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Case Report

Value of the electrocardiographic strain pattern in children and adolescents with end stage renal disease

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ABSTRACT

Background: Among several electrocardiographic LVH criteria, LVH strain pattern was implicated as the strongest marker of cardiovascular adverse outcomes in general population and hypertensives.
Objective: The aim of this work is to detect the prevalence, clinical relevance and determinants of ECG strain pattern presence in children and adolescents with ESRD.
Methods: The study was performed at Zagazig University Hospital during the year 2012–2013. The studied patients with ESRD undergoing hemodialysis (24 males and 26 females) were categorized into three groups according to ECG. All patients were subjected to laboratory investigations (albumin, fasting lipids, renal function, serum creatinine, K^+ serum, Ca^{2+} serum, phosphorus serum, alkaline phosphatase [ALP] serum, hemoglobin, pro BNP and parathormone), standard 12 leads surface ECG, and echocardiography (ECHO).
Results: ECG strain was the most prevalent electrocardiographic pattern among the studied patients (42% versus 26% normal ECG and 32% ECG LVH without strain) who clinically have more ischemic chest pain (81% in group III versus 23.1% in group I and 43.8% in group III, $p = 0.003$) and systolic hypertension ($p = 0.006$). The prolonged disease duration ($p = 0.005$), increased LVM ($p = 0.007$), higher TC ($p < 0.01$), lower Hb ($p = 0.02$) and increased internal carotid velocity ($p = 0.05$) were the independent variables associated with the ECG strain pattern.
Conclusion: Identification of ECG strain pattern is correlated with myocardial ischemia risk, so it could be helpful in risk stratification of children and adolescents with ESRD. Factors determining ECG strain are prolonged disease duration, increased LVM, higher TC, lower Hb and increased internal carotid velocity could become an important part of management of ESRD patients.

1. Introduction

Children share an increased risk for death from a spectrum of uremia-related cardiovascular abnormalities but in absence of classical atheromatus coronary artery disease.[1] Deaths attributed to cardiovascular disease (CVD) are highest among African American children and young adults on dialysis.[2,3]

The classic left ventricular (LV) strain pattern (ST segment depression and T-wave inversion) on the left precordial leads of standard resting ECG is a well marker of anatomic LV hypertrophy (LVH) presence.[4–6] However, the presence of this pattern of ventricular repolarization abnormality has been associated with a worse prognosis both in hypertensive subjects[7,8] and in general population.[9,10] Moreover, among several electrocardiographic LVH criteria, LVH strain pattern was implicated as the strongest marker of cardiovascular adverse outcomes.[7,9–11]

These untoward clinical consequences of electrographic LV strain pattern could be due to its association not only with underlying coronary heart disease[3,6] but also with cardiovascular risk factors, such as hypertension, diabetes, old age, and male gender.[3,6–11]

However, the associations of ECG strain with LVH and other cardiovascular risk factors in children and adolescents with ESRD have not been extensively investigated. It is potentially important to declare the additional value of ECG strain presence may provide in these patients beyond that obtained from echocardiographic LVH.

Therefore, we elected to detect the prevalence, the clinical relevance and determinants of ECG strain pattern presence in children and adolescents with ESRD.

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2. Patients and methods

2.1. Patients

All patients at pediatric nephrology unit in Zagazig University Hospitals who had been on HD for at least 1 month between January and September 2012 were considered for inclusion in the study. We excluded patients with congenital heart disease, pericarditis, pulmonary embolism, concurrent respiratory illness, left bundle branch block or atrial fibrillation.

The local ethics committee had approved the study protocol and informed consent was obtained from patient's guardians. Patient's records were reviewed for demographic data; relevant medications, etiology and duration of ESRD, history of angina and presence of cardiovascular risk factors. Blood pressure (BP) measurement and body mass index (BMI) were obtained.

2.2. Biochemical analysis

Routine electrochemistry, full blood count, serum creatinine (sCr), albumin, blood nitrogen urea (BUN), calcium, phosphorus, potassium, alkaline phosphatase, C-reactive protein (hsCRP), cardiac troponin (cTnI) analysis, serum iron and transferring, fasting lipid profile, pro BNP and parathormone were performed.

2.3. Electrocardiography

Standard resting 12-lead ECGs were recorded digitally by the same equipment at 25 mm/s paper speed and 10 mm/mV amplitude and analyzed by an independent observer unaware of other patients' data. Typical ST-T wave strain pattern was defined by presence of downsloping convex ST segment depression concomitantly with inverted asymmetrical T-wave opposite to QRS axis in leads V5 and V6.[9]

2.4. Echocardiography

It was carried-out using Siemens echocardiographic machine with a 3–5 MHz multiphase array probe by a single experienced cardiologist blinded to all of the clinical and electrocardiographic data of the patients. 2D guided M-mode images were obtained from the short axis parasternal view at the level of the tips of the mitral valve leaflets, and measurements of interventricular septal wall thickness, posterior wall thickness (PWT), and LV end diastolic diameter (LVEDD) were made at the peak of the ECG R wave according to the American society of cardiology convention.[12] The mean of 3 cycles was considered. LVM was calculated by anatomically validated cube formula of Devereux et al.[13] and was indexed to body surface (LVMI). Echocardiographic LVH was defined as LVMI $>116\ g/m^2$ in males and $>104\ g/m^2$ in females. Relative wall thickness (RWT) was measured as ratio of 2 (PWT/LVEDD) and considered increased if >0.43. Patterns of LV geometry were defined according to LVH and RWT: (1) normal (no LVH, normal RWT); (2) concentric remodeling (no LVH, increased RWT); (3) eccentric hypertrophy (LVH, normal RWT); and (4) concentric hypertrophy (LVH, increased RWT).[14] Regional wall motion (RRWM), ejection fraction (EF) by the Simpson's rule were detected trans mitral flow and tissue Doppler echocardiography were used for LV diastolic function assessment.

2.5. Statistical analysis

All of the statistics were performed by SPSS statistical package version 16.0. Continuous data were described as means and SD. Comparisons between patients groups were performed by ANOVA test in normally distributed data and by nonparametric Mann–Whitney test in asymmetrically distributed data. Associations with ECG strain were determined by stepwise multivariate logistic regression analysis. $p < 0.05$ was regarded significant.

3. Results

3.1. Baseline characteristics

By end of the study, 50 patients had been registered. Patients were classified according to ECG into group I with no LVH criteria (13, 26%), group II with LVH but no strain (16, 32%) and group III with LVH and strain (21, 42%).

Tables 1 and 2 show demographic, clinical, echocardiographic and laboratory variables in patients groups. Compared to groups I and II, patients with LVH and strain (group III) had longer renal disease duration (5.48 ± 1.97 years versus 2.76 ± 1.1 in group I and 3.02 ± 1.7 in group II, $p = 0.04$), typical chest pain (81% versus 23.1% in group I and 43.8% in group II, $p = 0.003$), higher systolic BP (141.4 ± 21 versus 123.1 ± 14.9 in group I and 122.5 ± 20.2 in group II, $p = 0.006$), more eccentric LVH geometry (57% versus 31% in group I

Table 1
Demographic, clinical and echocardiographic characteristics of the studied groups.

	Group I, Normal ($n=13$)	Group II, LVH ($n=16$)	Group III, LVH with strain ($n=21$)	p value
Age, $X \pm SD$	10.44 ± 3.4	10.25 ± 2.8	10.85 ± 4.1	0.7
Sex, n(%)				
Male	9 (69.2%)	6 (37.5%)	11 (52.4%)	0.2
Female	4 (30.8%)	10 (62.5%)	10 (47.6%)	
Duration, $X \pm SD$	2.76 ± 1.1	3.02 ± 1.7	5.48 ± 1.97	0.04*
CP, n(%)	3 (23.1%)	7 (43.8%)	17 (81%)	0.003*
HPN, n(%)	7 (53.8%)	13 (87.5%)	19 (90.5%)	0.01**
SBP, $X \pm SD$	123.1 ± 14.9	122.5 ± 20.2	141.4 ± 21	0.006*
DBP, $X \pm SD$	75.4 ± 11.9	74.4 ± 11.5	82.9 ± 15.9	0.1
LVED, $X \pm SD$	50.6 ± 3.0	51.1 ± 4.4	52.2 ± 2.8	0.6
LVES, $X \pm SD$	30 ± 4.5	33.1 ± 7.3	32.9 ± 6.6	0.4
EF, $X \pm SD$	70.8 ± 6.1	64.9 ± 9.7	65.5 ± 8.7	0.1
E-wave, $X \pm SD$	57.9 ± 3.6	38.3 ± 3.9	56.8 ± 3.1	0.4
A-wave, $X \pm SD$	65.7 ± 3.5	66.5 ± 3.9	64.8 ± 3.2	0.3
DT, $X \pm SD$	140.3 ± 3.9	140.5 ± 3.8	139.5 ± 3.7	0.7
Eccen-LVH, n(%)	4 (31%)	7 (44%)	12 (57%)	0.018*
↑LVMI, n(%)	3 (23.1%)	14 (88%)	19 (90%)	0.038**

CP, chest pain; HPN, hypertension; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVED, left ventricular end diastolic diameter; LVES, left ventricular end systolic diameter; EF, ejection fraction; DT, deceleration time; Eccen LVH, eccentric left ventricular hypertrophy; LVMI, left ventricular mass index.
* $p \leq 0.05$ versus GI and GII.
** $p \leq 0.05$ versus GI.

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