Parameters Of Blood Coagulation In Patients With Pulmonary Tuberculosis

Z KARTALOGLU, K Cerrahoglu, O Okutan, A Ozturk, R Aydilek

Abstract

It was defined increasing platelet count in pulmonary tuberculosis. In this study, another parameters of blood coagulation were examined consider role of platelets in coagulation. Sixty patients were evaluated. Fifty of 60 patients were pulmonary tuberculosis and 10 patients were bacterial pneumonia as a control group. All patients had not received any drug that affected parameters of blood coagulation.
It was examined platelet count - volume, bleeding time, coagulation time, prothrombine time (PT), partial thromboplastin time, activated partial thromboplastin time.
PT increased significantly in 28 patients (56 %) with pulmonary tuberculosis. Control group had normal PT. We have did not find significantly different among another blood coagulation parameters in the cases.
As a conclusion, we suggested that PT increased in patients with pulmonary tuberculosis such as platelet count. But this finding had clinical value. It was need further studies for understanding these variation in patients with pulmonary tuberculosis.

INTRODUCTION

Tuberculosis is a chronic granulomatous infection caused by Mycobacterium Tuberculosis. Tuberculosis can cause an increase in Erythrocyte Sedimentation Rate (ESR), anemia and lymphopenia.

Studies have also documented an increase in platelet counts in pulmonary and pleural tuberculosis ([1,2]). In patients who underwent surgery, an increase in thrombin formation and coagulopathies have been observed ([3]).

In this study, we aimed to investigate the other coagulation parameters based on our previous study (1) in which we found increased peripheral platelet counts in patients with pulmonary tuberculosis.

MATERIAL AND METHODS

A total of 60 hospitalized patients were enrolled from December 1997 to April 1998 at Camlica Hospital of Chest Diseases. Fifty cases were sputum culture positive for M. tuberculosis with a certain diagnosis of tuberculosis. The remaining 10 cases were non-tuberculous pneumonia patients on grounds of clinical and laboratory findings. They formed the control group.

All patients had a workup of history, physical examination and routine laboratory tests. An automatic hematological counter (Coulter Micro dif 18, USA), performed blood counts. Also, coagulation parameters of bleeding time, clotting time, prothrombin time (PT), partial thromboplastin time (PTT), activated partial thromboplastin time (aPTT) were measured in each patient. Venous blood samples for PT and aPTT were collected from an antecubital vein, anticoagulated with citrate (1ml of 3.8% sodium citrate and 9 ml of venous blood), centrifuged at 1000 g for 20 minutes and studied in 2 hours. A ACL-200 coagulometer (nephelometric centrifugal analyzer, Italy) was used for aPTT and PT. Normal values for PT and aPTT are 10.7 -

13.0 seconds and 30-34 seconds, respectively.

None of our patients was receiving a drug that could affect the coagulation parameters. All tests were carried out before commencement of antibiotic therapy. Patients with cardiac, hematological, endocrinological or any other systemic medical condition were excluded from the study.

Results are displayed as mean (SD. Correlation analysis was performed with Pearson's correlation coefficient. The results of two groups were compare with the Mann-Whitney U test. A p value of less than 0.05 was considered significant.

RESULTS

There were 54 males and 6 female in the study group with a mean age of 24.02 (6.08. Mean age for the tuberculosis and pneumonia subgroups were 22.04 (5.51 and 26.12 (7.41, respectively. The differences between the groups were insignificant (p>0.05).

The results of coagulation parameters are shown table I. Mean PT was significantly increased in the tuberculosis group with respect to the pneumonia group (p=0.0001). Twenty-eight patients in the tuberculosis group (56 %) had an increased PT whereas virtually all measurements in the pneumonia group were within normal range.

Figure 1

Table I: Results of coagulation parameters (Mean (SD)

Parameter	Pulmonary Tb (n=50)	Pneumonia (n=10)	p
Platelet count	318.011± 91.61	260.00 ± 38.75	0.01
Bleeding time	1.20 ± 0.48	1.10 ± 0.20	0.19
Clotting time	6.00 ± 1.22	6.19 ± 0.49	0.77
PT	19.55 ± 4.44	11.57 ± 0.52	0.0001
PTT*	32.28 ± 9.05	30.28 ± 7.54	0.13
APTT**	36.54 ± 8.54	34.12 ± 7.04	0.48

PT: Prothrombin time

*: Partial thromboplastin time

**: Activated partial thromboplastin time

Correlation was sought among coagulation parameters in the tuberculosis group. Correlation analyses were shown in table II.

Figure 2

Table II: Correlation analyses in patients with pulmonary tuberculosis. Other analyses were not significant.

Parameters	r	p
Platelet counts - platelet volume	- 0.3992	0.004
Platelet counts -PT	+ 0.2957	0.03
Platelet counts - bleeding time	- 0.4966	<0.001
Bleeding time - PT	- 0.2851	0.04
Clotting time - PT	- 0.3829	0.006

PT: Prothrombin time

DISCUSSION

Various inflammatory cells, cytokines and mediators are involved in the formation of granulomatous lesions encountered in tuberculosis ([4]). Of variety of cytokines, interleukin-6 (IL-6) has been known to promote platelet production ([5]). Activated T cells release IL-6 in patients with pleural tuberculosis ([6]). Experimental studies have also found an activation of the coagulation pathway in the setting of pleural tuberculosis ([7]).

In tuberculosis patients who undergo surgery, those who had widespread fibro-cavitary disease were observed to have marked increase in thrombin formation and manifest coagulation abnormalities perioperatively (3). Another study found impairment in coagulation parameters in patients with alcoholic pulmonary tuberculosis ([8]).

None of our cases had alcoholism. However, PT was prolonged in patients with pulmonary tuberculosis. This was not clinically significant. Cytokines and mediators emerging from a tuberculosis lesion are considered to prolong the PT. Also, we found the relation between platelet counts and tuberculosis in previous study (1). Our current results on correlation among the other coagulation parameters are considered appropriate on a hematological basis.

Preoperative administration of low dose heparin has been found to be useful in preventing the various coagulopathies in these patients ([9]). Decreased production and increased pulmonary vascular update of antithrombin III causes the hemostatic dysfunction in patients with pulmonary tuberculosis ([10]). Robson et al. ([11]) reported that severe pulmonary tuberculosis was often complicated by deep venous thrombosis. They observed that elevated plasma fibrinogen with impaired fibrinolysis coupled with a decrease in antithrombin III and reactive thrombocytosis

would appear to favor the development of deep venous thrombosis in pulmonary tuberculosis.

In conclusion, alterations take place in coagulation parameters of pulmonary tuberculosis patients. Analyses at the molecular level in conjunction with clinical findings will shed light to the subject.

CORRESPONDENCE TO:

Zafer KARTALOGLU Assoc. Prof. GMMA Camlica Chest Diseases Hospital 81020 Acibadem ISTANBUL / TURKEY Tel: 902163257250 Fax: 902163257257 e-mail:zkartaloglu@hotmail.com

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Author Information

Zafer KARTALOGLU, Assoc. Prof.
Chest Physician, Chest Diseases, GATA Camlica Chest D Hospital

Kamil Cerrahoglu, M.D.
Chest Physician, Chest Diseases, GATA Camlica Chest Disease Hospital

Oguzhan Okutan, M.D.
Chest Physician, Chest Diseases, GATA Camlica Chest Disease Hospital

Ahmet Ozturk, Assoc. Prof.
Physician, Hematology, GATA Haydarpasa Training Hospital

Recep Aydilek, Prof.
Chest Physician, Chest Disease, GATA Camlica Chest Disease Hospital