Comparison Of 12-Lead-Ecg High-Frequency Filtering In An Outpatient’s Population

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Abstract

Background: the accurate recording and precise analysis of the ECG traces is crucial for a good clinical interpretation, especially in outpatient assessment before surgical interventions, in whom the ECG is the first and often unique cardiological evaluation. The 2007 AHA/ACC/HRS recommendations for the standardization and interpretation of the electrocardiogram suggests an High-frequency filtering cut-off of at least 150Hz for all adolescents and adult ECGs to eliminate potential invalidations of any amplitude measurements used for diagnostic classification. However the mostly used 40Hz filtering allow a better graphic resolution of the traces. Aim of this study is to compare the 150 and 40Hz high pass filtering in a population afferent to the hospital and candidate to surgical interventions.

Methods: each patient underwent to a 12-lead ECG in double high frequency filtering (150Hz and 40 Hz), analyzed by two blinded cardiologist. The baseline characteristics and ECG signal differences were collected also in terms of subjective quality perception (grading between 1 as poor quality and 3 good quality).

Results: a total of 1582 patients were analyzed (42% males), 97.5% was in sinus rhythm with an average heart rate of 68.2±11.5 bpm, RBBB and LBBB was present in 7.4% and 2.5% respectively; 2.2% of patients was in atrial fibrillation and 7% had a 1st degree AV block at baseline ECG. ST-T anomalies were seen in 33.7% and 11.6% had Q waves > 1mm in at least one lead; 1.1% had pacemaker’s spikes visible. Analyzing the trace’s differences at 150Hz and 40Hz, the study population did not show any statistical difference in terms of ST and T wave abnormalities, presence of significative Q waves and visible pacemaker spikes (p=0.26; 0.79; 0.74; 1 and 1, respectively). However the QRS amplitude (manually measured adding the maximal positive and negative QRS deflection in precordial leads) demonstrated a significative difference between the groups (p<0.0001), this finding was reflected also in Left ventricular hypertrophy diagnosis that was significatively different between the two traces (P<0.0001) in favour of 150 Hz, and a difference in J point elevation diagnosis in favor of 40 Hz (P=0.007). As expected, in 40Hz ECG there was an average reduction in QRS amplitude of 2±2.1 mV and 25  patients (0.01%) were not diagnosed with left ventricular hypertrophy, obviously the subject with borderline QRS amplitude for Sokolow criteria. There was instead a significative difference in favor of 40 Hz traces in terms of perceived quality (P < 0.0001). Of note, in the percentage of poor quality traces (0.6% Vs 10.1% respectively) a 45% of the traces was judged not readable by the physician (4 Vs 71 traces).

Conclusions: the 40Hz ECG filtering permits an accurate ECG analysis. It may underestimate the left ventricular hypertrophy in a small percentage of patients at a cost of a significative amelioration in perceived traces’ quality.
Long-Term Arrhythmic Prognosis In Patients With Biopsy-Proven Myocarditis, Studied By Cardiac Magnetic Resonance Imaging And Electroanatomic Mapping


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Abstract

Background: Data on long-term follow-up in patients (pts) with biopsy-proven myocarditis are conflicting, particularly those regarding ventricular events.

Purpose: To determine the prognostic variables for ventricular arrhythmias (VA) in pts with biopsy-proven myocarditis.

Methods: We prospectively studied consecutive pts with endomyocardial biopsy (EBM)-proven myocarditis (M1 Group). Control group was represented by pts presenting with suspected myocarditis, without EBM evidence of myocarditis (M0 Group). All pts underwent cardiac magnetic resonance imaging (MRI), coronary angiography, electrophysiological study (EPS) and electroanatomic mapping (EAM). Implantable cardioverter defibrillator or loop recorder was implanted in a subgroup of pts, following guidelines. The primary endpoint was the occurrence of sustained VA at follow-up (FU).

Results: 49 pts with biopsy-proven myocarditis were enrolled in Group M1, and 10 patients in Group M0 (mean age 41±15 vs 42±13 years respectively, p=0.87; men 59% vs 70% respectively, p=0.52). There were no statistically significant differences between the 2 groups regarding clinical variables and imaging parameters. Group M1 showed greater involvement of the right ventricle (RV), expressed as wider low-voltage area, compared to group M0, both at bipolar and unipolar mapping (Table 1). At 37±24 months of FU, there were 12 VA in group M1 vs 1 VA in group M0 (24% vs 10%, p=0.44). VA predictors among Group M1 were the presence of left ventricular systolic dysfunction (HR 3.5, 95% CI 1.3-9.4, p=0.01) and ventricular tachycardia (VT) induction at EPS (HR 5.3, 95% CI 1.9-14.9, p=0.001). At multivariate analysis, inducible VT remained the only independent predictor of VA in pts with myocarditis (HR 4.1, 95% CI 1.3-12.6, p=0.015).

Conclusions: A higher degree of RV unipolar and bipolar mapping alterations was observed in pts with biopsy-proven myocarditis, confirming that substrate alteration at EAM reflects histological abnormalities in these pts. VT inducibility was the only independent predictor for VA in pts with biopsy-proven myocarditis.

Table 1: Right Ventricle EAM

<table>
<thead>
<tr>
<th></th>
<th>Group M1 (n=49)</th>
<th>Group M0 (n=10)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bipolar Low Voltage (&lt;1.5 mV) Area (%)</td>
<td>10.0 ± 10.4</td>
<td>2.4 ± 3.2</td>
<td>0.04</td>
</tr>
<tr>
<td>Bipolar Scar Area (&lt;0.5 mV) (%)</td>
<td>3.9 ± 6.9</td>
<td>0.4 ± 0.6</td>
<td>0.015</td>
</tr>
<tr>
<td>Unipolar Low Voltage (&lt;5 mV) Area (%)</td>
<td>21.1 ± 20.1</td>
<td>6.7 ± 10.7</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± standard deviation
Heart Conduction System Disorders In Cardiac Sarcoidosis

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Abstract

Introduction: Sarcoidosis is a rare, multisystem, granulomatous disease of unknown etiology. It usually presents in young adults but has been reported in children and in the elderly, especially in Japan. Noncaseating granulomas are the pathological hallmark of sarcoidosis. Sarcoidosis usually affects the respiratory system or mediastinal lymph nodes in more than 90% of cases, but may involve almost any organ.

Early autopsy studies suggest up to 27% of patients with sarcoidosis have myocardial involvement.

Isolated cardiac sarcoidosis, that is, with no detectable evidence of sarcoidosis in other organs, is rare. A recent review of sarcoidosis placed isolated cardiac involvement at 2%, one of the least common manifestations.

Although Sarcoidosis is not commonly fatal, cardiac involvement may be responsible for more than two-thirds of deaths. In Japan, cardiac involvement is more common in older women and responsible for as many as 58% to 85% of deaths from sarcoidosis. The most frequently involved area is the ventricular septum [one third], followed by the inferior wall, anterior left ventricle, right ventricle, and lateral left ventricle.

Cardiac sarcoidosis may be an asymptomatic accompaniment to pulmonary disease or may be the presenting feature of sarcoidosis. The clinical manifestations of cardiac sarcoidosis include conduction disorders, arrhythmias, atrial and ventricular arrhythmias, congestive heart failure, valvular pathology, pericardial effusions, and sudden cardiac death.

Cardiac complications are the second leading cause of sarcoidosis-related death, after respiratory complications, in the United States. Patients with CS have a worse prognosis than patients without cardiac involvement.

Early Screening for cardiac involvement in patients with sarcoidosis in other organ systems is critical as sudden death may be the first manifestation of cardiac sarcoidosis. Performing a detailed clinical history to identify cardiac symptoms (including syncope, presyncope, or palpitations), a 12-lead electrocardiogram (ECG), and an echocardiogram is recommended.

Further screening is advised if any of the initial screening investigations yields an abnormality, cardiovascular magnetic resonance (CMR) or fluorodeoxyglucose positron emission tomography (FDG-PET).

Screening for cardiac involvement in specific clinical presentations in patients without known sarcoidosis can also identify CS patients early in their disease course. The clinical diagnosis of cardiac sarcoidosis (CS) is therefore critically important to the timely planning of therapeutic strategies.

We present two patients with Cardiac Sarcoidosis.

Purpose: Our purpose is to raise awareness of cardiac involvement in Sarcoidosis and its clinical implications.

Patients and Materials: We describe two patients with cardiac Sarcoidosis which the initial presentation was heart conduction abnormality.

Case 1: The first patient is a healthy 43-year-old male, works as park ranger. Presented with Syncope and transient complete atrio-ventricular block (CAVB) on ECG which required Cardio-Pulmonary Resuscitation (CPR), temporary and shortly after a permanent pacemaker implantation. Further investigation included : chest X-ray, CT and Gallium-scan indicate evidence of Hilar and Axillar lymphadenopathy. The final diagnosis of Sarcoidosis was established by Mediastinal lymph node biopsy.

Case 2: The second patient is a 58-year-old female with hyperlipidemia, recently diagnosed with Pulmonary Sarcoidosis (presented two years earlier by cough and dyspnea) and was proven by a CT scan (demonstrating enlarged mediastinal lymph-nodes) and by mediastinal lymph node biopsy. The patient presented to us with fatigue, dizziness, pre-syncope and evidence of a high degree atrio-ventricular (AV) block (without signs of cardiac ischemia) required permanent pacemaker implantation. Prior to the implantation there was no evidence of inducible tachyarrhythmia on an Electro-Physiological-Study (EPS).

Summary and Conclusions: We describe two patients with Cardiac Sarcoidosis initially presented with high degree AV Block required pacemaker therapy.

One patient already diagnosed with Pulmonary Sarcoidosis and in the second one the diagnosis was established during the recent event. Our recommendation, supported by expert opinion, is to preform early screening to patients with Sarcoidosis for cardiac involvement by detailed clinical history, a 12-lead ECG and echocardiogram. When needed other modalities as CMR also can be performed. Thus save lives.
The Left Ventricular Noncompaction Syndrome In 45 Adults: Clinical Variants, Follow-Up And Outcomes

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Abstract

Purpose: to study clinical variants, follow-up and outcomes of the left ventricular noncompaction (LVN) syndrome by adult patients.

Methods: The diagnosis of LVN was established in 45 adults (21 females, 18-76 years, on the average 42.7 ± 15.3) on the basis of visual criteria, in 9 patients using three methods (Echo-CG, MRI, CT), in 28 patients using any two methods. Were also performed endomyocardial/intraoperative myocardial biopsy and autopsy (n=11) with viral DNA investigation (real-time PCR), anti-heart antibody investigation. The mean follow-up was 12 [5.0; 28.5] months, from 3 month to 12 years.

Results: there was a high frequency of the association of LVN syndrome and other heart disease by adults: 6 patients (13%) had congenital heart disease (atrial and ventricular septal defects, pulmonary artery stenosis, bicuspid aortic valve, vascular malformations), 3 (6%) – hypertrophic cardiomyopathy, 2 (4%) – arrhythmogenic right ventricular cardiomyopathy, 1 (2%) – genetic myodystrophy with cardiac involvement, 1 (2%) – restrictive cardiomyopathy, 1 (2%) – Danon disease. Only one patient with LVN syndrome had no symptoms (but dilated cardiomyopathy, DCM, by Echo-CG). In 10 patients (23%) LVN had “idiopathic” arrhythmias mask, in 15 patients (35%) – mask of DCM, in 4 patients (9%) LVN was first identified at the same time with acute myocarditis. In 5 cases (11%) LVN was associated with ischemic heart disease, but in 2 patients (11%) the initial diagnosis “ischemic heart disease” was false. The associated myocarditis was in 26 patients (58%) detected, incl. 11 patients by morphologic study of myocardium. The virus was found in 6 patients in the myocardium (parvovirus B19 in 5, human herpes virus type 6 in 2, herpes simplex virus type 1, 2 in 1 and Epstein-Barr virus in 1) and in 11 patients in the blood.

Heart failure was in 36 patients (80%) and angina pectoris in 10 patients (22%) diagnosed; 6 patients had a typical signs of the myocardium infarct due to coronary artery atherosclerosis, necrosis in myocarditis, embolism from left ventricle. The incidence of atrial fibrillation was 24% (11 patients), stable and unstable ventricular tachycardia – 62% (28 patients), left bundle branch block – 16% (7 patients). The average LV diastolic size was 6.1±0.9 cm, LV ejection fraction 35.4±15.0%. Eleven patients (24%) without anticoagulants had intracardiac thrombi; 10 of them had LV ejection fraction less than 40%. Three patients (7%) had embolism (renal, pulmonary, myocardium infarct, stroke). The devises was in 18% patients (40%) implanted: CRT in 1 (2%), ICD in 11 (24%), pacemakers in 3 (7%). In two patients was cardiac transplantation performed. The morality was 11.1% (5 patients).

Conclusions: LVN syndrome in adults is vary polymorphic and often associated with other cardiomyopathy (18%) and myocarditis (58%). The frequency of complications (incl. stable ventricular tachycardia, embolism) is high, that requires development of differential approaches to the treatment.
Abstract

Purpose: evaluate treatment stages of dilated cardiomyopathy (DCM) in a patient with Emery-Dreifuss muscular dystrophy (EDMD).

Methods: male patient, 38 y.o., suffers low progressive skeletal myopathy from the childhood. Patient’s mother has no clinical signs of myopathy but got a pacemaker at the age of 54 y.o. Now she revealed moderate dilation of the cardiac chambers with ejection fraction (EF) 50%. Two patient’s sons are healthy. From the age of 32 years patient had arrhythmias and minimal EF reduction. In 2012, he showed signs of DCM, sick sinus syndrome, transient AV block II degree type 1, paroxismal atrial flutter, and unsustained ventricular tachycardia (VT).

Results: The examination revealed walking difficulties, moderate knees and elbows contractures, high CK level, normal intelligence, significant arrhythmias (atrial flutter, AV block I-II, right bundle branch block, about 5.000 PVCs per day), and DCM (left ventricle diastolic diameter 6.6 cm, EF 42%). Mutations analysis was performed by PCR-based direct Sanger sequencing of coding area and adjacent intronic areas of genes of interest. Two genetic variants were detected: frame-shift deletion c.del619C in EMD gene causing premature stop-codon appearance and protein shortening (p.236X), and c.IVS4-13T>A in LMNA gene. Both variants were not found in control group of 100 healthy volunteers. The patient’s mother revealed a deletion c.del619C in the heterozygous state.

Radiofrequency ablation of the cavotricuspid isthmus was performed, and dual-chamber ICD was implanted. The patient received amiodarone, but during 5 month after ICD implantation developed persistent incisional atrial flutter with a progressive impairiment of cardiac pump function (EF 16%). Signs of the associated myocarditis were not detected. Electrical defibrillation was effective, but complicated by sustained VT. During the following week patient had repeated appropriate shocks. ECMO and urgent heart transplantation were successfully performed. Morphologic study revealed diffuse myocardial fibrosis, atrophy and hypertrophy of cardiomyocytes. After basiliximab induction therapy, prednisone, tacrolimus and mycophenolate were assigned. The follow-up period is 12 month with significant health improvement.

Conclusions: cardiac involvement in EDMD can rapidly progress and requires regular monitoring. In all cases of impairment, should be excluded myocarditis. Verification of a genetic variant is essential for treatment strategy. Despite the peripheral myopathy and limitations in the application of anesthetics, heart transplantation can be performed successfully.
A Rare Case Of Flecainide Poisoning Complicated By Cardiogenic Shock


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Abstract

A 38 years-old woman was admitted to the Emergency Room in cardiogenic shock and obnubilated. Her past history was known because of a recent ablation of an AV accessory pathway, previously refractory to flecainide, and included a congenital hydrocephalus with reduction of mental abilities. Flecainide had been suspended since the ablation and no information on recent drug assumption was available. The EKG showed sinus bradycardia, a prolonged P wave, 1° AVB, RBBB and LAFB (QRS 200 ms) (Figure 1). Routine laboratory tests were unremarkable. Severe biventricular dysfunction was found (LVEF 35%) at echocardiogram.

Activated charcoal was administered. Pulmonary embolism and cerebrovascular events were excluded with a CT scan. The patient was then intubated and admitted to the Intensive Care Unit. Support fluid therapy and low-dose inotropic drugs were started. Over the next 36 hours EKG returned to baseline (Figure 2) and LVEF improved to 50%. Flecainide plasma levels were 2600 ng/ml at 12 hours and 1000 ng/ml at 26 hours from admission (NR 200-1000 ng/ml).

After recovery, the patient confessed inappropriate flecainide assumption and was therefore referred to psychiatric consult.

Figure 1: EKG on admission: sinus rhythm. Prolonged P wave (100 ms). First degree atrioventricular block (PQ 230 ms). Right bundle branch block (QRS interval 200 ms). Left anterior fascicular block.

Figure 2: EKG on day 2nd: sinus rhythm. First degree atrioventricular block (PQ 210 ms). Right bundle branch block (pre-existing).