ECG Abnormalities in Cirrhotic Pediatric patients

Thesis

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Abstract

The study included 30 patients diagnosed to have liver cirrhosis due to any aetiology by biopsy and 30 clinicaly free children of the same age group were also included as a control group.

The diseased group was subjected to thorough history taking, clinical examination and laboratory investigation in the form of serum bilirubib and albumin, liver enzymes, prothrombin time and complete blood count; they also had abdominal sonar to detect ascites. Both cases and control group underwent resting 12 lead ECG assessments.

Key Words : ECG Abnormalities - Cirrhotic Pediatric patients

Contents

List of abbreviations

ALT	Alanine aminotranseferases
AMA	Antimitochondrial antibodies
ANA	Antinuclear antibodies
ANP	Atrial natriuretic Peptide
ASD	Atrial septal defect
AST	Aspartate amino transferases
AV	Atrio-ventricular
Bil D	Direct bilirubin
Bil T	Total bilirubin
BNP	B type natriuretic peptide
CBC	Complete blood count
CHF	Congestive heart failure
CO	Carbon mono oxide
CT	Computed Tomography
ECG	Electrocardiograph
EHBA	Extrahepatic biliary atresia
HB%	Haemoglobin percent
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HPS	Hepato pulmonary syndrome
LAH	Left atrial hypertrophy
LBBB	Left bundle branch block
LKM	Liver kidney microsomal antibodies
LV	Left ventricle
LVH	Left ventricle hypertrophy
No	nitric oxide
MRI	Magnetic Resonance Imaging
MRA	Magnetic Resonance Angiography
PELD	Pediatric End stage Liver Disease
PFIC	Progressive familial intrahepatic cholestasis
PWD	P wave dispersion
QTc	QT interval corrected to heart rate
QTd	QT interval dispersion
RAH	right trial hypertrophy
RSA	Respiratory Sinus Arrhythmia
RBBB	Right bundle branch block
RBCs	Red blood corpusles
RR average	Average RR intervals
RV	Right ventricle

RVH	Right ventricle hypertrophy
TIPS	Transjuglar Intrahepatic Porto systemic Shunt
SD	Standard deviation
SMA	Smooth muscle antibodies
SVT	Supraventricular tachycardia
TNFά	tumour necrosis factor ά
VSD	Ventricular septal defect
WBC	White blood cells
WHO	World Health Organization
WPW	Wolf-Parkinson-White syndrome

List of Figures

List of Figures (follow)

List of Tables

Introduction and Aim of the Work

Liver cirrhosis is a form of chronic liver injury that that represents an end stage of virtually any progressive liver disease ***(Hardy and Klienman, 2007).***

The prevalence of liver cirrhosis is difficult to estimate, because many persons with compensated cirrhosis do not exhibit signs or symptoms of the disease and because noninvasive studies lack sensitivity to detect cirrhosis at early stages. Moreover there is tremendous geographical variability of cirrhosis worldwide, depending on the prevalence of the causative factors such as viral hepatitis B and C, metabolic liver disease, iron overload, autoimmune liver disease. The incidence of cirrhosis is high in children with chronic hepatitis, especially of the autoimmune type (89%) followed by hepatitis B(32%). cirrhosis may occur early, irrespective of cause***(Vajro et al.,1990).***

Liver cirrhosis is associated with several cardiovascular abnormalities. Shortly after recognition of the hyperdynamic circulatory state in the liver, **Ma** and **Lee** studies in 1996 suggested that cardiac contractile response during increased demand was subnormal in cirrhotic patients. This finding resulted in the recognition that a unique form of high output cardiac dysfunction occurs in chronic liver disease.

The pathogenesis of cardiac dysfunction in liver disease is likely involves a number of abnormalities , alteration in cardiac ß adrenergic signaling***(Al Hamoudi et al, 2006)*** , decreased plasma membrane fluidity, increased cardiac nitric oxide production***(Baik et al, 2005)***, elevated circulatory level of catecholamines ***(Milani et al,2006)***, have all been suggested to contribute to cardiac dysfunction.

Clinically cardiac dysfunction is often mild or latent in cirrhosis a finding some have attributed to the after load reducing effect of systemic vasodilatation that decreased cardiac work ***(Mandell and Tsou, 2008).***

Electrophysiological abnormalities including prolonged repolarization and impaired cardiac excitation-contraction coupling have been demonstrated in cirrhotic patients ***(Tervesani et al, 2003).***

QT interval prolongation is currently considered one of the electrophysiological abnormalities of the cirrhotic cardiomyopathy, which significantly affects the hemodynamic homeostasis in decompensated cirrhosis. QT interval prolongation is common in cirrhosis, irrespective of its etiology ***(Zambruni et al, 2008).***

Delayed cardiac repolarization may adversely affect survival under stressful conditions challenging heart function, such as sepsis, bleeding or major surgery including liver transplantation (***Zambruni et al., 2008).***

The aim of the present study was to detect the prevalence and types of ECG abnormalities among cirrhotic pediatric patients. we also aimed to delineate the correlation of these abnormalities to the aetiology, duration, severity of cirrhosis and other risk factors.

Patients and methods

Study Design:-

a prospective cross sectional study including 30 cirrhotic children and 30 age and sex matched clinically free children as a control group. The study was conducted in the time period between November 2008 and April 2009.

Study setting:-

The study group was recruited from the hepatology outpatient clinic at the Cairo university children hospital Parents of the participating children gave their consent to the study. .

Inclusion criteria:

* Cirrhosis of any etiology ,diagnosis of cirrhosis is done by liver biopsy

* Age rage between 4 months and 14 years

* Both sexes are included .

Exclusion criteria:

* Congenital heart disease.

* Acute superimposed illness.

* Hepatorenal syndrome.

* Other system organ disease.

* Antiarrhythmic drug intake

*Electrolytes imbalance

Methods

All cases and controls were subjected to full history taking, examination, and standard 12 ECG and laboratory investigations.

History taking

All the infants underwent thorough history taking and examination. The history includes the following:

* Personal history with special reference to age and sex.

* Complaint

* History of present illness included:

1- Onset, course and duration.

2- Age of onset

3- Presence of jaundice, colour of urine and stools. Abdominal distension, haematemesis, melena, bleeding from orifices, bleeding under the skin, pruritus, skin rash, arthritis, medications, fainting attacks, palpitations and other system affection symptoms.

4- Past history with emphasis upon total parental nutrition, incubator care, previous hospitalization, surgical intervention, blood transfusion and medications.

5- Family history of consanguinity, similar condition in the family and history of previous abortions and sib death.

6- Developmental history.

7- Nutritional history.

8- Vaccination history.

Examination:

Each child was examined thoroughly. The general examination included:

* Vital signs: heart rate, blood pressure, temperature and respiratory rate.

* Anthropometric measures.

* Examination for jaundice ,pallor .lower limb edema ,purpuric eruptions and clubbing

* Chest examination

* Full cardiac examination to detect any structural cardiac anomalies.

* Abdominal examination (inspection, palpation, percussion) to detect dilated abdominal wall veins, organomegaly, ascites and cutaneous manifestations of liver disease (spider angiomas, vascular spiders, palmar erythyma and xanthomas).

Laboratory investigations:

Children underwent:

* Complete blood count.

* Total and direct bilirubin level estimation.

* Aspartate and alanine aminotransferases (AST and ALT).

* Alkaline phpsphatase.

* Prothrombin time and concentration.

* TORCH screening, autoantibodies i.e.: antinuclear antibodies(ANA),smooth muscle antibodies (SMA), antimitochondrial antibodies(AMA), liver kidney microsomal antibodies (LKM) according to clinical setting.

Abdominal Sonography:

Abdominal Sonography to detect:

* Liver, spleen and kidney sizes.

* Omental thickening and other signs of portal hypertension.

* Ascites.

Percutaneous liver biopsy

As a prerequisite for inclusion ,result were recruited from the patients' charts

Upper endoscopy

Upper endoscopy was performed in selected cases.

Standard 12 lead ECG:

A standard 12 lead ECG was performed during sinus rhythm. All routine measurements were taken(heart rate , rhythm, axis, P wave amplitude and duration, PR interval, QT interval, QRS complex(voltage amplitude of R and S waves in V1&V6. the presence of respiratory sinus arrhythmia was determined

.

Particular stress was done on

P wave :-

P wave max, min and P wave dispersion

The onset of pwave was defined as the junction between the isoelectric line and the beginning of the P wave deflection. The offset of the P wave was defined as the junction between the isoelectric line and the end of P wave deflection.

Electrocardiogram with measurable Pwave is less than 9 ECG leads were repeated. P wave dispersion was calculated as the difference between the max and min P waves. (***Dilareris et al, 1998)***

QT intervals

QT intervals were measured in all 12 leads if possible , but in at least six leads. The average of three consecutive beats for each lead was taken whenever possible.

The QT interval was measurd from the beginning of the QRS complex to the end of the T wave . the end of the T wave was defined as the return of voltage to the isoelectric line . if a U wave was present measurements were taken to the nadir of the curve or notch between the T and U waves.

QT intervals were corrected for heart rate using Bazett's formula($QTc=QT/\sqrt{RR}$).

QT intervals reported as the maximum QT found on the 12 lead ECG (QTmax).