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RESEARCH ARTICLE

A CASE OF BRADYCARDIA IN A CHILD

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ABSTRACT

A 6 months old male infant came with lethargy and poor feeding. On evaluation there was bradycardia. Positive family history of sibling death and hearing loss in another sibling were present. ECG shows long QT interval and B/L sensori-neural hearing loss. On the basis of long QT and hearing defect a diagnosis of Jervell- Lang - Nielsen syndrome was suspected and child was started on beta blocker therapy. epicardial pacemaker insertion was done and child was given discharge.

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INTRODUCTION

A 6 months old male infant referred from outside hospital for lethargy and poor feeding for 1 day. There was no history of fever, vomiting, failure to gain weight or excessive cry during micturition. There was no history of sudden loss of consciousness or seizures. The child is third born of third-degree consanguineous parents, delivered by LSCS as previous delivery was LSCS. Antenatal and neonatal period was uneventful. His developmental milestones were normal. There is a family history of unexplained sibling death at 6 months of age. Another sibling had bilateral hearing loss since birth. On examination child was lethargic, airway was clear, respiratory rate was 38/min, with bilateral normal vesicular breath sounds, pulse rate was 62/min, regular and all peripheral pulses were palpable, CRT<2 sec, B.P. 86/50 mm Hg. There was no murmur on auscultation and no facial dysmorphism.

Investigation revealed Hb 7.2,TC10400, Platelets 158000,Na134,K 4.3,Mg 2.25, Ca 9.94, ABG:pH7.38/ $pCO_2$30/$pO_2$96/$HCO_3$17.7/BE-7.4/Lactate3.3/ Na134/ K4.4/ Ca1.26/Hb7.8/Glc85, ECG revealed bradycardia with prolonged QT interval, echocardiography revealed structurally and functionally normal heart.

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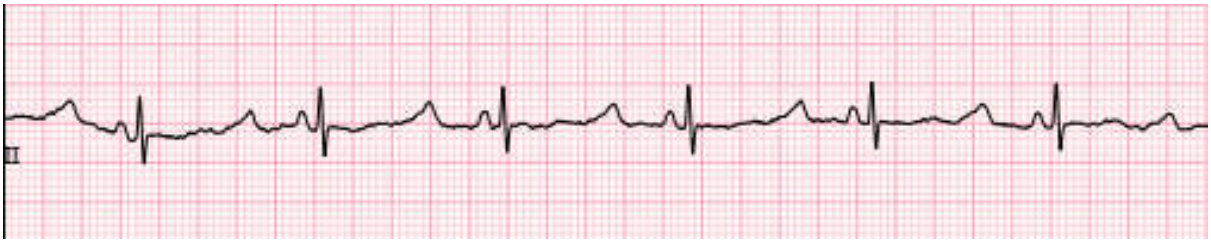
Hearing assessment done (BERA) showed bilateral sensorineural hearing loss. Based on the ECG and the presence of hearing loss diagnosis of Long QT Syndrome (Jarvell Lange Syndrome) was suspected**.** Results of genetic studies awaited. For bradycardia Propranolol was started @3 mg/kg/day in three divided doses, but dose could not be increased in view of bradycardia. Epicardial Pacemaker Insertion was done and child was given discharge on Propranolol.

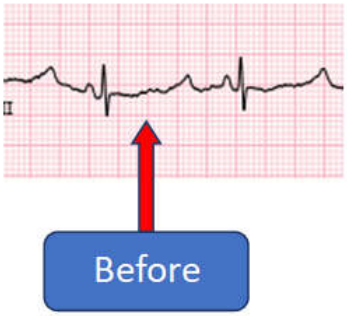
DISCUSSION

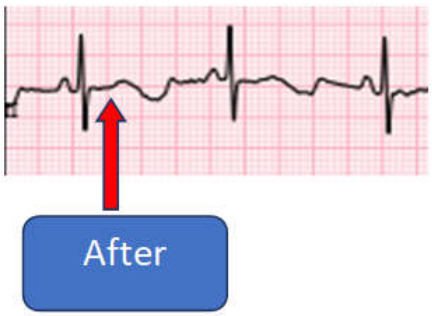
Long QT Syndrome (LQTS) is a disorder of myocardialrepolarization characterized by a prolonged QT interval on the ECG and ventricular arrhythmias, usually torsades-de-pointes, that may result in sudden death. First reported in 1957 by Anton Jervell and Fred Lange Nielsen. Disease prevalence is estimated to be from 1 in 5,000 to 1 in 20,000. Jervell and Lange-Nielsen (1957) first described families in Norway with this syndrome, which is associated with congenital deafness, syncopal spells, and a family history of sudden death(1). The disease is transmitted by Autosomal recessive manner. Romano-Ward syndrome, reported independently by Romano and colleagues in Italy (1963)(2) and Ward in Ireland (1964)(3). In this syndrome all the features of Jervell and Lange-Nielsen syndrome will be present but the patients will have normal hearing. This disease is much more common and inheritance is autosomal dominant**.** Anderson-Tawil syndrome is sometimes designated as LQT7, in which the QU interval, rather than the QT interval, is **prolonged**, along with muscle weakness (periodic paralysis),



Long QT interval in ECG

ECG before and after Propranolol showing reduced QT interval





ventricular arrhythmias, and developmental abnormalities. Timothy syndrome is associated with webbed fingers and toes and long QTmeasurement (4)

Acquired causes of long QT syndrome: Prolongation of the QT interval can be caused by a numberof drugs, electrolyte disturbances, and other underlying medicalconditions. A similar ionic mechanism may be involved asin congenital LQTS. Those individuals who manifest acquired long QT syndrome are believed to be genetically predisposed for the condition.

Drug induced:

Antibiotics-Erythromycin, Clarithromycin, Telithromycin, Azithromycin, Trimethoprim-Sulfamethoxazole
Antifungal-Fluconazole, Itraconazole, Ketoconazole
Tricyclic Antidepressants

Class IA and III antiarrhythmic; so, they should be avoided.

Electrolyte Abnormalities: Hypokalemia, Hypocalcemia, Hypomagnesemia

Others: Complete AV Block, Sick Sinus Syndrome, Anthracycline Toxicity, CCF, Myocarditis, Hypothyroidism
The family history is positive in 60% of patients.Deafness is present in 5%(5). Presenting symptoms may be syncope (26%), seizures (10%), cardiac arrest (9%), presyncope, or palpitation (6%) (5).

Majority of these symptoms occur during exercise or with emotion(5). They usually manifest in the first decade but can present by the end of the second decade of life. The first manifestation may be cardiac arrest which can occur in the setting of intense adrenergic arousal, intense emotion, and during or after rigorous exercise. Swimming appears to be a particular trigger.

The ECG shows the following:

- A prolonged QT interval with a corrected QT interval (QTc) usually longer than 0.46 second
- Abnormal T-wave morphology (bifid, diphasic, or notched)
- Bradycardia (20%), second-degree AV block, multiform Premature Ventricular Complexes, and monomorphic or polymorphic VT (10%–20%) may be present. All of these ECG findings are considered risk factors for sudden death.

Initial diagnostic strategy

- History of presyncope, syncope, seizure, or palpitation and family history are carefully examined.
- Causes of acquired LQTS are excluded.
- ECG is examined for the QTc interval and morphology of the T waves. ECGs are also obtained from immediate family members.

Diagnostic dilemma:

Holter monitoring(6)
Exercise testing(6)
Epinephrine test
Genetic testing

Management: Avoid all medications causing long QT

- Catecholamines and sympathomimetics should be avoided
- No competitive sports; avoid swimming also
- Parents education regarding compliance of medications

Medical Management:

- Beta Blockers: Propranolol;Beta-blockers, at full blocking dose, represent the therapy of choice and are greatly effective in reducing the mortality (from 73 per cent to 6 per cent).

- Cardiac pacemakers(7)
- Left cardiac sympathetic denervation: If syncopal attacks are not eliminated by the medical therapy, then the ablation of the left stellate ganglion along with the first thoracic ganglia is the most rational and specific therapy(8)
- Implantable Cardioverter Defibrillator (ICD)

Prognosis:

The prognosis is very poor in untreated patients, with an annual mortality rate as high as 20% and 10-year mortality rate of 50%. Beta-blockers may reduce mortality to some extent, but they do not completely protect patients from sudden death. An ICD appears promising in improving prognosis.

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