Biomedical research group, BIRDEM, Dhaka for allowing me to use the laboratory facilities for doing platelet aggregation. I also thanks of Md. Imdadul Akhter Khan of Techno worth Associates Limited. Dhaka for allowing me to use the laboratory for estimation of plasma of plasma fibrinogen level and to Md. Shahauddin Lab. Tech of Biochemistry, NICVD for doing platted count

#### **References:**

1. Boon NA, Fox KAA. Disease of cardiovascular system Edwards CRW. Boucher IAD, Haslett eds. Davidson's Principles and practice of Medicine 17<sup>th</sup> edn. ELBS churchill Livingstone. London: 248-54.
2. Christopher H, Edm KC, Nicholas A, Boon NK. Colledge. Davidson's Principles and Practice of Medicine 2001; 19<sup>th</sup> ed. Edinburgh: p 425.
3. Naimi S, Gold Stein K, Prosci S. Studies of Coagulation and fibrolysis of the arterial and venous blood in normal subjects and patients with atherosclerosis. *Circulation* 1903; 27: 904-17.
4. Me Donald L, Edgil M. Coagulability of the blood in is chemic heart disease. *Lancet* 1957; 7: 457-60.
5. Wilhelmssen L, Svardsudd K, Bengtson KK, Larson B, Selin L, Tibbin G. Fibrinogen as a risk factor for stroke and myocardial infarction. *N Eng J Med* 1984; 311:501-5.
6. Steele P, Weily HS, Davies 11, Genlon E. Platelet function studies in coronary artery disease *Circulation* 1973; 68 : 1194-1200.
7. Broadhurst P, Kelleher C, Ilughest L, Imeson ID, Ralery HB. Fibrinogen, factor VII clotting activity and coronary artery disease severity, *Atherosclerosis* 1990; 85: 169-73.
8. Fauchs J, Weinberger I, Roienbeu Z, Joshua H, Almozlino A, Agmon J. Circulating aggregated Platelets in coronary arien disease. *Am J cardiol* 1987; 60: 534-7.
9. Wu KK, Hoak Jc. A new method to the quantitative detertion of platelet aggregates in patients with artemal insufficiently. *Lancet* 1974; 19: 924-6.
10. Fuster V, Verstraete M. thnwhos in cardio-aseular disorders. WB Saunders company 1992; 45: 25-9.
11. Hashini JA, Afroze N, Syed BA. Fibrnogen levels in patients suffering from ischemic heart disease. *J Atheeoceler Res* 1969; 10: 277-81.
12. Pilgeram LO. Relation of Plasma fibrinogen concentration changes to human arteriosclerosis, *Atherosclerosis Research Laboratory*. 1960: 600-4.
13. Mchta J, Mchta P, Pepine CJ. Platelet aggregation in nortic and coronary; venous blood in patients with and without coronary disease. *Circulation* 1978; 58: 881-6.