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Role of Quantitative Electromyography in Detecting Subclinical Skeletal Myopathy in Patients with Idiopathic Dilated Cardiomyopathy

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Abstract: Information concerning skeletal muscles in idiopathic dilated cardiomyopathy (DCM) patients could be clinically helpful as the disease-causing insult may be shared by both types of striatal muscles. Quantitative electromyography (QEMG) analysis permits the evaluation of less obvious changes with reliable objective measurements of the Motor Unit Potential. The study aimed at evaluating the skeletal muscle function using quantitative EMG to try to detect subclinical myopathy in patients with idiopathic DCM. The study included 30 patients with idiopathic DCM, 21 complaining of fatigue and exercise intolerance and 9 did not, all had previously underwent conventional qualitative EMG studies with normal results. They were evaluated by quantitative EMG using the multi-MUAP technique. No statistically significant difference was detected in the studied parameters between the patients and the reference values nor between the patients with fatigue and those without, neither compared to each other nor to the reference values. We concluded there was no detectable subclinical myopathy in the studied DCM patients

Key words: Quantitative electromyography · Myopathy · Dilated cardiomyopathy

INTRODUCTION

The pathology of skeletal muscle in heart failure (HF) patients (any origin) has been investigated [1, 2]. Information concerning skeletal muscle in dilated cardiomyopathy (DCM) patients could be clinically helpful as the disease-causing insult may be shared by both types of striatal muscles. Most DCM patients do not show the clinical markers of myopathy [3]. Some studies found subclinical skeletal myopathy in patients with DCM using muscle biopsy [3, 4] and electromyography [4].

Motor unit action potentials (MUAPs) represent the summated activity of a portion of synchronously firing muscle fibers of a motor unit. They reflect the structure of the motor unit: diameter, distribution and number of muscle fibers [5]. Structural alterations of motor units are reflected as changes of the MUAPs. Quantitation of the MUAP parameters such as duration, amplitude and phases has been used to characterize neuromuscular disorders [5]. Quantitative EMG (QEMG) analysis permits the evaluation of less obvious changes with greater accuracy that gives reliable objective measurements of the Motor unit potential (MUP) [6].

This study aimed at evaluating the skeletal muscle function using quantitative EMG to try to detect subclinical myopathy in patients with idiopathic DCM with HF who underwent conventional qualitative EMG studies with normal results.

MATERIALS AND METHODS

After being approved by the local ethical committee and after informed consent 30 Patients diagnosed, after a general and cardiological examination and battery of investigations including laboratory work up, electrocardiography, echocardiography, coronary angiography, as having idiopathic dilated cardiomyopathy were recruited from the cardiology department at Cairo University (Kasr El-Ainy) hospital and subdivided in Functional classes (FC) according to the New York Heart Association Functional Classification (NYHA) [7]. Patients with known etiology for the dilated cardiomyopathy (ischemic, …), those with preexisting neuro-muscular disorders and patients on oral anticoagulants were excluded from the study. The patients underwent conventional qualitative EMG studies with normal results.

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Quantitative EMG examination was carried out to the patients in the Clinical Neurophysiology Unit, Kasr El Ainy hospital using a *Dantec Keypoint* apparatus and a conventional disposable concentric needle electrode with a recording area of 0.07 mm² to the right vastus lateralis muscle.

Quantitative analysis was done using the multi-MUAP Analysis technique, which is relatively less time consuming than the classical signal averaged MUP analysis. The technique was used as described by Bischoff *et al*. [5]. The needle electrode was inserted perpendicular to the skin. The electrode was then redirected in skew angels and with different depths. Two or three skin insertions, spaced at least 10 mm, preferably perpendicularly to the fiber direction have been performed. A weak contraction has been used, usually giving 3-5 MUPs from each recording. A special notice has been taken to a constant recording position during 5 seconds preceding the analysis. The target number of MUPs was 20 but more MUPs have been aimed for, to allow easy editing by deletion, rather than by resetting cursors.

For each patient the mean values and standard deviations (SD) of each MUAP parameter were calculated: Amplitude (μV) Duration (ms) Thickness (area divided by amplitude) and size index (normalized thickness).

The values were compared to the reference data of Bischoff *et al*. [5].

Statistical Analysis: Data was analyzed by Microsoft Office 2003 (excel) and Statistical Package for Social Science (SPSS) version 16. Parametric data was expressed as mean ± SD and non parametric data was expressed as number and percentage of the total. Comparing the mean ± SD of 2 groups was done using paired and unpaired student's t test. P value < 0.05 was considered significant.

RESULTS

The mean age of the patients was 46±12 years, 28 males (93%) and 2 females (7%). 7 patients were hypertensive, 6 were diabetic, 8 patients smokers and 2 patients had a history of renal impairment. According to clinical HF condition there were 14 patients belonging to NYHA FC I and II and 16 patients belonging to NYHA FC III and IV.

21 patients complained of fatigue and exercise intolerance. Table 1 shows that there was a statistically significant difference between the two groups of functional classes as regards fatigue.

Clinical and laboratory data, including the creatine phosphokinase (sCPK) which showed normal levels, are shown in table 2.

From table 2 there was no significant difference between both groups regarding their age, blood pressure, diabetes and renal function test (P> 0.05).

Table 1: Comparison between functional classes and fatigue

	Classification				
Fatigue	NYHA 1 and 2 (n=14)		NYHA 3 and 4 (n=16)		p value
Yes	5	35.7%	16	100.0%	0.000*
No	9	64.3%	0	0	

*Statistically significant

Table 2: Clinical and laboratory differences between both studied groups

	With fatigue	Without fatigue	P value
Age (mean ± SD)	48.7±12.3	41.2±11.3	0.131
Duration of disease(months)	45.5±31.7	14.3±6.4	0.007*
Systolic blood pressure	117.6±14.8	118.9±22	0.545
Diastolic blood pressure	76.4±9.4	78.9±11.7	0.545
Diabetes(n)	6	0	0.07
Renal disease(n)	1	1	0.5
NYHA III and IV	16	0	0.00*
Heart rate	85.9±9.7	78.9±10.5	0.632
Urea	49.3±30.4	38.7±19.1	0.343
Creatinine	0.6±0.2	1.2±0.3	0.563
Creatine phosphokinase	83.9±27.8	64±44	0.144

*Statistically significant

Table 3: Differences between two groups regarding echocardiogrphic data

	Fatigue	Non fatigue	P
LVESD	6.1±0.8	5.6±0.8	0.99
LVEF	29.2±4.4	34.4±5.5	0.009*
RVD1	4.1±0.8	3.7±0.5	0.118
RVD2	3.1±0.8	3.2±0.5	0.859
RVD3	7.714±1.0594	7.100±0.9247	0.143
TAPSE	1.852±0.3341	1.833±0.1414	0.871
PASP	45.10±9.170	33.67±4.664	0.001*
E	0.7333±0.25852	0.6333±0.17628	0.301
A	0.5310±0.22318	0.8367±0.92899	0.160
DT	182.67±87.161	166.67±53.742	0.615
Jet area	9.491±21.5044	2.522±1.8295	0.345

*Statistically significant

LVESD: left ventricle end-diastolic diameter, LVEF: left ventricle end-systolic diameter, LVEF: left ventricle ejection fraction, RVD: right ventricle diameter, TAPSE: transannular plane systolic excursion, PASP: pulmonary artery systolic pressure E, A: velocity, DT: deceleration time,

Table 4: Patients data as compared to reference values

Quantitative EMG	Patients Mean±SD	Reference values Mean±SD	P Value
Amplitude (μv)	665.62± 211.26	687±239	0.67
Duration (msec)	12.09±1.08	11.7 ±1.9	0.29
Thickness	1.7±0.1	1.72±0.23	0.65
Size index	1.2±0.3	1.24±0.39	0.62

Table 5: Fatigued Patients data as compared to reference values

Quantitative EMG	Fatigue Patients Mean±SD	Reference values Mean±SD	P Value
Amplitude μv	684.08±241.8	687±239	0.96
Duration msec	11.84±1.0	11.7 ±1.9	0.75
Thickness	1.73±0.19	1.72±0.23	0.86
Size index	1.3±0.34	1.24± 0.39	0.53

Table 6: Non Fatigued Patients data as compared to reference values

Quantitative EMG	Non Fatigue Patients Mean±SD	Reference values Mean±SD	P Value
Amplitude μv	610.25±57.76	687±239	0.34
Duration msec	12.55±1.03	11.7 ±1.9	0.19
Thickness	1.76±0.19	1.72± 0.23	0.62
Size index	1.26±0.29	1.24± 0.39	0.88

Table 7: Fatigued Patients data as compared to non fatigued Patients data

Quantitative EMG	Fatigue Patients Mean±SD	Non Fatigue Patients Mean±SD	P Value
Amplitude uv	684.08±241.8	610.25±57.76	0.38
Duration msec	11.84±1.0	12.55±1.03	0.09
Thickness	1.73±0.19	1.76±0.19	0.69
Size index	1.3±0.34	1.26±0.29	0.76

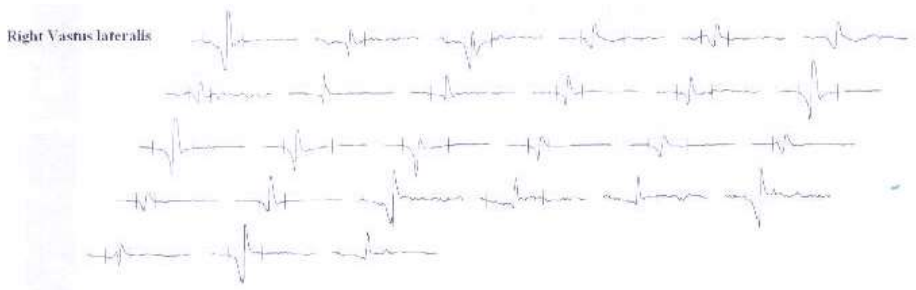
The fatigued patients had a longer duration of disease than non fatigued patients ($p = 0.007$) and exhibited more severe symptoms ($p < 0.01$).

Echocardiogrphic data are summarized in table 3. The left ventricle end-diastolic diameter (LVEF) appeared to be significantly reduced in fatigued patients than non fatigued with significant elevation of pulmonary artery systolic pressure (PASP).

Regarding the quantitative EMG data there was no statistically significant difference in the studied parameters as compared to the reference values:

mean amplitude, mean duration, mean thickness and mean size index.

In addition there was no statistically significant difference between the patients who were complaining of fatigue and exercise intolerance and those who were not, compared to each other and to the reference values (Tables 4-7) trace 1.



Trace 1: The accepted MUAPs in one of the patients

DISCUSSION

In our study all patients with DCM had no evidence of subclinical skeletal myopathy using quantitative electromyography.

This finding is contrary to the finding of Caforio *et al*. [4] who found by muscle biopsy and quantitative EMG subclinical skeletal myopathy in patients with DCM [4]. Also a study found in skeletal muscle biopsy some non specific subclinical muscles damage that was unrelated to the functional class and duration of DCM [3]. The present study did not include a muscle biopsy.

The negative findings in the EMG, even using quantitative techniques, do not exclude myopathy as in myopathies there may be patchy distribution of abnormalities within the muscle and sparing of some of the muscles [8].

The irregular distribution of EMG abnormalities might have several causes: First, there are degenerative and regenerative processes that occur simultaneously. Some muscle fibers might be atrophic, while others hypertrophic. MUPs with large amplitudes and short durations might be obtained when a hypertrophic fiber is close to the electrode tip [9]. In addition, abnormalities might be patchy if only one fiber type is involved. The distribution of EMG changes will then follow the distribution of this fiber type, which may vary with the depth of the muscle [9]. The values, expressed as mean values, were normal probably because the small MUPs were mixed with normal or large ones [9]. That was why Stalberg *et al*. [9] proposed the outlier method as a way to detect abnormalities in quantitative EMG, which defines abnormality by using extreme values. They compared it with conventional mean values and found the outlier method to be more sensitive in myopathies. The same conclusion was reached by Datoios *et al*. [10]

The fact that the EMG protocol used, mainly examines slow twitch type I muscle fibers [11], while type II fiber atrophy was found to be evident in heart failure [12], may be one of the causes of the negative results.

Our patients had also normal serum CPK, which could indicate that the patients had no muscle disease, however the CPK may be normal in cases with mild or slowly progressive myopathies [8]. Also a study of 114 candidates for myopathy on the basis of persistently increased CPK levels and muscle weakness found that skeletal muscle biopsy allowed a definite diagnosis of myopathy in 10.5% of cases and a probable diagnosis in 7.9% [13].

Molecular studies showed that although the cardiac involvement, DCM-like phenotype, is often found in the patients with muscular dystrophy, a large number of the patients with isolated DCM do not manifest with the skeletal muscle phenotype. The etiological link between hereditary cardiomyopathy and inherited skeletal muscle myopathy has raised the question as to how the mutations in the genes/proteins, expressed both in skeletal and cardiac muscles, cause heart-specific disease phenotypes in the isolated DCM. The most probable explanation was that the difference in the clinical phenotypes, muscular dystrophy and DCM, can be caused by mutations in specific and/or different functional domains affecting specific functions [14].

We concluded that we could not find by QEMG any evidence of subclinical skeletal myopathy in patients with idiopathic DCM, whether this is due to lower sensitivity of the selected EMG technique or due to actual absence of myopathy, needs to be confirmed by further studies with larger numbers of patient, using QEMG techniques with higher sensitivities, examining a muscle biopsy and genetic testing.

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