Pak Heart J

FREQUENCY OF LEUKOCYTOSIS IN NEW ONSET ATRIAL FIBRILLATION

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Date Received: June 04,2016 Date Revised: September 29,2016 Date Accepted: January 24,2017

Contribution

MFM,SUR conceived the idea and planned study. AN, BUMS helped in collection, assembly and interpretation of the data. MFM did critical revision of the article for important intellectual content. All authors contributed significantly to the submitted manuscript.

All authors declare no conflict of interest.

This article may be cited as: Maqbool MF, Rehman SU, Noeman A,Saqib BUM.Frequency of leukocytosis in new onset atrial fibrillation Pak Heart J 2017; 50 (02): 127-30.

ABSTRACT

Objective: To determine the frequency of leukocytosis in new onset atrial fibrillation patients presenting to a tertiary care cardiac centre.

Methodology: A single center, cross sectional survey conducted from May 2014 to April 2015. Patients with new onset atrial fibrillation presenting to cardiology department of Punjab Institute of Cardiology were included in the study after fulfilling inclusion criteria. Blood samples were taken at admission for total leukocyte count. Percentage of patients with new onset atrial fibrillation and leukocytosis was recorded.

Results: A total of 250 patients were included in the study . Mean age was 54.1 ± 4 years with 71.6% of patients were males. Among patients of atrial fibrillation, 25.6% of patients had leukocytosis while remaining 74.4% of patients had normal leukocyte counts.

Conclusion: Frequency of leukocytosis is high in patients with new onset atrial fibrillation. Therefore all patients with new onset atrial fibrillation should be screened for leukocytosis to rule out possible source of infection that can help in proper treatment and prevention of atrial fibrillation.

Key Words: Leukocytosis , New onset atrial fibrillation, Atrial arrthymias, Infection

INTRODUCTION

Almost 3 million Americans and 4.5 million Europeans suffer from paroxysmal or persistent atrial fibrillation, rendering it as the most common cardiac arrthymia.¹ Atrial fibrillation is classified as paroxysmal, persistent, permanent or lone.² Many models are developed to accurately predict the risk of atrial fibrillation.³ Among them, the most widely accepted model based on Framingham Heart Study population predicts individuals ten year risk based on age, sex, treatment of hypertension, body mass index, systolic blood pressure, PR interval and heart failure.⁴

Preclinical and clinical studies showed that inflammation is also a risk factor for development of atrial fibrillation.⁵ Preoperative raised white blood cell count predicted postoperative atrial fibrillation.⁶ There seems some association between raised white cell count and increased incidence of atrial fibrillation which has not been extensively studied. In one study based on Framingham heart population the incidence of atrial fibrillation is significantly associated with leukocytosis. The median leukocyte count was $6.4 \times 10^9 \pm .26 \times 10^9$ during a median 5 year follow up period and 82 patients developed atrial fibrillation.⁵

White blood cell count is a simple and routinely available test, which is considered a marker of systemic inflammation but not used to predict the incidence of atrial fibrillation. It is not even used in predictability risk factor model of atrial fibrillation based on Framingham heart population, which is verified in two cohort studies.⁷ Current study will help us to determine the risk associated with raised white blood cell count in individuals with other risk factors for atrial fibrillation and early treatment of this reversible cause may help to decrease morbidity and mortality related to it.

METHODOLOGY

A single center, cross sectional survey was conducted from May 2014 to April 2015 having patients with new onset atrial fibrillation (diagnosed by ECG) between age of 20 to 60 years, presenting to cardiology department of Punjab Institute of Cardiology (PIC) . Patients were included in the study after obtaining informed consent. Smokers, hypertensive, diabetics, hyperlipidemics and patients with underlying valvular heart disease and connective tissue disorders were excluded from the study. All cases were treated according to hospital protocols. Under aseptic conditions phlebotomy was done to collect 5ml venous blood in a standard EDTA vial for analysis. WBC count was recorded and subsequently divided into normal and abnormal values. Data collected was entered and analyzed in the SPSS version 19. Mean with standard deviation was calculated for guantitative variables like age and WBC Count and frequency and percentages in case of categorical variables like gender and leukocytosis. Data was stratified

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for age groups and gender. Post stratification Chi square test was used to determine the role of chance. A p< 0.05 was considered significant.

RESULTS

A total of 250 patients were included in our sampled population with mean age of 54.16 ± 4.0 years (range from 45 to 59 years) of age. Patients were divided into two groups according to their ages. Group A having 35 patients (14%) with age between 41 to 50 years while remaining 215 patients (86%) were in Group B with age between 50 to 60 years. (Table I)In our study 179 patients (71.6%) were males. About 64 patients (25.6%) had positive results for Leukocytosis while remaining 186 patients (74.4%) had normal counts. To determine the distribution of leukocytosis gender wise we calculated the assocciation between gender and leukocytosis with non-significant (p = 0.096) (Table 2).

The P value for association between different age groups and leukocytosis was also non-significant (p=0.216). (Table 3)

Table 1: Frequency Distribution of Sampled Population By Age Groups (n=250)

| | FREQUENCY (n) | PERCENT % |
|-------------------------|------------------|--------------|
| Group A 41 -50 Years | 35 | 14.0 |
| Group B 51-60 Years | 215 | 86.0 |
| Total | 250 | 100.0 |

Table 2 : Association of Gender and Leukocytosis in Study Population (n=250) $\,$

| | | LEUKOCYTOSIS | | TOTAL |
|--------|------------|--------------|----|-------|
| | | YES | NO | |
| GENDER | MALE (n) | 58 | 13 | 71 |
| | FEMALE (n) | 128 | 51 | 179 |
| TOATAL | | 186 | 64 | 250 |

Using Chi - Square Test, p value = 0.096 (Non-significant)

Table 3: Assocaition of Age Groups and Leukocytosis in Study Population (n=250)

| | | LEUKOCYTOSIS | | TOTAL |
|------------|---------------|--------------|-----|-------|
| | | NO | YES | |
| AGE GROUPS | 51-60 Years | 157 | 58 | 215 |
| | 41 - 50 Years | 29 | 6 | 35 |
| TOTAL | | 186 | 64 | 250 |

Using Pearson Chi - Square, p value = .216 (Non - Significant)

DISCUSSION

Different clinical and pre- clinical studies have shown association of systemic inflammation with atrial fibrillation⁵. Elevated levels of C - reactive protein and leukocytosis were found independent risk factors for development of atrial fibrillation.^{8,9} In results of a recent study based on Framingham heart population patients with increased total leukocyte counts have increased incidence of atrial fibrillation.⁵ Even other cardiovascular diseases are also linked with inflammation including coronary artery disease, diabetes mellitus and hypertension.¹⁰ This is creating opportunities to target inflammation for prevention of atrial fibrillation.¹¹⁻¹³Using the Framingham Heart Study population, predetermined risk factors i.e. age, sex, systolic blood pressure, treatment for hypertension, PR interval, clinically significant cardiac murmur, body mass index, and heart failure, were incorporated into a risk prediction model that predicts an individual's absolute risk over 10 years.⁴

About (25.6%) had leukocytosis while remaining 186 patients (74.4%) did not have leukocytosis in our sampled population. The frequency of leukocytosis is quite high (25.6%) in patients with new onset atrial fibrillation. This result implies that all patients with new onset atrial fibrillation should be screened for leukocytosis and a possible source of infection should be ruled out.

In elderly patients, more focus should be on respiratory tract infection and urinary tract infections. Although in our sample, there was no effect of age on presence of leukocytosis in patients with atrial fibrillation. 35 patients (14%) were between 41 to 50 years of their age while remaining 215 patients (86%) were between 50 to 60 years. When we cross-tabulated age groups with leukocytosis p value resulted non-significantly (p=0.216). About 58 patients who had leukocytosis were between 51 to 60 years of age however rest of 6 were between 41 to 50 years.

About patients with mean age of 54 years ranged from 45 to 59 years were included in the study. Age distribution of included patient shows still younger group with a mean age of 54 years is having new onset atrial fibrillation.

In our study sample (n=250) 179 patients (71.6%) were males while remaining 71 patients (28.4%) were females. More male patients were included in the study, which may be either due to sampling error (non probability sampling) or health seeking behavior of our population, which led to decrease in presentation of female gender to tertiary care hospitals. In our study, to determine the distribution of leukocytosis in males and females, we cross tabulated gender with leukocytosis the results were non-significant (p= 0.096). Of total 13 male patients and 51 female patients showed positive results for leukocytosis.

CONCLUSION

It is concluded that the frequency of leukocytosis is high in patients with new onset atrial fibrillation . All patients presenting with new onset atrial fibrillation should be screened for leukocytosis which can have a better impact on treatment of patients with atrial fibrillation .

REFERENCES

- Kunz JS, Hemann B, Atwood JE, Jackson J, Wu T, Hamm C. Is there a link between gastroesophageal reflux disease and atrial fibrillation? Clin Cardiol2009; 32(10):584-7.
- 2. Naccarelli GV, Varker H, Lin J, Schulman KL. Increasing prevalence of atrial fibrillation and flutter in the United States. Am J Cardiol 2009;104(11):1534-9.
- 3. Wann LS, Curtis AB, January CT. 2011 ACCF/AHA/HRS focused update on the management of patients with atrial fibrillation (Updating the 2006 Guideline): a report of the American College of Cardiology Foundation/ American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2011;57(2):223-42.
- Schnabel RB, Sullivan LM, Levy D, Pencina MJ, Massaro JM, D'Agostino RB, et al. Development of a risk score for atrial fibrillation (Framingham Heart Study): a community-based cohort study. Lancet 2009;373(9665):739-45.
- Rienstria M, Sun JX, Magninani JW, Sinner MF, Lubitz S, Sullivan LM, et al. White blood cell count and risk of incident atrial fibrillation. Am J Cardiol 2012;109 (4):53 3-7.
- Fontes ML, Amar D, Kualk A, Koval K, Zhang H, Shi W, et al. Increased preoperative white blood cell count predict postoperative atrial fibrillation after coronary artery bypass surgery. J Cardiothorac Vasc Anesth 2009;23(4):485-7.
- Schnabel RB, Aspelund T, Li G. Validation of an atrial fibrillation risk algorithm in whites and African Americans. Arch Intern Med 2010;170(21):1909-17.
- Aviles RJ, Martin DO, Apperson-Hansen C, Houghtaling PL, Rautaharju P, Kronmal RA, et al. Inflammation as a risk factor for atrial fibrillation. Circulation 2003;108 (24):3006-10.
- Letsas KL, Weber R, Burkle G, Mihas CC, Minners J, Kalusche D, et al. Pre-ablative predictors of atrial fibrillation recurrence following pulmonary vein isolation: the potential role of inflammation. Europace 2009;11(2):158-63.

- 10. Engelmann MD, Svendsen JH. Inflammation in the genesis and perpetuation of atrial fibrillation. Eur Heart J 2005;26(20):2083-92.
- 11. Boos CJ, Lip GY. The role of inflammation in atrial fibrillation. Int J Clin Pract 2005;59(8):870-2.
- 12. Sjoholm A, Nystrom T. Endothelial inflammation in insulin resistance. Lancet 2005;365(9459):610-2.
- Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation 2002;105(9):1135-43.