ORIGINAL RESEARCH PAPER

Cardiology

KEY WORDS:

PREDICTION OF NEW-ONSET ATRIAL FIBRILLATION BY ATRIAL TISSUE DOPPLER IMAGING - A METHOD TO MEASURE TOTAL ATRIAL CONDUCTION TIME

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BACKGROUND

Atrial fibrillation constitutes the commonest cardiac rhythm disorder and is the major determinant of Outcome in valvular, myocardial, and ischemic heart disorders. There is no simple tool available to predict the onset this arrhythmia.

Electrophysiological parameters are too complex and can not be obtained in bedside.

Tissue Doppler analysis of atrial muscle gives us an opportunity to measure the critical total atrial conduction time non invasively.

OBJECTIVE

To predict new-onset Atrial Fibrillation (AF) by measuring the total atrial conduction time by atrial tissue Doppler imaging.may affect mitral anulus velocities. A localized disease process, such as lateral myocardial infarction, can result in mitral anulus velocities being lower at the lateral anulus than at the septal anulus.

PATIENTS AND METHODS INCLUSION CRITERIA

159 Patients who came to our cardiology out patient department between April 2008 and March 2010 were randomly selected and included in the study.

EXCLUSION CRITERIA

Patients with history of AF, atrial flutter, atrial tachycardia, age <18 years, previous pacemaker implantation or an implantable cardioverter-defibrillator were excluded from the study.

The following parameters were studied

- Age
- Sex
- Occupation
- Socioeconomic status
- Diabetes
- Hypertension
- Coronary artery disease
- Rheumatic heart disease
- Underlying disease like chronic obstructive pulmonary disease (COPD), thromboembolic events, heart failure, myopathies etc were studied.
- Medication use (beta blockers, verapamil, diuretics, anti arrhythmic etc)
- ECG

1. Heart rate(bpm)
2. P-wave duration
3. PR interval
4. QRS duration (ms))

- Tissue Doppler imaging

1. PA-TDI interval - (The time from the initiation of the P wave on the ECG (lead II) to the peak of A' wave on the lateral left atrial tissue Doppler tracing obtained in apical 4-chamber view) was measured
- 2.Maximal A' –wave velocity (cm/s)(lateral left atrial tissue Doppler tracing)

- Dimensions (Echo cardiography)

1. Aorta diameter(mm)
2. Left atrial dimension(mm)
3. Left ventricular end diastolic diameter(mm)
4. Left ventricular end systolic diameter(mm)
5. Inter ventricular septum width (mm)
6. Posterior wall width(mm)
7. Left ventricular mass(gm)
8. End diastolic volume(ml)
9. End systolic volume(ml)
10. Caval vein(mm)

- Left ventricular function

1. Left ventricular ejection fraction (%)
2. Hypokinesia

- Mitral valve Doppler assessments

1. Maximal E – wave velocity (m/s)
2. E – wave deceleration time (ms)
3. Maximal A – wave velocity (m/s)
4. E/A ratio

- Valvular disorders were studied.

RESULTS

- The average age of the patients was 52.2 yrs,
- Male to female ratio was 1.5:1,
- Most of them belonged to low socioeconomic status,
- 20 patients (12 %) developed new-onset AF.
- These patients had a longer PA-TDI interval than patients who remained in sinus rhythm (176 ms vs. 148 ms, $p < 0.05$).

TABLE -2 PATIENT CHARACTERISTICS WERE SHOWN BELOW

SI NO	CHARACTERISTICS	ALL PATIENTS (N=159)	NO AF DURING FOLLOW-UP	AF DURING FOLLOW-UP	P VALUE
1	HYPERTENSION	95(60%)	(N=139) 87(63%)	(N=20) 8(40%)	0.0857
2	CAD	16(10%)	15(11%)	1(5%)	0.6954
3	DIABETES	20(13%)	18(13%)	2(10%)	1.0
4	THROMBOEMBOLIC EVENTS	8(5%)	7(5%)	1(5%)	1.0
5	RHD	70(44%)	60(43%)	10(50%)	0.6335
6	HEART FAILURE	14(8%)	10(7%)	4(20%)	0.0795
7	COPD	60(38%)	56(37%)	4(20%)	0.0899
8	MYOPATHIES	10(6%)	8(6%)	2(10%)	0.6156

1. The above table shows patient characteristics with predominantly hypertension, rheumatic heart disease, and chronic obstructive pulmonary disese(COPD).
2. Among the patients with myopathies (total no of patients-10), restrictive cardiomyopathy were-2, hypertropic obstructive cardiomyopathy-2, dilated cardiomyopathy were 6.
3. Some of the patients who were hypertensive also had diabetes and (COPD).Among them some had heart

failure and thromboembolic events which is explained in the table above.

4. Age, diabetes, hypertension, Coronary artery disease (CAD), thromboembolic events, Rheumatic heart disease(RHD), Heart failure, Chronic obstructive pulmonary disease (COPD), myopathies and medication use were comparable between the groups which developed & not developed atrial fibrillation.
5. Maximal A' –wave velocity (cm/s) (lateral left atrial tissue Doppler tracing) were Rheumatic heart disease (total no of patients-70),mean maximal A'-wave velocity is 8.4 cm/s, restrictive cardiomyopathy(total no of patients -2)-7.1 cm/s, systemic hypertension(total no of patients -95) – 8.9 cm/s, hypertropic obstructive cardiomyopathy(total no of patients -2)-7.5 cm/s, dilated cardiomyopathy (total no of patients-6)-7.4 cm/s, patients who had systemic thromboembolism(total no of patients-8)-9.3 cm/s, chronic obstructive pulmonary disease(total no of patients-60) – 9.5 cm/s.
6. Left atrial diameter(mean) measured by 2d echo were, rheumatic heart disease- 4.46cm, restrictive cardiomyopathy-5.9 cm, hypertropic obstructive cardiomyopathy- 3.25cm, systemic hypertension-3.8cm, patients who had systemic thromboembolism-4.6 cm, dilated cardiomyopathy-5.25 cm.
7. Left ventricular ejection fraction (mean %) measured (m-mode) were rheumatic heart disease-55.1%, restrictive cardiomyopathy-61.5%, hypertropic obstructive cardiomyopathy-65%, dilated cardiomyopathy-31.8%, systemic hypertension- 57.9%, patients who had heart failure-48%.
8. Patients who were hypertensive had mean left ventricular posterior wall thickness – 10.4cms, and mean interventricular septal thickness-10.71cms, hypertropic obstructive cardiomyopathy mean left ventricular posterior wall thickness-11.5cm, mean interventricular septal thickness- 26.5cms.
9. Mitral valve Doppler measurements, Maximal E – wave velocity (m/s) and Maximal A – wave velocity (m/s) (mean values) were rheumatic heart disease- 0.55&0.38 m/s, restrictive cardiomyopathy -1.5&0.5 m/s, hypertropic obstructive cardiomyopathy- 0.35& 0.95 m/s, dilated cardiomyopathy- 1.4&0.73 m/s, systemic hypertension-0.68&0.74 m/s, and patients who had coronary heart disease – 0.63&0.72m/s respectively.
10. E/A ratio(mean) were rheumatic heart disease -1.3, restrictive cardiomyopathy- 3, hypertropic obstructive cardiomyopathy -0.7, dilated cardiomyopathy-2.03, systemic hypertension-0.91, and patients who had coronary heart disease – 1.01.
11. New onset AF was more common in patients with rheumatic heart disease (50%) (Especially with mitral regurgitation), chronic obstructive pulmonary disease (20%), restrictive cardiomyopathy (10%) and patients with previous history of Heart failure (20%). These findings are explained by the table below.

TABLE 3 PATIENTS WHO DEVELOPED ATRIAL FIBRILLATION (N=20)

SI NO	DISEASE	NO (%)
1	RHD	10 (50%)
2	COPD	4 (20%)
3	RESTRICTIVE CARDIOMYOPATHY	2 (10%)
4	PRIOR CHF	4 (20%)

DISCUSSION

The hemodynamic function of the left atrium (LA) [3] primarily modulates the left ventricular (LV) filling through its three components: a reservoir component during ventricular systole, a conduit component during early ventricular diastole, and a booster pump component during late ventricular diastole. Left atrial appendage (LAA) is a highly contractile structure with a pattern of contractions totally different from that of the LA main body. It is more compliant and therefore plays an important role in the LA reservoir function, especially during increases in the LA pressure or volume Atrial fibrillation (AF) is the most common cardiac arrhythmia associated with an at least two-fold increase in morbidity and mortality, and occurs in 0.4% of the general population, increasing to 5% in those >65 years old. With the loss of atrial booster pump function, the LA–LV pressure gradient during early LV filling is enhanced by elevation of the LA pressure to maintain stroke volume. Thus, a reduction in both LA compliance and volume has been observed with the onset of AF that further decreases cardiac function and increases the risk of thrombo-embolism.

In our study, new onset AF was more common in patients with rheumatic heart disease (50%) (especially with mitral regurgitation), chronic obstructive pulmonary disease (20%), restrictive cardiomyopathy (10%), and patients with previous history of heart failure (20%).

Atrial electrical activation[2], as assessed by the PA-start interval, began at the RA and followed through the IAS, to the inferior and posterior LA walls. This is the known normal electrical activation process, as obtained by invasive electrophysiology techniques.

Left atrial enlargement, increased left ventricular wall thickness[1], left ventricular diastolic dysfunction and a reduced left ventricular fractional shortening are predictive echocardiographic parameters for new-onset AF. There is no simple tool available to predict the onset this arrhythmia. Electrophysiological parameters are too complex and can not be obtained in bedside.

In our study, TDI was done by placing a 2mm sample volume PWD at the lateral wall of the left atrium, just below the mitral annulus.

The main new findings of this study[2] are as follows:

(1) Some TVE-derived variables indirectly reflect the atrial electrical activation that follows the known activation process as revealed by invasive electrophysiology.
(2) The regional and global atrial mechanical function is explained by an upward movement of the atrial walls at the region near the A-V ring with a continuous reduction of this movement towards the upper levels of atrial walls.
(3) The atrial mechanical function is quite similar in all LA walls; however, all indices of mechanical function were higher in the RA than in the LA. The difference in the atrial velocities at different sites was attributed to an atrial free-wall motion higher than that of the bounded IAS. Furthermore, the larger pectinate muscle in the RA can perhaps generate a more pronounced and sustained longitudinal movement in the relatively low pressure system of the right ventricle.
(4) The present study showed that all atrial walls actively moved upwards from the region of the A-V ring at late diastole, with a reduction of this movement towards the upper parts, thus empting the atria and contributing towards the last part of filling of the LV. This longitudinal movement of the atrial walls is probably related to the longitudinal endocardial muscular fibers along the walls of the LA and RA. The more pronounced

longitudinal movement in the RA may be explained in part by the larger pectinate muscles in the RA, but also by the lower pressures in the heart's right side. To what extent circumferential contraction of the atrial muscle fibers might contribute to atrial mechanical function is unknown. Anatomically, the large amount of circumferential muscle fibers present in the vestibules of the RA and LA might imply some kind of circumferential or radial contraction of the atria.

Other uses of measurement of total atrial conduction time:

The assessment of pure mechanical atrial function by means

of atrial wall movements may give more concrete clues about the recovery process of atrial electromechanical function after conversion for atrial fibrillation and flutter and can give additional patho-physiological insights on the thrombo-embolic process that occur in some of those patients. TVE-derived parameters may also give additional patho-physiological information on the process of atrial electromechanical remodeling that occurs in patients with sustained supra-ventricular tachy-arrhythmias

Ischemic heart disease[3]:

Atrial contractile dysfunction appears early in ischemic heart disease (IHD), irrespective of previous myocardial infarction, co-existing systolic dysfunction, or severity of diastolic dysfunction. Yu et al, found that the VA' measured at mid-level of the IAS and the lateral LA in the apical four chamber view were drastically reduced in 118 patients with IHD when compared with 100 normal subjects. A poor LV-ejection fraction and the presence of a restrictive LV filling pattern were the most important determinants of LA contractile dysfunction in IHD.

In our study, 10% of our patients had Coronary artery disease, and a significant number developed AF during follow-up.

Advanced heart failure and cardiac resynchronization therapy[3]:

In this study, 8% of the study patients were included as they had heart failure. 20% of those patients developed AF during follow-up. Even these patients might improve with CRT therapy.

Mitral stenosis and mitral regurgitation[3]:

Due to inflow obstruction, the atrial booster pump contributes less to LV filling in mitral stenosis even during sinus rhythm, despite a proportional increase, with increasing severity, in the LA preload. The impaired atrial reservoir and pump function are associated with a reduction in LA compliance and intrinsic myocardial contractility. In addition, the LAA is inevitably affected in mitral stenosis with reduced contribution to overall LA emptying fraction and increased risk of thrombus formation. In a study by Wang et al, published in 2005, the LSW and LDW tissue velocities recorded during sinus rhythm at the lateral wall or at the tip of the LAA were markedly reduced in subjects with mitral stenosis when compared with normal subjects. Systolic velocity was further decreased in patients with spontaneous echo contrast (SEC) in the LAA than those without. In this present study, the bulk of the patients were suffering from RHD (44%) and the incidence of atrial fibrillation during follow-up was also high in those patients, in concordance with the previous studies (50% developed new onset AF).

CONCLUSIONS

- Tissue Doppler imaging can be used to measure total atrial conduction time.
- A prolonged PA-TDI interval may predict the development of new-onset AF.