TORSADE DE POINTES ASSOCIATED WITH ENCEPHALITIS*

*Dursun Alehan MD***, Naci Ceviz MD***, Alpay Çeliker MD*****

SUMMARY: Alehan D, Ceviz N, Çeliker A. (Cardiology Unit, Department of Pediatrics, Hacettepe University Faculty of Medicine, Ankara, Turkey). Torsade de pointes associated with encephalitis. Turk J Pediatr 1999; 41: 395-398.

Torsade de pointes is a polymorphic ventricular tachycardia. Causes of torsade de pointes are well described. Although intracranial disease can produce dramatic electrocardiographic (ECG) changes, we are not aware of previous cases with torsade de pointes and encephalitis. We report a case with encephalitis who developed torsade de pointes, and was treated with temporary ventricular pacing and magnesium infusion.

Key words: torsade de pointes, encephalitis.

Torsade de pointes is a polymorphic ventricular tachycardia, characterized by a continuous twisting of the QRS axis around an imaginary baseline on the electrocardiogram (ECG)[1]. Causes of torsade de pointes are well described[1,2]. Although we know that intracranial disease can produce dramatic ECG changes[3], we are not aware of the concurrence of torsade de pointes and encephalitis in a previous case. We report herein a case with encephalitis who developed torsade de pointes in the absence of a known cause, and was treated with temporary ventricular pacing and magnesium infusion.

Case Report

A two-year-old girl was admitted to the Pediatric Emergency Unit with fever, diarrhea, vomiting and decreased consciousness over the previous two days. Physical examination revealed an unconscious child with hyperactive deep tendon reflexes, bilateral positive Babinski reflex, signs of severe dehydration, and normal funduscopic findings. During physical examination the patient developed a generalized seizure which terminated after intravenous diazepam infusion. In view of the state of unconsciousness, positive meningeal irritation signs and the seizure, a computed brain tomography was obtained and a lumbar puncture was performed. Computed tomography revealed minimal brain edema. No view consistent with mass or hemorrhagic lesion was determined. Cerebrospinal fluid (CSF) was clear. Biochemical examination of CSF was in normal limits, and 22 lymphocytes per mm^3 were detected on microscopic

* From the Cardiology Unit, Department of Pediatrics, Hacettepe University Faculty of Medicne, Ankara.

** Associate Professor of Pediatrics and Pediatric Cardiologist, Hacettepe University Faculty of Medicine.

*** Pediatrician and Fellow in Pediatric Cardiology, Hacettepe University Faculty of Medicine.

**** Professor of Pediatrics and Pediatric Cardiologist, Hacettepe University Faculty of Medicine.

examination. Bacterial and fungal cultures did not reveal an agent, and the culture for tuberculosis was also negative. We could not perform any diagnostic study for viruses, but considering CSF findings and gastroenteritis, we assumed that encephalitis was probably viral in origin. ECG showed a sinus rhythm with a heart rate of 160 beats per minute, normal QRS axis, and no signs of ventricular enlargement. PR and QT intervals were in normal limits according to the age and heart rate. Echocardiographic findings were normal. Serum sodium, potassium and calcium levels were in normal limits. Blood pH was 7.20 and HCO_3 was 5 mEq/L. After the proper $NaHCO_3$ infusion, pH reached normal limits. For the following six days, the patient was clinically stable and there were no additional problems except those relevant to meningoencephalitis. On the 7^{th} day of admisison, arrhythmia was detected during monitorization and ECG revealed torsade de pointes (Fig. 1). During the episode, serum K^+ was 3.2 mEq/L, Ca^{++} 9 mEq/L and Mg^{++} 2 mEq/L. A temporary ventricular pacemaker, with a rate of 100 beats per minute, was inserted and ventricular tachycardia stopped. Propafenone (300 mg/m^2) was started for prevention of recurrences. Despite temporary ventricular pacemaker and propafenone, torsade de pointes episodes recurred but for shorter durations than previous ones. Thus, intravenous magnesium sulfate injection at a bolus of 2 g, followed by a continuous infusion of 10 mg/min was performed. Following the magnesium sulfate infusion, no new episode was observed. The pacemaker was removed on the 13^{th} day of the insertion but oral propafenone was continued. Two months after discharge, the patient had sequelae of encephalitis but no episode of torsade de pointes was observed on 24-hour Holter monitoring.

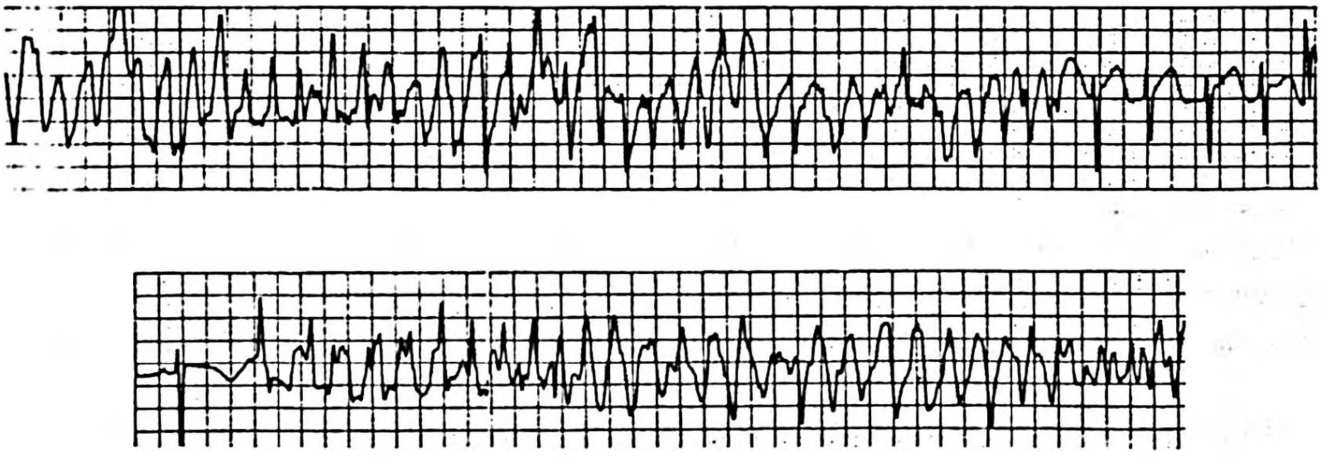

Fig. 1: Electrocardiographic tracings from two separate attacks showing the torsade de pointes.

Discussion

Prolongation of ventricular repolarization associated with the development of torsade de pointes can be observed in many clinical conditions, commonly referred to as prolonged QT syndromes. These syndromes are divided into two

groups: 1. idiopathic long QT syndrome and adrenergic-dependent torsade de pointes, and 2. acquired prolonged QT syndromes and pause-dependent torsade de pointes[1,2]. Jackman et al.[3] gives a detailed classification of this clinical condition. Since we could not demonstrate a known cause for torsade de pointes in our patient, we thought it might be due to encephalitis.

Intracranial diseases, most notably subarachnoid hemorrhage, but also intracerebral hemorrhage, cerebrovascular occlusive disease, trauma, and infection, can produce dramatic ECG changes. Hersch[4] determined electrocardiographic changes in adult patients with subarachnoid hemorrhage, pyococcal meningitis, intracranial space-occupying lesions, labor pneumonia, and in normal adults. QT prolongation was present in nine (45%) patients with subarachnoid hemorrhage, but not in patients with meningitis. It has been suggested that QT prolongation in patients with intracranial hemorrhage may be associated with severe ventricular arrhythmias, based on the observation of torsade de pointes in two of 72 consecutive patients with intracranial hemorrhage[3]. Although there are several reports about torsade de pointes that has developed during the course of intracranial hemorrhage, especially subarachnoid hemorrhage, we have no information about the concurrence of intracranial infections and torsade de pointes. And, to our knowledge, this is the first report of torsade de pointes associated with encephalitis. In the previous reports of torsade de pointes associated with subarachnoid hemorrhage, treatment consisted of either propranolol or pharmacologic blockade of the stellate ganglion with lidocaine. As a result, adrenergic pathways may play a role in torsade de pointes in association with intracranial diseases[3]. The most effective treatment in torsade de pointes related to acquired long QT syndromes is rapid electrical pacing of the heart (90-100 beats/min). In addition, magnesium sulfate, as a safe, rapid, and easily applied treatment, may be effective in suppressing recurrence of torsade de pointes, regardless of plasma magnesium levels[1,5,6].

We were able to treat the torsade de pointes in our patient with temporary ventricular pacing and intravenous magnesium sulfate infusion. After discontinuation of pacing and magnesium infusion, the episodes did not recur and the patient was discharged with the sequelae of encephalitis.

In conclusion, torsade de pointes may develop during the course of encephalitis, a clinical picture which has not been described before.

REFERENCES

1. Napolitano C, Priori SG, Schwartz PJ. Torsade de pointes: mechanisms and management. Drugs 1994; 47: 51-65.

2. Leenhardt A, Coumel P, Slama R. Torsades de pointes. J Cardiovasc Electrophysiol 1992; 3: 281-292.

3. Jackman WM, Friday KJ, Anderson JL, Aliot EM, Clark M, Lazzara R. The long QT syndromes: a critical observations and a unifying hypothesis. Prog Cardiovasc Dis 1988; 31: 115-172.
4. Hersch C. Electrocardiographic changes in subarachnoid haemorrhage, menigitis, and intracranial space-occupying lesions. Br Heart J 1964; 26: 785-793.
5. Roden DM. Torsade de pointes. Clin Cardiol 1993; 16: 683-686.
6. Mansmann HC. Consider magnesium homeostasis: III: cytochrome P450 enzymes and drug toxicity. Ped Asthma Allergy Immunol 1994; 8: 7-28.