GW23-e0569

LONG QT SYNDROME AND TORSADES DE POINTS IN A PATIENT WITH ACUTE HEPATITIS E VIRUS INFECTION: AN UNUSUAL CASE

doi:10.1136/heartjnl-2012-302920n.6

[1]Zhou Aiqin, [1]Xie Dongming, [1]Tian Kejun, [2]Zhou Yun, [1]Zhou Aiqin. *[1]Department of Cardiology, The First Affiliated Hospital of Gannan University, Ganzhou,341000, China; [2]Department of Gastroenterology, The First Affiliated Hospital of Gannan University, Ganzhou341000, China*

Objectives One patient with long QT syndrome, torsades de points and acute hepatitis E virus infection are described. ECG demonstrated sinus bradycardia with abnormal towering U-wave in chest leads and a QT interval of 511 ms (QT_C=550 ms). She was treated with lidocaine, electrical conversion, temporary and permanent pacemaker and remains well 6 months later. This report, however, provides questionable evidence, in one case, of an association between the long QT syndrome and acute hepatitis E virus infection.

Methods An unusual case.

Results

1. Clinical Data: A 62-year-old woman was admitted with a 7 days history of fever (37.6°C), jaundice and nausea and 3 days history of repeated syncope. She didn't have history of syncope and family history of long QT syndrome (LQTS), consanguinity, or sudden cardiac death (SCD). Her initial ECG demonstrated sinus bradycardia with abnormal towering U-wave in chest leads and a QT interval of 511 ms (QT_C=550 ms). Cardiac structure and function were normal by echocardiography. Liver echography showed a mild splenomegaly and hepatauxe. Laboratory investigation revealed a low serum sodium of 124 mmol/ l and a normal serum potassium of 3.65 mmol/l. On admission, her alanine amino transferase level was elevated (226 IU/l), whereas her gamma-glutamyl transferase level

(165 IU/l), total bilirubinemia (371.8 μmol/l), direct bilirubin (193.5 μmol/l) and indirect bilirubin (178.3 μmol/ l) were concomitantly high. Total serum protein and serum globulin were within usual ranges (61.1 g/ l, 35.4 g/l, respectively). In contrast, a severe Hypoalbuminemia was observed (serum albumin, 25.7 g/l). She tested positive for HEV RNA and anti-HEV antibodies. Serological testing for hepatitis A, B, and C, cytomegalovirus, and HIV were negative or showed past immunisation. No autoimmune hepatitis-associated antibodies were found. She was diagnosed with LQTS, torsades de points (TdP), acute viral hepatitis E, hyponatremia, hypoalbuminemia. The patient suffered a cardiac arrest due to TdP requiring resuscitation on the admission day. She had many further non-sustained episode. She was treated with lidocaine, electrical conversion and was immediately catheterised for temporary transvenous ventricular pacing at the bedside. Sodium Chloride, Magnesium sulphate and potassium were administered. The patient had received compound glycyrrhizin as well as reduced glutathione sodium, methylprednisolone, albumin, and blood plasm at the same time. The pacing rate was set to 90 beats/min and gradually dropped to 70 beats/min when she remained stable 2 days later. Syncope recurred when the pacemaker was not working due to dislocation at week 4 and at week 8. Transaminase levels and bilirubinemia were slowly normalised at week 9. ECG demonstrated sinus bradycardia with normal T-wave and U-wave and a QT interval of 450 ms (QT_C=411 ms) when the liver function nearly returned to normal. She was offered an ICD which she declined. A permanent AAI pacemaker (ST.JUDE 2406L) and unipolar endocardial lead (ST.JUDE 1642T/52 cm) were implanted surgically at week 10. Pacing rate was 70 beats/min. She was discharged from hospital 3 months later and the syncope never occurred during the 6 months follow-up.

Conclusions

2. Discuss: The clinical and electrocardiographic features of LQTS are stereotypic. Abnormal prolongation of the QT interval identifies patients at increased risk for TdP as well as giant, abnormal T–U waves as seen in our patient. In this case, the patient is female, ECG demonstrated sinus bradycardia, and laboratory investigation revealed hyponatremia and abnormal liver function. The patients had no obvious structural heart disease and history of administration of QT prolongation drugs. She was diagnosed with acute viral hepatitis E. The relationship of acute viral hepatitis E and LQTS have not been reported. It remains unclear whether acute hepatitis E virus infection itself was the cause of QT prolongation, or just a coincidence. In this case, the QT interval shortened but didn't return to normal after correction of the liver function, so the genetic LQTs is also possible. The further scanning of the gene has an important significance for diagnosis and differential diagnosis. An individual's response to QT-prolongation upon exposure severe disease depends to a certain extent on genetic disposition controlling intrinsic myocardial properties or signalling pathways. Sustained bradycardia is associated with long-QT syndrome and TdP in human beings. The patients had significant sinus bradycardia. It is reported that TdP is pause-dependent in most of LQTs patients (particularly adult females). The beneficial effects of pacing in high risk LQTS patients probably relate to the prevention of bradycardia and pauses and the shortening of long QT interval. Syncope recurred when the pacemaker was not working due to dislocation in our case. In patients with LQTS, ICD implantation for secondary prevention of SCD is a class I indication. ICDs are thought to be the most effective treatment in the prevention of arrhythmic SCD in LQTS but our patient declined. β-blockers remain first-line treatment for LQTS, however, our patient has contraindication during hospitalisation. In summary, factors predisposing to QT-prolongation and higher risk of TdP include older age, female sex, slow heart rate, electrolyte abnormalities, organic diseases, genetic predisposition and so on. Our awareness and understanding of the mechanisms of LQTS will help to identify patients at risk and reduce their exposure to risk factors.