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Empty Sella Syndrome Presenting with Ventricular Tachycardia

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Abstract In some cases, Empty sella syndrome (ESS) is associated with pituitary deficiency. Although it is well known that pituitary hormones have effects on in many organs and systems, few studies have addressed the relationship between hypopituitarism and heart/arrhythmia. This unusual situation leads to discussion of the possible causes and management. We present a case of a 19-year-old male with an onset of cardiac arrhythmia was finally diagnosed as hypopituitarism. The patient recovered quickly by the means of hydrocortisone therapy. Most of pituitary hormones returned to normal, as well as no ventricular arrhythmia occurred any more. Good outcomes were observed that suggesting pituitary hormones may have effects on cardiac conduction system. However, the exact mechanisms need to be further studied.

Keywords: *Empty Sella Syndrome, hypopituitarism, cardiac arrhythmia*

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1. Background

Empty sella syndrome (ESS) is characterized by the herniation of the subarachnoid space into the sella turcica, which is often associated with stretching of the pituitary stalk and flattening of pituitary gland [1,2]. Most ESS patients are asymptomatic, while the others may be associated with variable clinical conditions such as neurological, visual and/or endocrine disorders [3]. Pituitary hormones are necessary in human bodies, playing important roles in many organs and systems, such as glucolipid metabolism, hemopoietic, cardiovascular, central nervous and so on, but only a few studies addressed the relationship between hypopituitarism and arrhythmia.

2. Case Report

A 19-year-old male was admitted to our department because of non-sustained ventricular tachycardia (VT). He did not complain of palpitation, dizziness, chest pain, dyspnea, weakness, sweat or loss of consciousness while VT or frequent premature ventricular contraction (PVC) occurring. But there were two episodes of coma respectively in 2 years and 2 month ago. Two years ago, he became to be fatigable and pale, the coma occurred after acute gastroenteritis. At that time, he was found subclinical hypothyroidism, mild anemia, leucopenia, and frequent PVC. In the following-up visits, TSH was always above the upper limit with normal FT3, FT4, Hb fluctuated between 100g/L and 125g/L, and WBC ranged from 2.5*10^9/L to 5.0*10^9/L. Levothyroxine was taken discontinuously. Two months ago, being found drowsy but arousable, he was sent to the intensive care unit immediately. There was no history of nausea, vomiting, tic of limbs and foaming at the mouth. After admission, his body temperature was 38.9°C, heart rate was 110 beats per minute (bpm), blood pressure was 145/83mmHg, and respiratory rate was 22 breaths per minute. Physical examination revealed rough breathing, and moist rale was heard on the bilateral lungs. Cardiovascular and abdominal examination were unremarkable, nervous and visual fields could not be assessed. WBC was 9.9*10^9/L (3.5-9.5*10^9), Hb was 149g/L (130-175 g/L), platelets was 154*10^9/L) (100-300*10^9/L). Coagulation factors like FV, FIX, FX, FXI, FXII were below lower limit. Sodium was 142mmol/L (135-145 mmol/L), potassium was 4.1mmol/L (3.5-4.5mmol/L), serum iron was 7.5umol/L (10.6-36.7 umol/L), transferrin was 1.1g/L (2.0-3.6g/L), and total iron binding capacity was 29.9umol/L (50-70 umol/L). TORCH, EB virus, HIV, type A and B influenza virus, hepatitis B, hepatitis C, syphilis, tuberculosis antibody, rodenticide, ragged red fiber and Coomb's test were normal. ECG showed frequent premature ventricular contractions and non-sustained ventricular tachycardia probably from the outflow tract of right ventricle (Figure 1). 24-hour Holter

monitor showed the number of PVC was 27665. Chest CT revealed infectious lesions in the upper lobe of bilateral lung and the lower lobe of the left lung. Cranial CT revealed a low-density arc-shaped shadow at the left frontal cranial, which was considered as a possibility of arachnoid cyst or subdural effusion. Lumbar puncture (clear fluid with normal proteins) and echocardiogram were normal. After empirical antibiotic treatment (from ceftizoxime, meropenem, teicoplanin to moxifloxacin and azithromycin), supplying blood coagulation factor, reducing intracranial pressure and raising leukocytes for 4 days, his consciousness began to recover and could move under the doctor's instructions. Body temperature dropped to normal 7 days later. Finally, he was discharged with ventricular arrhythmia (24-hour Holter monitor showed the number of PVC was 35685 and non-sustained ventricular tachycardia) and abnormal coagulation function.

Afterwards, he went to our department. On examination, his weighted 65kg for 1.80m, the body mass index (BMI) was 20.06 kg /m^2. A careful review of the patient's history revealed that over the last two years, he began demonstrating signs of progressive indifference, lack of communication, decreases of activity. Further physical examination revealed loss of mustache, soft and pale skin, short and thin fingers, and normal genital. This new information, combined with the history of severe infection, strongly suggested a possible aetiology of hormonal dysfunction. And then, we performed endocrine tests, adrenal gland CT, cerebral MRI and cardiac MRI. As expected, the results supported a diagnosis of hypopituitarism, hypocortisolism, subclinical hypothyroidism and gonadal hormone disorder: TSH, 17.310 uIU/ml (0.49-4.90uIU/ml); free T3, 3.7pmol/L (3.1-6.9pmol/L); free T4, 10.1pmol/L (9.5-24.5pmol/L); 8am/4pm/0am cortisol, <1.50nmol/L; 8am ACTH, 3.82pg/ml (4.70~48.80pg/ml); 4pm ACTH, 2.23 pg/ml (4.70~48.80pg/ml); 0am ACTH, 2.02 pg/ml (4.70~48.80pg/ml). A cerebral MRI revealed empty sella turcica, a small area with a high T1 and T2 signal in the hypophyseal fossa (Figure 2). A cardiac MRI showed increased chordae tendinae in the right ventricle. No abnormality revealed on adrenal gland CT. In the light of these results, he was diagnosed as ESS with pituitary insufficiency, thus 30mg/day hydrocortisone was given for treatment. Seven days later, a very good response was noted, the patient became active and talkative, and physical examination revealed a few mustaches, the skin became rough. A three-day HOLTER showed no VT or PVC any more. After discharge, he kept on doing physical exercises and remained asymptomatic in one month. At the time of a routine follow-up, most of pituitary hormones and coagulation factors have returned to normal (Table 1 and Table 2). Expectedly, no VT was recorded in 24-hour Holter monitor.

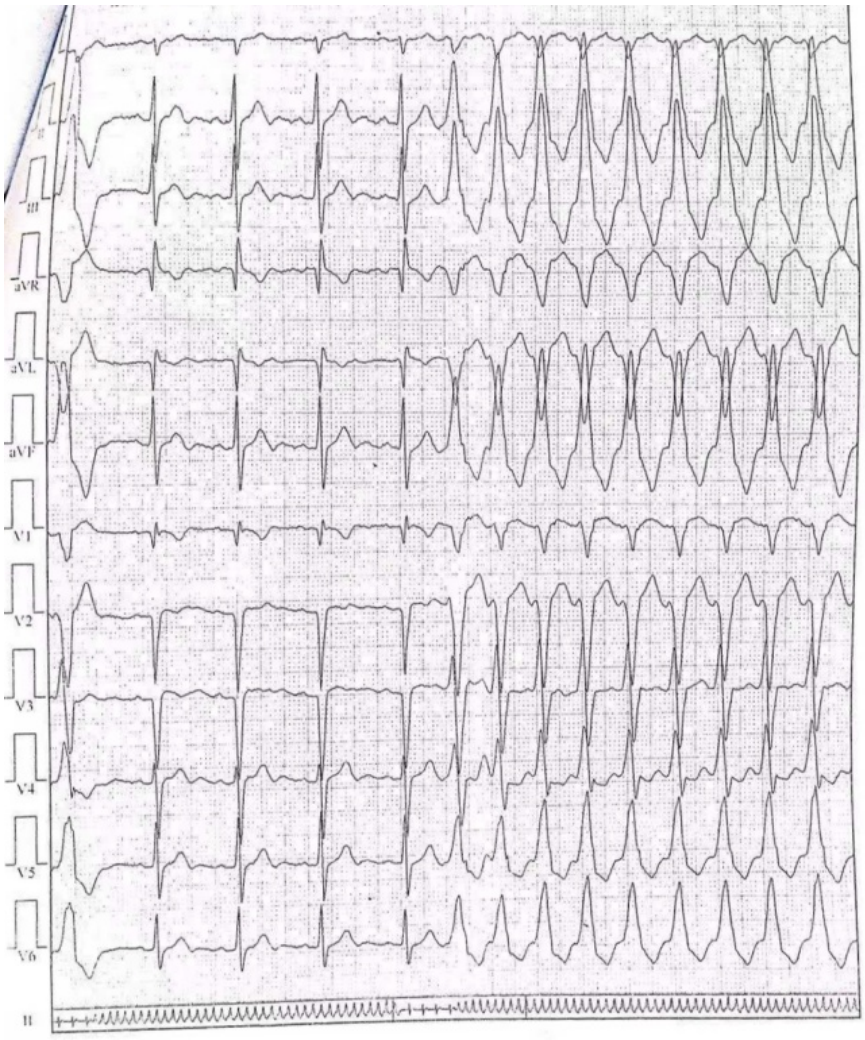

Figure 1. Monitoring by ECG on admission demonstrating ventricular tachycardia

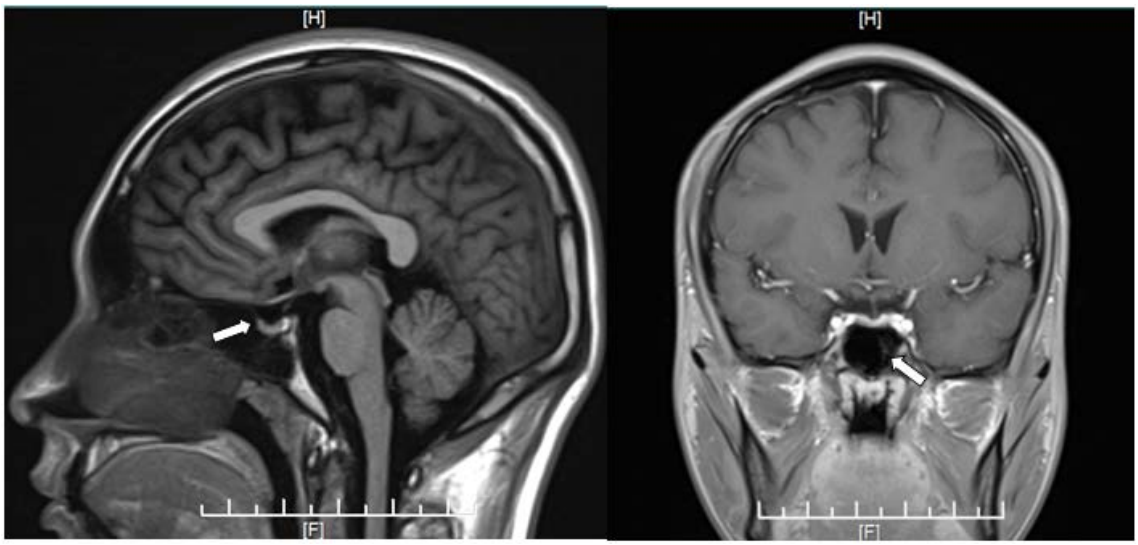

Figure 2. MRI of the empty sella (Sagittal and Coronal images after contrast medium administration)

Table 1. Serum pituitary hormone levels before and after treatment, and at a follow-up visit

Hormone	22th Jul	13th Jun	14th Aug	Reference Values
Thyroid Stimulating Hormone	17.31	9.64	9.51	0.49-4.91uIU/ml
Free testosterone index	59.22		157.58	20.4-81.2
Sex hormone binding globulin	56.40	21.80	16.00	11.20-78.10nmol/L
Dehydroepiandrosterone sulfate	48.66	51.77	29.93	45.10-385.00ug/dl
Estradiol	43.60	46.10	52.00	0-56pg/ml
Testosterone	963.25	>1009.40	727.14	142.39-923.14ng/dl
Luteinizing hormone	11.39	10.06	3.71	2.80-6.80mlU/ml
Follicle stimulating hormone	1.72	2.83	1.62	1.3-11.8mlU/ml
Prolactin	64.57	26.86	33.30	4.1-18.4ug/L
Progesterone	< 0.12	0.19	<0.12	0.12-0.61ng/ml
Growth hormone		0.34	5.60	0.02~1.23ng/ml

Table 2. Coagulation factors levels before and after treatment, and at a follow-up visit

Time	17 th May	29 th May	10 th Jul	22 th Jul	13 th Jun	14th Aug	Reference Values
II	72.2	81.1	83.5	55.0	48	90	70-120%
V	66.9	42.0	63.5	63.0	58	83	70-120%
VII	73.7	67.0	60.4	63.0	80	98	70-120%
VIII	85.3	44.1	42.9	44.0	56	68	70-150%
IX	56.2	29.0	37.1	55.0	97	106	70-120%
X	50.8	56.9	64.0	51.0	52	93	70-120%
XI	62.4	48.2	52.6	48.0	70	154	70-120%
XII	14.0	22.7	24.2	49.0	40	48	70-150%

3. Discussion

Lown et al. observed that steroids had effects on auriculoventricular conduction without any implication of mineral metabolic derangements, but might be associated with sympathetic nervous system [4]. Beyond that, hypopituitarism, hypothyroidism, and Addison disease are rare causes of prolonged cardiac repolarization though little evidence supporting that hypothyroidism or subclinical hypothyroidism can predispose to ventricular dysrhythmias [5]. A few studies have reported patients suffering from hypopituitarism presented with long QT interval and torsades de pointes (TDP) [6,7]. Right ventricular outflow tract tachycardia (RVOT) is focal in origin, often occurs in the absence of structural heart disease or fractionated electrograms [8,9]. Mechanistically, the VT is triggered result from cAMP-mediated delayed afterdepolarizations (DADs) due to intracellular calcium overload [10]. Patients with subclinical hypothyroidism presented cardiac repolarization abnormalities equally with overt hypothyroidism [11,12,13]. Alonso et al. observed that TSH lengthened the action potential and slightly depolarized the resting membrane potential due to a reduction in Ito and IK1 channels expression [14]. Fernandez-Ruocco et al. found the densities of Ito and IKur decreased while ICa-L increased in hypothyroid rats [15]. These studies provide a basis for development of prolonged QT and QTc intervals and TDP, but not mention the INa-Ca channel which playing an important role in DADs. However, little evidence indicates the relationship between adrenocortical insufficiency or hyperprolactinemia and ventricular tachycardia, even the other types of arrhythmia. In conclusion, cardiac arrhythmia may be associated with endocrine disturbances. Endocrine screening should be followed when hormonal abnormalities are suspected in ESS. Moreover, not all the

"idiopathic arrhythmia" is idiopathic, assessment of hormone may be considered to some extent.

Author Statement

Huang Zhou & Xue Kuang carried out and participated at the clinical procedure and the manuscript demonstration. Xianbin Lan, Huaan Du, Wenjiang Chen & Zulong Xie participated at the follow up of the patient. Yuehui Yin designed the study and helped to draft the manuscript. All authors read and approved the final manuscript.

Declaration of Competing Interest

The authors have no conflicts of interest or funding to disclose.

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