Serum Prolactin Level in Patients of Ischemic stroke

Sankalp Kumar Tripathi[1], Pallavi Kamble[1], M.G. Muddeshwar[2]

ABSTRACT

Introduction: There is an important role of platelet aggregation in thrombus formation. Prolactin is a newly recognized platelet stimulator. It potentiates ADP-induced platelet aggregation. The aim of our study was to find if there is any relation between ischemic stroke and serum prolactin level.

Material and methods: 45 patients with the diagnosis of acute ischemic stroke and 45 age and sex matched controls with no past history of ischemic vascular disease were included in the study. These patients were admitted with diagnosis of acute ischemic cerebrovascular stroke.

Result: It was found that the patients of ischemic stroke were having higher serum prolactin level as compared to the controls. Mean prolactin level in ischemic stroke patients was 11.8 ng/ml and control group was 6.6 ng/ml (p value < 0.01).

Conclusion: Presence of higher levels of prolactin may be meaningful in etiology of ischemic events.

Keywords: Serum Prolactin, Ischemic stroke

INTRODUCTION

Cerebrovascular disease is one of the most frequently occurring and signifiant neurological disorders among the adult population.[1,2] Inflammation and hypercoagulability are linked to the pathogenesis of atherosclerosis and its clinical manifestations such as coronary or peripheral artery disease and stroke.[3,4] Platelet activation is one of the central mechanisms in arterial thrombogenesis and in the pathophysiology of ischemic stroke.[5-7] Prolactin increases activation and aggregation of platelets.[8] Prolactin directly increases ADP and P-selectin expression which leads to platelet activation.[9,10] Large-size platelets are more reactive to this effect.[11] The aim of our study was to find if there is any relation between ischemic stroke and serum prolactin level.

MATERIAL AND METHODS

45 patients with the diagnosis of acute ischemic stroke and 45 age and sex matched controls with no past history of ischemic vascular disease were included in study. There were 24 males, 21 females in patient group and 23 males, 22 females in control group (p = 0.834) [Table 1]. The mean age of the ischemic stroke patient was 69.8 ± 9.4 years and control was 65.6 ± 7.2 years (p = 0.292) [Table 1]. There were no statistically significant differences in gender and age distribution in case and control groups.

These ischemic stroke patients were admitted in Medicine unit of Govt. Medical College, Nagpur. Diagnosis of acute ischemic cerebrovascular stroke was based on patient's histories, neurological examinations, and brain images (computed tomography and/or magnetic resonance imaging). Patients with the history of diabetes mellitus, hypertension, coronary artery disease, peripheral arterial disease, chronic renal failure, hepatic failure, malignancies, metabolic/endocrine disorders, head trauma, previous intracranial surgical procedures, epilepsy, autoimmune diseases, collagen tissue diseases, alcohol consumption and/or substance abuse were excluded from the study.

Patients with platelet dysfunction and/or abnormal platelet counts, bone marrow diseases and hematological conditions that may lead to suppression of bone marrow were excluded from the study. Patients using various drugs that have effects on bone marrow or on prolactin metabolism such as anti-psychotics, hormone treatments were also not included in the study.

Subjects selected for the control group were people with no history of ischemic cerebrovascular accident, myocardial infarction and peripheral artery disease. Persons with bone marrow dysfunction and prolactin metabolism disorders were excluded from the control group. Patient's venous blood sample was taken within 24 hours of admission, with 12 hours of fasting. Sandwich ELISA method was used for serum prolactin measurement by ELISCON PRL Kit. Results were compared between ischemic stroke patient and control groups.

STATISTICAL ANALYSIS

Quantitative parameters were examined in terms of mean and standard deviation. For statistical analysis SPSS Windows software version 15.0 was used. Student t-test was used to examine the differences in quantitative parameters between the two groups. P value less than 0.05 was considered statistically significant.

RESULT

Serum prolactin level was found significantly increased in patients of ischemic stroke (11.8ng/ml) as compared to controls (6.6 ng/ml) with a p value < 0.01 [Table 2].

DISCUSSION

These findings suggest that prolactin contributes to the pathophysiology of ischemic cerebrovascular event. Prolactin contributes to this process by increasing platelet

	Controls	Cases
Total number	45	45
Number of males	23	24
Number of females	22	21
Age (mean ± SD) in years	65.6 ± 7.2	69.8 ± 9.4

Table-1: Age and Sex distribution of cases and controls

[1]Assistant Professor, [2]Professor and HOD, Department of Biochemistry, Government Medical College, Gondia, Nagpur, Maharashtra, India

Corresponding author: Dr. Sankalp Kumar Tripathi, Plot no. 71 Durga Nagar, Manewada Road, Nagpur-440024, Maharashtra, India

How to cite this article: Sankalp Kumar Tripathi, Pallavi Kamble, M.G. Muddeshwar. Serum prolactin level in patients of ischemic stroke. International Journal of Contemporary Medical Research 2016;3(12):3459-3460.

	Controls (n = 45)	Cases (n =45)	P value
Serum Prolactin level (ng/ml)	6.6 ± 2.1 (mean ± SD)	11.8 ± 6.2 (mean ± SD)	< 0.01

Table-2: Serum Prolactin levels among cases and controls

activation. Wallaschofski et al showed that there is a dose-dependent expression of CD62p with prolactin in patients of cerebrovascular ischemic stroke.[11] Increased prolactin levels were seen in the blood of patients with transient ischemic attack and ischemic stroke.[11] Wallaschofski et al also noted the higher rates of venous thromboembolism in patients with prolactinoma.[9] They attributed these results to increased platelet activation by prolactin via ADP.[8,10] Raaz et al found that ADP stimulated P-selectin expression was increased by prlactin in patients with acute coronary syndrome.[11]

CONCLUSION

This study concludes that hyperprolactinemia might be considered as a risk factor for ischemic stroke which mediates its thrombogenic effect through enhanced platelet reactivity. Further studies are required to assess its role in prognosis of ischemic stroke.

REFERENCES

1. Fisher M, Francis R. Altered coagulation in cerebral ischemia: platelet, thrombin, and plasmin activity. Arch Neurol. 1990;47:1075–9.
2. Gawaz M, Langer H, May AE. Platelets in inflammation and atherogenesis. J Clin Invest. 2005;115:3378-84.
3. Fateh-Moghadam S, Htun P, Tomandl B et al. Hyperre sponsiveness of platelets in ischemic stroke. Thromb Haemost. 2007;97:974-8.
4. Toghi H, Suzuki H, Tamura K, et al. Platelet volume, aggregation, and adenosine triphosphate release in cerebral thrombosis. Stroke. 1991;22:17–22.
5. Biller J, Love BB, Schneck MJ. Ischemic cerebrovas cular disease.In Bradley WJ, Daroff RB, Fenichel GM, Jankovic J, eds. Neurology in Clinical Practice 5th edn. Butterworth Heinemann. Elsevier. 2008:1165-1220.
6. Zeller JA, Tschoepe D, Kessler C. Circulating platelets show increased activation in patients with acute cerebral ischemia. Thromb Haemost. 1999;81:373–7.
7. Marquardt L, RufA, Mansmann U, et al. Course of platelet activation markers after ischemic stroke. Stroke. 2002; 33:2570–4.
8. Wallaschofski H, Donne M, Eigenthaler M, et al. Prolactin as a novel potent cofactor for platelet aggregation. J Clin Endocrinol Metab. 2001;86:5912–9.
9. Wallaschofski H, Kobsar A, Koksch M, et al. Prolactin receptor signaling during platelet activation. Horm Metab Res. 2003;35:228–35.
10. Wallaschofski H, Kobsar A, Sokolova O, et al. Differences in platelet activation by prolactin and leptin. Horm Metab Res. 2004;36:453–7.
11. Raaz D, Wallaschofski H, Stumpf C et al. Increased prolactin in acute coronary syndromes as putative Co-activator of ADP-stimulated P-selectin expression. Horm Metab Res. 2006;38:767-72.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 13-11-2016; **Published online**: 26-12-2016