

Echocardiographic Predictors of Non Rheumatic Atrial Fibrillation

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Abstract

Objective: to assess echocardiographic structural&functional changes in non rheumatic AF

Patients And Methods: 200 Patients were divided into 3 groups:

Group A: Patients with AF,Group B: Patients have one or more of risk factors (hypertension, IHD, DM, HF, old age) without AF,Group C: normal healthy individuals.

A full medical history and complete physical examination and the following investigation were arranged: FBS, RBS, ECG, CXR and echocardiograph.

Result :The study show there is a significance increase in LAD ,LVEDS ,LVEDD and decrease in fractional shortening in group A &B And group A greater than group B and both groups greater than group C.

Conclusion: Subjects with increase LA size, LVEDS, LVEDD and decrease in fractional shortening were at a significance increase risk for development of AF. This study clarifies the role of cardiac structural& functional characteristics' in relation to susceptibility to AF .so the best method for reducing the adverse complication of AF through prevention of development of AF.

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Introduction

Atrial Fibrillation (AF) :is the most common chronic arrhythmia associated with an increased risk for cardiovascular disease morbidity and mortality in general population[1]. Its characterized by totally disorganized atrial activation without effective atrial contraction .the ventricular response is irregularly irregular usually between 100-160 beats/min. in untreated patients with normal AV conduction[2].

Prevalence and incidence

The Prevalence of AF double with each advancing decade of age from 0.5% at age 50-59 y to almost 9% at age 80-89 y[3].

It's higher in male than female at all age groups on all biennial examination [4].

Persons with loud heart murmur .HF, MI had substantially higher prevalence of AF [3].

The incidence of AF also doubles with each successive age decade beyond 50 y.

So that almost 10% of persons who reach 80y of age can expect to acquire this serious cardiac rhythm disturbance.

Persons with HT, DM, HF, valvular heart disease and IHD had higher incidence of AF

Risk of stroke associated with AF increased steeply from 1.5% at age 50-59 y to 22.5% at age 80-89 y[3].

Causes of AF

1-AF may occur in apparently normal heart (lone AF)[8-9]

2-underlying heart diseases include:

Valvular heart disease, IHD, HF, myocarditis, congenital heart disease,

cardiomyopathy) ,pericardites ,hypertensive heart disease[5,8,10]

3-non cardiac causes: thyrotoxicosis ,pheochromocytoma ,electrolyte disturbance, systemic infection ,chronic lung disease ,pulmonary hypertension , pulmonary embolism ,drugs ,stress condition ,exercise .[3,5,11]

4-normal aging changes in atrium [8]

Clinical features and diagnoses of AF

AF is either asymptomatic particularly in elderly or symptomatic usually presented with palpitation, dizziness or with complication of AF (stroke ,pulmonary edema ,angina).

Physical finding including variation in intensity of first heart sound, absence of a-wave in jugular venous pulse, irregular heart beats. ECG show irregular ventricular rate usually between 100-160 b\min, absence p wave (2-10)

Treatment

A-in acute hemodynamically decompensation D.C cardio version should be employ between 100-200 joules .in the absence of decompensation should be treated with digitalis ,B.blockers ,verapamil ,diltiazem ,type 1 and 2 anti arrhythmic agent Anticoagulation is indicated prior DC. Or in high risk for emboli .in refractory cases need radio frequency ablation and pace maker

implantation.[2-5] ,ACEI in LV dysfunction or after MI[12]

B-treatment of primary cause.

C-treatment of complication.[5]

Patient And Methods

A prospective study was conducted in alyarmouk teaching hospital during the period between august 1998-august 1999 200 patient were included in this study .all patients were assessed according to questionnaire from which information were obtained about age, sex ,D.M.,HT, IHD ,HF .and duration and treatment of these illnesses .then complete physical examination and fallowing investigation were arranged : FBS ,RBS ,ECG ,CXR ,and echocardiography

Results

The patients were divided in to 3 groups according to the history, physical examination and investigation:

Group A: 75 patients those with non rheumatic AF.

Group B: 75 patients those have one or more risk of AF but have no AF

Group c: 50 normal persons

Table 1: The Number and patient with AF and associated diseases.

	Group-A	Group-B	Group-C
No.of patients	75	75	50
Age (mean)	60±6	56±6	60±8
No.of patients with HT	41	49	
No.of patients with DM	14	15	
No.of patients with IHD	59	61	
No.of patients with CHF	57	27	

Table 2: Echocardiographic measurements to 3 groups .

	Group-A	Group-B	Group-C
LAD(cm)	4.5±0.8	3.5±0.6	3±0.5
LEVSD	4.8±1.2	3.9±0.9	3.05±0.5
LEVDD	5.9±1.2	5.1±0.9	4.5±0.6
Fractional shortening	19.5±7.2	23±7	31±6.5
IVS	1.03±0.3	1±0.3	1±0.3
PWT	1±0.3	1±0.2	1±0.2

Table 3: Echocardiographic comparison between male and female.

	male	female	p-value
LAD(cm)	4.7±0.7	4.2±0.7	0.01
LEVSD	5	4.2±1	0.0006
LEVDD	6.5±1	5.3±1	0.00004
Fractional shortening	17.5±5.5	22±7.8	0.005
IVS	0.9±0.3	1.15±0.5	0.006
PWT	97±0.3	1.07	0.11

Discussion

200 patients divided in to 3groups 100 males and 100 females the mean age (62±2), hypertensive patients are 45% (90 patients), DM 13%(26 patients), IHD 60%(120 patients), and CHF 42%(84 patients)

Left atrial enlargement >4.5±0.8cm is detected in those patients with AF (group A) and its greater than group B were LAD is(3.5±0.5). And group A and B is more normal control sample (LAD 3±0.5). So AF was secondary to LA enlargement.

In a study by Volegman et al[15] suggest that enlargement of LAD increase incidence of AF and when LAD greater than 6.5cm had recurrence AF compared with patient maintaining normal sinus rhythm.

LAD play a role in treatment of AF and LAD is frequently enlarged in patient with AF. And in patients whom the LAD exceed 4.5 cm it not be possible to correct AF to sinus rhythm despite therapy (Marke et al)[5].

Also our study show increase in LVESD, LVEDD, decrease in LV fractional shortening in patient with AF (group A)

greater than group B and group B greater than normal(group C)

This is compatible with a study done by Sonyam et al [13] which suggest that enlargement LAD increase LVESD, increase LVEDD, increase LV wall thickness, decrease LV fractional shortening were at increase risk for the development of non rheumatic AF.

More recently echocardiographic determination of LA size to examine the association between the LA enlargement and presence of AF; Henry et al[16] studied 265 subjects with either isolated mitral or aortic valve disease or asymmetric septal hypertrophy and found that AF was common when the LAD exceed 4.0 cm but rare when it was <4cm, by demonstrating stepwise increase in LA size from sinus rhythm to transient AF to chronic AF, these investigation suggested that AF was secondary to LA enlargement.

While more recent prospective study with limited follow up with pre existing LAD > 4.5cm did not demonstrate an increase susceptibility to AF. A recent follow up study



have provided evident that LA size increases after the onset of AF.

There is a significant difference between male and female echocardiography indices (table 3) were LAD ,LVESD ,LVEDD ,greater in male while fractional shortening is less in male .this compatible with other study proved the incidence of AF is higher in male than female at all age groups(3)

Conclusion

In this study subjects with increased LA size increase LVESD , increase LVEDD, and decrease LV fractional shortening were at significantly increase risk for development of non rheumatic AF.

This study clarifies the role of several cardiac structural and functional characteristics in relation to susceptibility to AF. Precursors of AF may enhance our understanding of mechanism responsible for this dysarrhythmia .so the best method for reducing the adverse complication associated with AF is through prevention of development of AF.

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