CASE REPORT

INTERMEDIATE

CLINICAL CASE

Ventricular Fibrillation

A Rare Initial Presentation of Thyrotoxic Periodic Paralysis

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ABSTRACT

Thyrotoxic periodic paralysis is a life-threatening complication of hyperthyroidism characterized by transient episodes of muscle paralysis and hypokalemia, commonly seen in Asian men. We present a rare case of ventricular fibrillation as the initial presentation of thyrotoxic periodic paralysis. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:1434–1437) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

HISTORY OF PRESENTATION

A 48-year-old Vietnamese man presented with out-of-hospital ventricular fibrillation (VF) cardiac arrest (**Figure 1**) following a low-speed single-vehicle collision. He was defibrillated in the field, with restoration of sinus rhythm. It was documented that he also received potassium and amiodarone during resuscitative efforts. He remained unresponsive, was intubated, and was transported to the hospital, where chest tubes were placed bilaterally for management of pneumothoraces induced as a result of bilateral finger thoracostomies performed by emergency

medical services during resuscitative efforts. He was subsequently admitted to the intensive cardiac unit and was started on a postarrest cooling protocol. His electrolytes and vital signs were normal on admission. He was found to have several ventricular arrhythmias on day 2 of his hospitalization in the setting of severe hypokalemia (1.9 mmol/L). The type of ventricular arrhythmias included paroxysmal supraventricular tachycardia, nonsustained ventricular tachycardia, and premature ventricular complexes (**Figure 2**). He remained hemodynamically stable during this time.

PAST MEDICAL HISTORY

The patient had no past medical history. He did not drink alcohol or smoke. He did not use illicit drugs and had no family history of cardiac disease.

DIFFERENTIAL DIAGNOSIS

The initial differential diagnosis of sudden cardiac arrest in an otherwise healthy middle-aged man included acute coronary syndrome. Other differential diagnoses considered were noncardiac disorders such

LEARNING OBJECTIVES

- To recognize TPP as a potential cause of ventricular arrhythmias.
- To understand and list the complications associated with TPP.
- To recognize the completely reversible nature of hypokalemia, ventricular arrhythmias, and muscle paralysis with prompt treatment of hyperthyroidism.

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as pulmonary embolism, electrolyte abnormalities, hemorrhage, or trauma, such as cardiac contusion, with motor vehicle accident seen in this case.

INVESTIGATIONS

On admission, the patient was promptly taken for cardiac catheterization, which showed normal left ventricular function and no coronary artery disease. Results of computed tomography (CT) of the chest were negative for pulmonary embolism, but the imaging did show a large left pneumothorax requiring chest tube placement. The transthoracic echocardiogram (TTE) showed no wall motion abnormalities, with normal left ventricular ejection fraction of 55% to 60%. No signs of cardiac contusion were seen on the TTE. Given the patient's unexplained VF, further evaluation included thyroid function tests, which showed findings consistent with profound hyperthyroidism (thyroid-stimulating hormone [TSH] <0.01 $\mu\text{IU/mL}$; free thyroxine [T_4] >5 ng/dL; free triiodothyronine [T_3], 8.9 ng/dL). The constellation of hypokalemia, hyperthyroidism, and ventricular arrhythmias in a man of Asian descent raised concern for thyrotoxic periodic paralysis (TPP). Note was made that the patient also received amiodarone during resuscitative efforts and iodinated contrast material to facilitate CT scans performed as part of his initial trauma evaluation. Both amiodarone and iodinated contrast material are known to exacerbate hyperthyroidism, thus making prompt initiation of antithyroid medication important. A head CT angiogram showed diffuse enlargement of the thyroid gland compatible with goiter.

MANAGEMENT AND CLINICAL COURSE

Aggressive potassium repletion and selective β -blocker (metoprolol) initiation were 2 steps taken to

manage ventricular arrhythmias during the diagnostic work-up. Given the high degree of suspicion for TPP, prompt treatment of hyperthyroidism with propylthiouracil (200 g orally every 4 hours) was started, and aggressive potassium repletion was continued. The patient was also transitioned to a high-dose nonselective β -blocker, propranolol (60 mg orally every 6 hours), to inhibit the peripheral conversion of T_4 to T_3 . Free T_4 levels were followed every 2 to 3 days to assess the response to the treatment plan. Because the patient remain afebrile and otherwise hemodynamically stable, thyroid storm was an unlikely diagnosis in the patient whose altered mental status was explained by other factors. Thus, no stress-dose hydrocortisone was started at the time. Use of amiodarone and iodinated contrast material in CT scans was minimized. Cardiac arrhythmias and hypokalemia episodes resolved promptly after initiation of treatment. In 48 hours after initiation of treatment, the patient's mental status improved on a minimal vent setting and with minimal sedation. The patient was extubated, and the chest tube was removed. High levels of thyroid-stimulating immunoglobulin titer were detected showing Graves disease. After 10 days of antithyroid treatment, thyroid levels were still relatively high (TSH <0.01 $\mu\text{IU/mL}$; free T_4 , 2.43 ng/dL), and thus the patient eventually underwent a total thyroidectomy for medically refractory hyperthyroidism. He was discharged home after 2 weeks on a propranolol taper with a plan to initiate levothyroxine on an outpatient basis.

ABBREVIATIONS AND ACRONYMS

- CT** = computed tomography
- TPP** = thyrotoxic periodic paralysis
- TSH** = thyroid-stimulating hormone
- TTE** = transthoracic echocardiogram
- T_3** = triiodothyronine
- T_4** = thyroxine
- VF** = ventricular fibrillation

DISCUSSION

This case report illustrates the life-threatening complication of TPP. Previous case reports showed

FIGURE 1 Electrocardiogram Strip in the Field Before Admission Showed Ventricular Fibrillation

FIGURE 2 Ventricular Arrhythmias Seen on Day 2 of Hospitalization in the Setting of Severe Hypokalemia

Electrocardiogram showing runs of nonsustained ventricular tachycardia.

that TPP manifests with sporadic episodes of hypokalemia, muscle paralysis, and sometimes life-threatening ventricular arrhythmias. It is a rare presentation of thyrotoxicosis and is most commonly associated in Asian men with Graves disease. The pathogenesis is mostly attributed to increased $\text{Na}^{+}\text{-K}^{+}$ ATPase activity stimulated by thyroid hormone that is postulated to contribute to hypokalemia and muscle paralysis (1). According to review of published reports, muscle paralysis seen in these patients mostly manifests with acute proximal symmetrical lower limb weakness, but it can progress to involve all 4 extremities and the respiratory musculature (1-3). There have been multiple case reports of TPP in the past that manifested with episodes of muscle paralysis with subsequent ventricular arrhythmia, but none whose initial presentation was ventricular arrhythmia (4,5). To our knowledge, this is the first reported case of TPP with an initial presentation of VF. The patient did not have any known episodes of muscle paralysis, arrhythmias, and hypokalemia before this incident.

The differential diagnosis of the ventricular arrhythmias included cardiac contusion secondary to motor vehicle accident and acute coronary syndrome, both of which were ruled out, as noted earlier. It is known that thyroid homeostasis is altered during the

acute phase of cardiac arrest, and thus, thyroid function tests in this patient were not given much thought until severe sporadic episodes of hypokalemia and ventricular arrhythmias were seen on day 2 of hospitalization (6). The triad of severe hyperthyroidism, ventricular arrhythmias, and hypokalemia in an Asian man helped identify TPP early on in our patient. Although the patient's admission serum potassium measurement was normal, he may have been hypokalemic at the time of his motor vehicle accident, with resuscitative efforts in the field and subsequent cellular shifts resulting in normalization of serum potassium by the time of his arrival at the hospital. Emergency treatment of hypokalemia with intravenous potassium supplementation and of hyperthyroidism with propylthiouracil was essential in complete recovery. Published reports suggest that propranolol has been shown to reduce the frequency and severity of paralysis in patients with TPP. The β_1 -selective agents such as metoprolol are less likely to inhibit the β_2 receptor-mediated hypokalemic effect of epinephrine and may therefore be less likely to prevent paralytic episodes (7). Precipitating factors for the sporadic episodes of hypokalemia and ventricular arrhythmias, such as the administration of amiodarone and iodinated contrast material, were noted and avoided at an early point. The complete recovery of

TPP symptoms seen in our patient conveys the completely reversible nature of this condition. TPP, a life-threatening condition, is a preventable and reversible complication of hyperthyroidism. The purpose of this case report is mainly to raise awareness of this condition, which has not been as widely studied by cardiologists in Western countries as it has by physicians, especially endocrinologists, in Eastern countries. However, it is important for cardiologists to be vigilant about this condition during diagnostic investigations of cardiac arrests because TPP is becoming more common in Western countries with population mobility and genetic admixture (8).

FOLLOW-UP

No further episodes of muscle paralysis, hypokalemia, and cardiac arrhythmias were noted by the patient on the follow-up clinic visit 3 months after discharge.

CONCLUSIONS

This case demonstrates that awareness and recognition of TPP are necessary for cardiologists confronted with unknown ventricular arrhythmias associated with untreated hyperthyroidism, hypokalemia, and muscle paralysis. Moreover, prompt treatment is essential to prevent recurrent ventricular arrhythmias and to achieve complete recovery.

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