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Original article

Left atrial myxoma—influence of tumour size on electrocardiographic findings

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K E Y W O R D S

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A B S T R A C T

Objective: The data of 51 patients (33 females) who underwent excision of left atrial (LA) myxoma were retrospectively reviewed for correlation of tumour size and electrocardiographic (ECG) findings.
Methods and results: Mean age was 39.1 ± 15 years (range 9–53 years). The LA enlargement (LAE) on ECG was defined by standard criteria. The LAE in ECG in these patients did not correlate with echocardiographic LA dimensions or with the degree of left ventricular (LV) inflow obstruction. But it was found that the presence of LAE in ECG predicted maximum tumour dimension of > 5 cm and correlated with the degree of mitral regurgitation (MR). The LAE in ECG disappeared following surgery in 87.5% of patients.
Conclusion: The LA enlargement on ECG in a patient with LA myxoma signifies larger tumour size or the presence of significant MR but is not necessarily associated with an increased LA size or LV inflow obstruction.

Introduction

Myxomas are the most frequent benign primary tumour of the heart, and are most commonly located in the left atrium. There are very few reports describing usefulness of electrocardiograms (ECGs) in left atrial (LA) myxoma.[1,2] In this study, we retrospectively reviewed the ECGs of patients who underwent excision of LA myxoma in our institution. We analysed the effect of the tumour size and the effect of tumour removal on the ECG features.

Materials and methods

We retrospectively reviewed the records of all patients who underwent excision of LA myxoma and had a 12-lead ECG in sinus rhythm available for analysis before surgical excision of the tumour. All patients were evaluated preoperatively by echocardiography (ECHO) (two-dimensional, Doppler studies, and colour flow imaging) and many of the patients underwent coronary angiography.

All tumours were excised successfully through a septal incision during cardiopulmonary bypass with cardioplegic arrest. In some cases, the part of the atrial septum, where the tumour was attached was also excised and the residual defect was closed with a pericardial patch.

Standard 12-lead ECGs which were recorded at a paper speed of 25 mm/s, a sensitivity of 1 mV/cm and filter settings of 0.05–40 Hz. P-waves were analysed after photocopying the ECGs enlarging it to 200% size. The ECG intervals, including PR, QRS, and rate-corrected QT, were determined by averaging five consecutive beats from the rhythm strip record.

The P-wave duration in lead II (PDII) was defined as the time from the earliest onset of P-wave activity to the last activity. Lead V1 was used to measure the P-terminal Force (PTFV1), which was defined as the algebraic product of the terminal portion of the P-wave (seconds) and the negative deflection of the terminal portion of the P-wave (mV).[3,4] We used the generally accepted criteria of ECG LA enlargement (LAE), i.e. > 0.11 s of PDII and 0.004 mVs or more of PTFV1.[5,6] Only patients who satisfied both the ECG criteria were considered to have LAE. Another experienced investigator who was blinded to the clinical data repeated the ECG analysis. If there was a difference of opinion, then a third

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electrocardiographer analysed the tracings (this was done in 7/85 ECGs).

The ECHO records of the patients were retrieved. Left atrial dimension (LAD) was obtained by routine M-mode ECHO in the parasternal long-axis view. Left ventricular (LV) inflow gradient was obtained by Doppler evaluation by calculating velocity time integral of mitral inflow velocities. Mitral regurgitation (MR) was graded by standard Doppler criteria. Tumour dimensions were measured in three different planes. The maximum diameter in any of the plane was taken as a reference of size of the tumour in that plane. By calculating the average radius of the tumour in three different planes, on ECHO, the approximate volume was calculated using the formula $4/3\pi r^3$. The ECHO recordings were also analysed by two echocardiographers blindly and the opinion of a third person taken if there was disagreement.

As a routine, ECG and ECHO were repeated before discharge and the patients were reviewed at 3 months from the surgery. The significance of the difference in mean values for the various parameters before and after surgery was determined with Student's paired *t*-test. Results were considered significant if $P<0.05$. Statistical analysis was done using SPSS software version 10.

Results

There were 82 patients with LA myxoma, who were surgically treated in our institution between 1994 and 2003. Out of them, 51 patients (age 9–53 years) had a 12-lead ECG in sinus rhythm available for analysis preoperatively.

Of the total of 51 patients included in the study, 26 patients presented with symptoms of mitral inflow obstruction, nine patients with cerebrovascular accident, and seven patients presented with systemic symptoms such as fever and weight loss. Two patients presented with myocardial infarction and two patients were accidentally detected to have atrial myxoma.

The most common site of attachment of tumour was to the interatrial septum near fossa ovalis ($n=39$). Other sites of attachment were, to the LA roof in 9, posterior LA wall in 2, and to the posterior mitral leaflet in one patient. The latter patient underwent mitral valve replacement also.

Out of the 51 patients, there were 34 patients who had a tumour diameter in any given dimension >5 cm (Group I) and 17 had a maximum dimension of <5 cm (Group II). The mean PR interval in the ECG of patients in Group I was 0.18 ± 0.02 s (range 0.14–0.22) and in Group II was 0.17 ± 0.02 s (range 0.14–0.20). *P*=not significant. 28 patients out of 34 who had tumour size >5 cm, had LAE on ECG, while only 5/17 patients with tumour size <5 cm had LAE (Table 1). Thus, maximum tumour dimension of >5 cm correlated with LAE on ECG. When we analysed patients with and without LAE in ECG, more patients had tumour size >5 cm in the group with LAE. But the presence of LAE in ECG did not correlate with LA size on ECHO (Table 2).

The tumour volume of the patients ranged from 42.5 cm^3 to 209 cm^3 (mean value 95.6 cm^3). The LAE in the ECG correlated with the average tumour volume which was 112.9 cm^3 in patients with tumour size >5 cm, as compared to 59.9 cm^3 in tumours <5 cm ($P=<0.05$) (Table 1).

Table 1
Relationships of echocardiographic findings to tumour size.

	Tumour size >5 cm Group I ($n=34$)	Tumour size <5 cm Group II ($n=17$)	*P* value
LAE by ECG	28	5	<0.02
PR interval (sec)	0.18±0.02	0.17±0.02	0.7
LA size by ECHO	38.8±6.5	40.6±7.3	0.8
Tumour volume (cm^3)	112.9±63	59.9±12.9	<0.01
More than mild (2+) MR	$n=22$	$n=4$	<0.02
Mitral inflow gradient by ECHO (mmHg)	12.9±9	11±6	0.8

LAE: left atrial enlargement, ECHO: echocardiography, ECG: electrocardiography, LA: left atrium, MR: mitral regurgitation.

Table 2
Correlation of tumour size, volume, mitral regurgitation, and left atrial enlargement.

	LAE by ECG ($n=33$)	No LAE by ECG ($n=18$)
Tumour size		
>5 cm	28*	6*
<5 cm	5*	12*
LA size by ECHO (mm)	39±5**	39.3±6.6**
MR (mean grade-ECHO)	2.3±0.8	1.3±0.9

*$P<0.05$, ***P*=not significant. ECG: electrocardiography, ECHO: echocardiography, LA: left atrium, LAE: left atrial enlargement, MR: mitral regurgitation.

There was no significant difference in transmitral gradient in patients with larger or smaller tumours. The tumour size did not correlate with LV inflow obstruction.

A total of 22 patients in Group I had mild or more (2+ or more) MR, while only four patients in Group II had significant MR (Table 1). The mean grade of MR in patients who demonstrated LAE in ECG was 2.3+0.8 compared to 1.3+0.9 in patients who did not have LAE ($P=0.04$) (Table 2).

The ECG was available in 34 patients after surgery at 3 months follow-up. It was observed that LAE disappeared in the post surgical ECG in 21/24 patients who originally had LAE. The MR disappeared in 15 patients in Group I and in two patients in Group II after surgery, but none of the patients demonstrated more than mild MR after surgery.

Discussion

There are three commonly used criteria to define LAE in ECG. They are, total PD II >110 ms; PTFV1 >–0.04 ms, and P-wave notching in leads II with a peak-to-peak interval >40 ms.[3]

We used two of the above criteria combined together-prolonged P-wave and an increase of PTFV1, which are more sensitive and specific for LAE.[3] Jin et al.[4] reported that combination of the two criteria identifies patients most likely to have cardiovascular disease.

The LAE on ECG is produced by many factors like increase in LA size or intra-atrial pressure and intra-atrial conduction defects.

Fragola et al.[3] in a study of 1000 patients with LAE of different pathologies, found that in only 34% of the patients there was an agreement between LAE in ECG and LAE by ECHO. Also in a study by Scott et al.[7] in patients with mitral stenosis there was no direct correlation between LA size and LAE in ECG. In another study, Jin et al.[4] found that LAE on ECHO was present only in less than a third of the patients with LAE on ECG. Aggarwal et al. in a large series of patients with myxoma found only 35% of them demonstrating LAE on ECG.[8]

Josephson et al.[6] reported that the ECG pattern termed LAE appears to represent an interatrial conduction defect that can be produced by a variety of factors. They also opined that, considering cases of different aetiologies, only prolongation of interatrial conduction time was consistently related to the ECG pattern of LAE. The LA size or pressure was not predictably abnormal in patients with this pattern. They demonstrated that LAE in ECG correlated with ECHO LAE only in cases of mitral stenosis. In cases of cardiomyopathy, only LA pressure correlated with LAE in ECG, but not LA size. In cases of coronary artery disease, there was no correlation either with LA pressure or LA volume overload with LAE in ECG.

In a recent study Lee et al.[9] have found no correlation with LAE in ECG and ECHO, LA volume $>32\,mL/m^2$. Aggarwal et al. also found there is no correlation between tumour size and LAE.[8]

One of the two studies which correlated size of myxoma and ECG changes,[1,2] found that LAE in ECG disappears on removal of the tumour.[1] It is reported that large myxomas can produce LAE in the ECG.[2] In a study of 15 patients, Komiya et al.[1] reported the correlation of tumour weight to the presence of LAE in ECG. They found that tumours weighing $>16\,g$ produced LAE in ECG. Another study found correlation with size of the myxoma and pulmonary artery pressure.[5] The size of the tumour rather than the weight may be having more influence on LA pressure and intracavitary blood mass.[1] So, we analysed the tumour volume and correlated with LAE. We found that the larger tumours with a maximum dimension $>5\,cm$ and having a higher tumour volume produced LAE. But LA dimension as measured by ECHO was not different in patients with and without LAE in ECG.

In both groups with small and large tumours, we found that LA pressures were higher, indirectly, as indicated by the increase in transmitral gradient. There was significantly more MR in Group I which also might have contributed to the LAE. It is reported that mitral annular dilatation can be produced by LA myxomas, which can contribute to MR.[10] Annular dilatation can resolve after surgery due to remodelling. It is also reported that MR may be underestimated in the presence of myxoma,[11] though in our population none of the patients had significant MR after surgical excision.

Also increased intracavitary blood mass, which is supposed to affect heart-lead relationship, may have an effect in development of LAE in patients with larger tumours.[1] Whether the LAE correlates with intra-atrial conduction defect due to attachment of the tumour cannot be established.

We found that ECG evidence of LAE disappeared in most patients after excision of the tumour. This could be due to the relief of obstruction and decrease in transmitral gradient, decrease in MR, and removal of the tumour itself which might have decreased the intracavitary blood mass.

As the LA dimension by ECHO was not significantly different between the two groups, either the higher degree of MR or increased intracavitary mass may be the reasons for LAE in ECG in patients with LA myxoma.

Limitations of the study

1. Tumour volume was calculated in the assumption that the tumour is a sphere. But actually, the shape may vary from patient to patient and many times it may be different from a sphere.
2. It was a retrospective study and hence the recorded dimensions in some planes may be inaccurate.
3. The number of patients who had analysable ECGs after surgery were only 34/51 cases.
4. The LA size was measured by M-mode in parasternal long-axis view, which may not be accurate.

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