A severely obese patient surviving cardiopulmonary arrest and exhibiting an interesting relationship between excessive weight loss and electrocardiographic QT interval prolongation

Koichiro Shinozaki, Kenichi Matsuda, Shigeto Oda, Hidetoshi Shiga, Masataka Nakamura, Noriyuki Hattori, Hiroyuki Hirasewa

Abstract: We report a patient with severe obesity who suffered cardiopulmonary arrest (CPA) after drastic weight loss but successfully survived with cardiopulmonary resuscitation and critical care in the ICU. We describe the patient's care and discuss the relationship between drastic weight loss and secondary prolongation of corrected QT interval (long QT syndrome) with development of torsades de pointes (Tdp). The corrected QT interval (QTc) observed upon the first visit of the patient to the outpatient unit was 0.475 sec, with a body mass index (BMI) of 67.5. QTc was prolonged to 0.507 sec after drastic weight reduction to achieve a BMI of 60.8. That was further prolonged to 0.608 sec upon ICU admission following resuscitation. Postresuscitation treatment in the ICU was successful, and she was discharged from the ICU on the 7th day. Strict medical weight control after discharge from the ICU successfully shortened QTc within the normal range. The relationship between BMI and QTc observed in the present case suggests that severe obesity and excessive weight loss caused prolongation of QTc and CPA. These findings contribute to elucidation of the pathology of sudden death in the severe obese population.

Key words: ① cardiopulmonary arrest (CPA), ② obesity, ③ QT interval

Introduction

Sudden death in healthy individuals has recently gained public attention and has been ascribed to lifestyle-related diseases. Life-style-related diseases, represented by hypertension, diabetes mellitus, obesity, and hyperlipemia, which comprise the so-called "deadly quartet" and exacerbate arteriosclerosis and increase the risk of ischemic heart and cerebrovascular diseases. Accordingly, the importance of personal life-style in primary prevention of various diseases has recently been emphasized.

Obesity is a risk factor for acute pulmonary embolism and sleep apnea syndrome. Recently, obesity has also been proven to be a cause itself of prolongation of QT interval and sudden death.

We report a patient with severe obesity who suffered cardiopulmonary arrest (CPA) after drastic weight loss but successfully survived with cardiopulmonary resuscitation and critical care in the ICU. The objective of this case report is to describe the clinical course of the patient and to examine the relationship between drastic weight loss and secondary prolongation of corrected QT interval (long QT syndrome) with development of torsade de pointes (Tdp).

Case report

Patient: A woman aged 40.

Past clinical history/family history: Non-contributory.

Recent clinical history: She had developed severe obesity for 15 years before the present episode but had not responded to any medical treatment of it. At her first visit to the obesity clinic of a university hospital, she was 145 cm tall but weighed 142 kg, with a body mass index (BMI) of 67.5. She underwent a drastic weight reduction regimen of her own design for approximately one month to improve her condition (to body weight 127 kg, BMI 60.4). She was then admitted to the university hospital in March 2002 for surgical treatment of obesity including gastric refeeding and liposuction. No electrolytes or hematological abnormali-
ties were found on admission. She consumed a 1,200 kilocalorie per day diet with normal hospital food, without medication for diet or any other purpose. After breakfast on the 5th hospital day, she was found in CPA in the women’s bathroom of the hospital. She was urgently admitted to the ICU after return of spontaneous circulation via ACLS (advanced cardiovascular life support) with chest compression, intubation, and administration of epinephrine, without defibrillation. Laboratory findings on ICU admission revealed no abnormalities other than mild increases in WBC, AST, ALT, LDH, creatinine, CK, and blood glucose level with a decrease in triglyceride level caused by the stress associated with CPA (Table 1).

**Clinical course:** Figure 1 shows the clinical course during the first 24 hours after ICU admission. Controlled body temperature is shown in this figure. Hypothermia was induced with cooling blankets. The goal of temperature control was 36°C, so-called normothermia. We were unable to cool her further, since this might have induced arrhythmia. The electrocardiogram recorded upon ICU admission revealed an extremely prolonged QT interval, with a QTc value of 0.608 sec. QTc is an index of QT interval [10]. Thirty episodes of ventricular arrhythmia noted during this period, which included ventricular tachycardia and ventricular fibrillation, were treated 26 times by defibrillation and by antiarrhythmics such as isoprenaline, lidocaine, and Mg²⁺. No pacing therapy for ventricular arrhythmia was performed due to good response to defibrillation and difficulties with procedures required for pacing therapy due to her severe obesity. A typical electrocardiogram exhibiting Tdp during an episode of ventricular arrhythmia is shown in Figure 1. It demonstrates polymorphic ventricular tachycardia characteristic of Tdp with prolonged QT intervals. Diagnostic imaging results, including those of CT, coronary angiography, echocardiography, and pulmonary blood flow scintigraphy obtained on ICU admission were negative for cerebrovascular disease, ischemic heart disease, cardiomyopathy, and acute pulmonary thromboembolism. She was therefore diagnosed with long QT syndrome [11].

Figure 2 summarizes the clinical course throughout the entire ICU stay. Careful respiratory and hemodynamic management was initiated immediately upon ICU admission, in addition to the normothermia for cerebroprotection using cooling blankets as described above. This resulted in disappearance of ventricular arrhythmia from the 2nd ICU day and recovery of consciousness, enabling weaning from a ventilator on the 3rd ICU day and discharge from the ICU on the 7th ICU day. After leaving the ICU, the patient underwent strict medical weight control to assure slow weight reduction in the general ward, which decreased her body weight from 127 kg to 105 kg. She was finally discharged from the hospital without disorder of consciousness on the 110th hospital day.

Figure 3 illustrates the relationship between BMI and QTc observed during the entire hospital stay. A prolonged QTc of 0.475 sec was observed upon her first visit to the outpatient unit, with a BMI of 67.5 indicating severe obesity. The QTc was prolonged to 0.507 sec after the patient's own weight-reduction regimen, with a drastic fall in BMI to 60.8. QTc was extremely prolonged to 0.608 sec upon ICU admission following resuscitation. However, the strict medical weight control to assure slow weight reduction performed after discharge from the ICU successfully shortened her QTc to within the normal range. The electrocardiograms obtained over a part of the clinical course are shown in Figure 4.

**Discussion**

We have described a case of severe obesity with secondary long QT syndrome, development of Tdp, and occurrence of CPA.

The results of electrocardiography, CT, coronary angiography, echocardiography, and pulmonary blood flow scintigraphy suggested that the cause of CPA in the present case might have been ventricular arrhythmia, and excluded the possibility of cerebrovascular disease, coronary ischemic disease, cardiomyopathy, and acute pulmonary thromboembolism. Furthermore, the electrocardiogram recorded upon ICU admission demonstrated prolonged QT intervals and polymorphic ventricular tachycardia characteristic of Tdp [11]. The electrocardiogram established the diagnosis of long
Clinical course during first 24 hours after ICU admission

The electrocardiogram recorded upon ICU admission revealed an extremely prolonged QT interval, as indicated by a QTc value of 0.608 sec. Thirty episodes of ventricular arrhythmia, such as ventricular tachycardia and ventricular fibrillation, noted during this period were treated by defibrillation 26 times as well as antiarrhythmics. A typical electrocardiogram during Tdp is shown in this figure. It demonstrated polymorphic ventricular tachycardia characteristic of Tdp with prolonged QT intervals. She was therefore diagnosed long QT syndrome based on these electrocardiograms.

ECG, electrocardiogram; BP, blood pressure; HR, heart rate; BT, body temperature; VT, ventricular tachycardia; Vf, ventricular fibrillation.

Clinical course throughout the entire ICU stay

Careful respiratory and hemodynamic management was begun immediately upon ICU admission, as well as body temperature control for cerebroprotection using a cooling mat. This resulted in disappearance of ventricular arrhythmia from the 2nd ICU day and recovery of consciousness, and enabled extubation on the 3rd ICU day and final discharge from ICU on the 7th ICU day.

GCS, Glasgow coma scale; BP, blood pressure; HR, heart rate; VT, ventricular tachycardia; Vf, ventricular fibrillation; BT, body temperature.
Fig. 3  Relationship between BMI and QTc during the entire hospital stay
A prolonged QTc of 0.475 sec was observed upon her first visit to the outpatient unit with a BMI of 67.5 indicating severe obesity. The QTc was prolonged to 0.507 sec after a weight-reduction regimen of her own design to achieve a drastic fall to a BMI of 60.8. It was extremely prolonged to 0.608 sec upon ICU admission following resuscitation. However, strict medical weight control to assure slow weight reduction conducted after discharge from ICU successfully shortened her QTc within the normal range.
BMI, body mass index; CPA, cardiopulmonary arrest.

QT syndrome. Thus, in the present case, it appeared that exacerbation of long QT syndrome resulted in Tdp and CPA.

Neither the patient’s clinical history nor her family history was useful in determining the etiology of her long QT syndrome. It appeared that her long QT syndrome was not hereditary electrocardiographic abnormality.

In the present case, the patient was not taking medications that could induce secondary long QT syndrome, nor were electrolyte abnormalities noted upon ICU admission (K, Mg, Ca). The minor abnormalities on hematological examination upon ICU admission were ascribable to CPA-related poor oxygenation. While the possibility of a relationship between glucose tolerance disorder and prolongation of QT interval has been suggested, no history of glucose tolerance disorder was noted in the present case. The observed increase in blood glucose level might therefore have been the result of CPA-related insults and not the cause of long QT syndrome.

ElGamal et al. proposed that degree of lipid accumulation and BMI are correlated with QT interval: the higher BMI is, the longer the interval. Their argument is based on a decrease in RR interval due to autonomic disorder. Pietrobelli et al. examined electrocardiograms in 30 asymptomatic obese subjects for comparison before and after a 7-day, low-calorie diet. They reported a significant decrease in QT interval after the diet and suggested the possibility of involvement of cardiac muscle hypertrophy in it. Corbi et al. demonstrated that plasma free fatty acid concentrations correlated with QT interval, and suggested a possible cause-effect relationship between autonomic disorder and QT interval prolongation. Although some other reports have also suggested that obesity prolongs QT interval and that weight reduction at a con-
trolled rate decreases QT prolongation\[10\], the reasons for these changes are still unclear.

It has long been known that drastic weight reduction by inappropriate dieting without replenishment of essential nutrients can prolong QT intervals and increase the risk of sudden death\[10\]. A recent report has suggested the possibility of involvement of cardiac muscle cell atrophy in these changes\[10\]. In addition, inhomogeneity in repolarization of each cardiac muscle cell associated with left ventricular atrophy has been reported to be involved in prolongation of the QT interval in sudden death of patients with anorexia nervosa\[10\].

In addition to obesity itself, the drastic weight reduction regimen adopted immediately prior to hospital admission may have further prolonged the QT interval in the present case. QT prolongation during the ICU stay was subsequently normalized by strict medical weight reduction after discharge from the ICU. The long QT syndrome in the present case thus appeared to have been caused by two factors, severe obesity and drastic weight reduction.

On the other hand, the direct trigger of the Tdp in the long QT syndrome in the present case is still unclear. One factor known to trigger Tdp in congenital long QT syndrome is a change in autonomic tone induced by gene-specific triggers such as mental stress, auditory stimulation, or emotional stress. Which of these is the actual trigger depends on the genotype of the patient\[10\]. In addition, QT intervals are reported to be prolonged after meals\[30\]. Considering these observations, the Tdp in the present case might have been triggered by factors such as time of day (early morning), food intake (breakfast), urination, defecation, or emotional stress.

Thus, the Tdp in the long QT syndrome in the present case was most probably due to multiple factors. Successful elimination of cardiac arrhythmia in a single ICU day despite observation of QT prolongation throughout the ICU stay may have been due to improvement of general condition as well as appropriate doses of antiarrhythmics.

Conclusion

The relationship between BMI and QTc observed in the present case suggests that severe obesity and excessive weight loss caused prolongation of QTc. Further investigation of the relationships between obesity and sudden death and severe cardiac arrhythmia may therefore be warranted, considering the recent increase in number of obese individuals due to changes in lifestyle. These findings will aid elucidation of the pathophysiology of sudden death in the severe obese population.

Part of this study was presented at the 30th Annual Meeting of the Japanese Society of Intensive Care Medicine, Sapporo, 2003.

References