부산대학교 어린이병원
Pusan National University Children's Hospital

P3-1106

Acquired long QT syndrome in a 14-year-old boy with panhypopituitarism

Yoo-Mi Kim, Jung Ho Seo, Young-Tak Lim, Chong Kun Cheon, and Su Young Kim

Department of Pediatrics, College of Medicine, Pusan National University Children's Hospital, Yangsan, Korea

Disclosure statement

The authors have nothing to disclose or conflicts of interests in this study.

Introduction

- Acquired QT prolongation can caused by electrolyte abnormality, myocarditis, cerebrovascular disease, drug intoxication and hormonal disorders such as hypopituitarism, hypothyroidism, and adrenal insufficiency.
- QT prolongation may lead to torsade de pointes a form of polymorphic ventricular tachycardia which can cause sudden cardiac death.

Case

Brief history

A 14-year-old boy with hypopituitarism after trans-sphenoidal surgery (TSS) due to suprasellar mass who manifested bradycardia and QT prolongation on electrocardiogram.This subject was complained of blurred vision and bilateral temporal hemianopsia for 1 month. Brain magnetic resonance image (MRI) revealed 3cm sized suprasellar mass and TSS was performed. Germinoma was confirmed by pathology, and he received scheduled chemotherapy. As the subject showed polyuria and urine was not concentrated during chemotherapy, he started desmopressin 0.1mg bid. Before the cocktail test was done, his heart rate was decreased to 35 beat per minute and blood pressure was 90/60 mmHg while he was sleeping. Since transient hypomagnesemia (1.4mg/dL) was noted once, magnesium was promptly corrected. After then, no electrolyte imbalance and hypoglycemia were noted, the subject's heart rate was not improved. His heart rate was not increasing and QT interval was prolonged to 580ms on E.K.G, so 0.025mg/kg/hr of isoproterenol, beta agonist, was started. There was no family history of arrhythmia. A two-dimensional transthoracic echocardiography was taken it showed normal left ventricular systolic function and normal cardiac structure. Anterior pituitary function test was done, resulting in panhypopituitarism.

Physical examination

Height: 143.1cm (-2.22standard deviation score (SDS))
Body weight: 40.3 kg (-1.34 SDS) Body mass index (BMI) : 19.7kg/m^2
Growth velocity =2.5cm/yr
No goiter, Tanner stage: B1 P1, and testicle volume 3cc/3cc

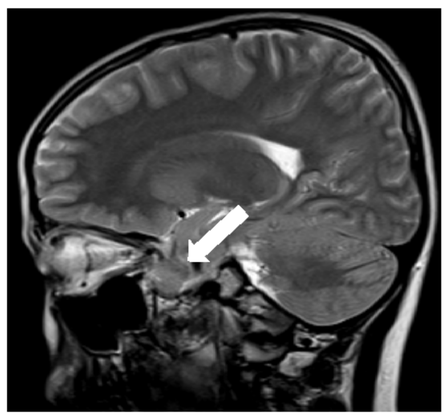

Fig. 1. A Sagittal T1-image of brain magnetic resonance image showing a 3 x 4.7 cm sized suprasellar tumor (white arrow). Pathologic analysis confirmed the diagnosis of germinoma.

Table 1. Endorine laboratory finding

Parameters (normal range)	At diagnosis (basal → peak)
GH (L-dopa test, peak>10ng/ml)	0.05→ 0.11
GH (insulin tolerance test, peak>10ng/ml)	0.03→ 0.1
TSH (0.7-6.4mIU/L)	0.15→ 0.8
Free T4 (0.8-2.2ng/dL)	0.67
ACTH (10-60pg/mL)	<5.0 → <5.0
Cortisol (5-25ug/dl)	0.56 → 1
LH (1-12 IU/L)	< 0.1→ 0.1
FSH (2-13 IU/L)	0.109 → 0.36
Testosterone (0.01-0.55 ng/ml)	< 0.2
IGF-1 (118.4-677.5 ng/mL)	34.13
IGFBP3 (3100-9500 ng/mL)	2850

GH, growth hormone; TSH, thyroid-stimulating hormone; ACTH, adrenocorticotropic hormone; LH, luteinizing hormone; FSH, follicle-stimulating hormone; ND, not done.

Table 2. Change of endocrine laboratory finding and electrocardiogram

Parameters (normal range)	At admission	Long QT	10 day after medication	8weeks later medication
TSH (0.7-6.4mIU/L)	ND	0.15	ND	0.001
Free T4 (0.8-2.2ng/dL)	ND	0.58	ND	1.29
ACTH (10-60pg/mL)	ND	<5	<5	<5
Cortisol (5-25ug/dl)	ND	0.56	ND	11.2
Heart rate (60-130/min)	75	53	101	60
QTc (350-450ms[1])	420	526	503	369

TSH, thyroid-stimulating hormone ACTH, adrenocorticotropic hormone; QTc, corrected QT interval; ND, not done.

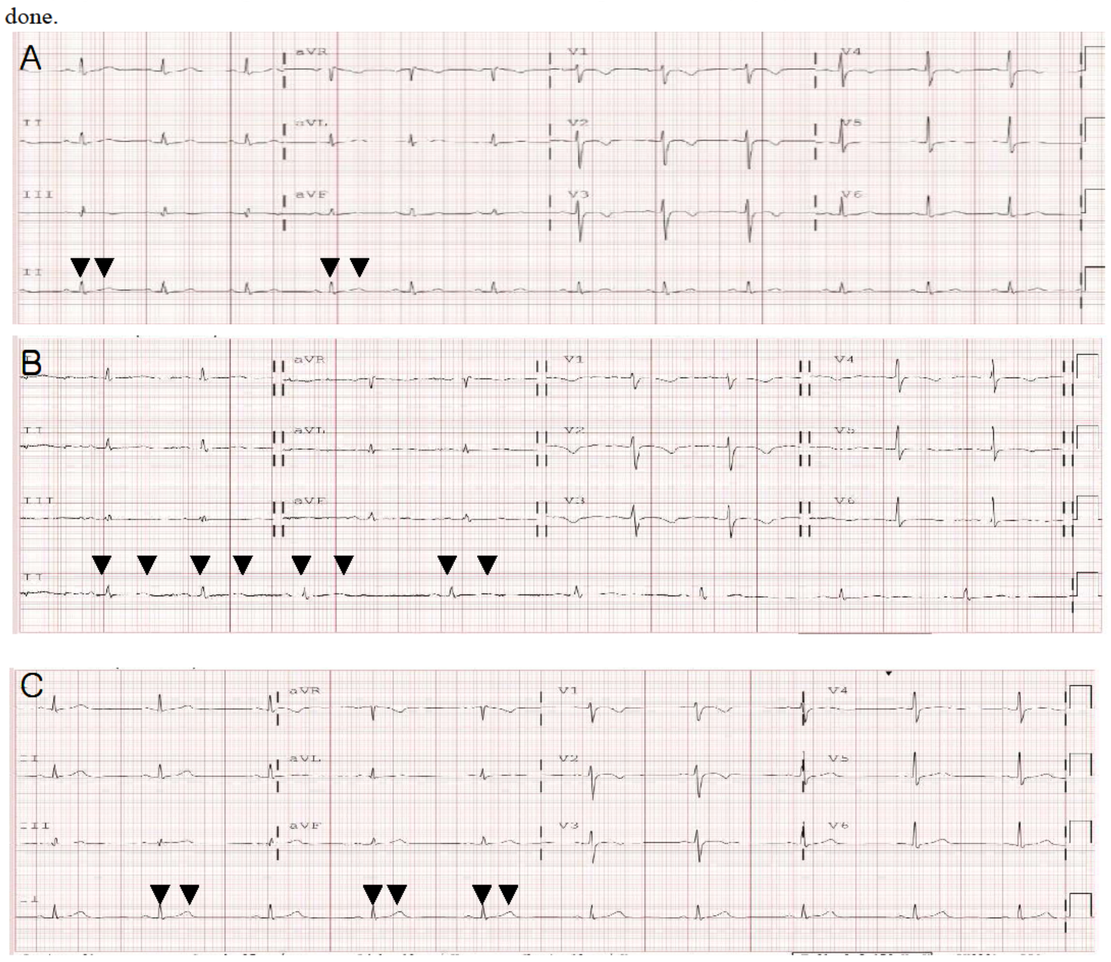

Fig. 2. Twelve-lead electrocardiogram (EKG) in patient. (A) Initial EKG showing normal sinus rhythm and QT interval and (B) significant ECG event with bradycardia and prolonged QT interval. (C) Eight weeks after starting the steroid and thyroid replacement therapy, electrocardiogram demonstrates normal QT intervals.

- After hormone replacement including hydrocortisone 5mg three times (10mg/m^2/d) and levothyroxine 50mcg per day, isoproterenol tapered off for five days and his heart rate was stable. Ten days after multiple hormone replacement, hormone levels were also corrected well and his vital sign and E.K.G were also improving. This subject's E.K.G showed a normal QTc range (369 ms) after 2months medication.

Discussion

- The mechanism of QT prolongation in hypopituitarism remains unclear
- Glucocorticoid insufficiency
  - Defect of modulating the ion channels of cardiac cells
  - ✓ Catecholamine release induced by hypoglycemia might cause arrhythmia
  - ✓ Hypomagnesemia induced by adrenal insufficiency
- Hypothyroidism
  - Decreasing cardiac contractility and heart rate
  - Slowing the conduction of electrical stimuli in the heart muscle

Conclusions

- This case highlights that hormonal disorders should be considered as a cause of arrhythmia or prolongation of QT intervals and this can be prevent and cured by appropriate hormone replacement therapy.

References

1. P. R. Rijnbeek, et al. European Heart Journal 2001;22:702–711.
2. Kang DG, et al. Korean Circ J 2013;43:340-342.
3. Arpaci D, et al. Case Rep Endocrinol. 2013;2013:989745.
4. Hajsheikholeslami F, et al. Int J Endocrinol Metab. 2013 Spring;11(2):117-9.

ESPE 2015
1106--P3
Pituitary
Yoo-Mi Kim
DOI: 10.3252/pso.eu.54espe.2015
Poster presented at: 54th Annual Meeting 1-3 October ESPE 2015 BARCELONA European Society for Paediatric Endocrinology
PosterSessionOnline.com