Doi: 10.32677/IJCR.2016.v02.i04.011 Case Report

Primary Hyperparathyroidism and Cardiac Arrest: Case report

Omaima Mahmood , Saeed AG Mahmood, Zahraa Mohammed

From, Hamad Medical Corporation, AlShefa Poly Clinic, Doha, Qatar

Correspondence to: Omaima Mahmood, BSc in Pharmacy, College of Pharmacy, Qatar, Email: omaimasa92@gmail.com.

Received: 07 October 2016 Initial Review: 16 October 2016 Accepted: 26 October 2016 Published Online: 03 November 2016

ABSTRACT

Hyperparathyroidism is a common endocrine disorder characterized by hypersecretion of parathyroid hormones due to abnormal activity of one or more parathyroid glands. Several cardiac abnormalities have been reported due to hyperparathyroidism. In this case report, a case of cardiac arrest due to hyperparathyroidism and hypercalcemia is presented and discussed. A female patient, 73 years old, was admitted to surgical ward (SICU) with malignant goiter extending to the retrosternum. She was a known case of hyperparathyroidism and hypercalcemia. After the operation, the patient was shifted to SICU for further management. Suddenly, the patient had sudden cardiac arrest and was successfully resuscitated within 2 minutes. The ECG showed a new Right Bundle Branch Block (RBBB) changes. Patient remained on ventilator, not obeying commands, and opened her eyes spontaneously with cough reflex. A week after, percutaneous tracheostomy was performed, and patient became hemodynamically stable, weaned from ventilator and allowed to breathe spontaneously. Although cardiac arrest is a rare complication, it should be expected by health care providers when dealing with patients with hyperparathyroidism and hypercalcemia.

Keywords: Cardiac arrest, Hyperparathyroidism, Hypercalcemia, Parathyroid hormones

Hyperparathyroidism (HPT) is a common endocrine disorder characterized by hypersecretion of parathyroid hormones due to abnormal activity of one or more parathyroid glands [1]. Parathyroid hormone (PTH) has a central role in maintaining the hemeostatis of serum calcium and phosphorus levels. In normal cases, when serum calcium levels drop, this triggers parathyroid gland to induce the secretion of parathyroid hormones, which accordingly triggers calcium resorption from the bones and raise serum calcium levels [2]. However, in the case of hyperparathyroidism, the overactivity of parathyroid glands causes abnormal secretion of parathyroid hormone which leads to abnormal clinical hypercalcemia [3].

This condition usually occurs with patients who are above 65 years; however, it can also affect younger population [4]. The frequency of HPT is reported to be 1 in 1000 individuals in general population. In addition, the ratio of this disease is equal between men and woman (1:1) [5]. There are two types of hyperparathyroidism, primary and secondary. In primary HPT, the cause of the disease is due to an enlargement in one or more parathyroid gland itself. About 85% of the cases are reported to be caused by parathyroid adenomas.

However, other causes such as diffuse parathyroid hyperplasia and carcinoma are found in most of the remaining cases (around 15%) [6]. On the other hand, secondary hyperparathyroidism is resulted by the overactivity of parathyroid gland due to other conditions outside the gland itself which initially cause the dropping in serum calcium levels [7]. The most common reported cause is kidney failure due to chronic renal insufficiency which accompanied by hypocalcaemia and hypophosphatemia. Other causes are vitamin D deficiency, calcium malabsorption and osteomalacia [8].

The main sign of primary hyperparathyroidism is hypercalcemia which caused by the resorption of calcium from the bone, reduction of calcium excretion from the kidneys and the rise of the absorption of calcium by the bowl [9]. There are several complications that are reported to be associated with this condition including kidney stones, osteoporosis, and clinical symptoms of hypercalcemia [10]. However, recent studies have indicated other types of complications which are the cardiovascular diseases. Studies have found that primary HPT is associated with cardiac death, myocardial infarction, ventricular hypertrophy, disturbances in renin-angiotensin system and functional changes in cardiac vascular wall [11]. In this case report, a rare case for a patient who experienced cardiac arrest due to primary hyperparathyroidism will be presented.

CASE REPORT

A female patient, 73 years old, was admitted to surgical ward (SICU) with malignant goiter extending to the retrosternum. She was admitted to rule out metastasis and for pre-operative assessment (goiter removal). She is a known case of non-insulin dependent diabetes mellitus, hypertension, coronary artery disease, hypothyroidism, hyperparathyroidism and hypercalcemia.

Different examinations were done for the patient including CT scan, endocrinology test, echocardiogram etc. The CT scan found a left ventricular enlargement with retrosternal extension with absence of any cervical lymphadenopathy or pulmonary metastasis. In addition, Iodine 131 total body scintigraphy was performed and found an increase uptake in thyroid glands.

Biochemistry test were also done and all the results were normal including serum creatinine, sodium, potassium etc. In addition, hematology test revealed the following: Hemoglobin (HB) 12.1 g/dl, Platelets (Plt) 205000, Hct 38.5%. Other cardiac related investigations were also performed including echocardiogram, ECG and chest x-ray.

The echocardiogram found a mild left ventricular (LV) dysfunction with ejection fraction (EF) of 47%, LV diastolic dysfunction, LV and left atrial enlargement and hypokinetic inferioposterior wall. In addition, the result of ECG indicated the presence of sinus rhythm and LV hypertrophy. Lastly, chest x-ray found the presence of cardiomegaly. Endocrinology examination was also done and the following results were found (table 1):

Table 1 - Endocrinology test results

Parameters	The result	Normal levels
Parathyroid hormones	147 pg	15-65 pg
Vitamin D	19 ng	20-80 ng
Corrected calcium	2.89 mmol/L	2.1- 2.6 mmol/L
Albumin	40 g/L	35- 50 g/L
Free triidothyronine	2.5 pmol	3.4 -6 pmol
Thyroid Stimulating Hormone (TSH)	0.22 mIU /L	0.45- 4.5 mIU/L

The patient went under general anesthesia for operation which took about 6 hours. The induction was smooth and the intubation was easy. The patient experienced one episode of hypotension needed ephedrine and received 2 liters of crystalloids. After operation, she was shifted to SICU for further management.

In SICU, the patient was given diprivan for sedation and remifentanyl for analgesia. In addition, labetalol was given to reduce the blood pressure and pamidronate 60 mgs IV every 12 hours to treat hypercalcemia. On the day after, she weaned from ventilator and extubated.

Suddenly at 18:00 pm, patient experienced sudden cardiac arrest and was successfully resuscitated within 2 minutes. The ECG showed a new Right Bundle Branch Block (RBBB) changes. In addition, noradrenaline was started to maintain hemodynamics. On the day after, spiral CT chest showed minimal bilateral pleural effusion with no evidence of pulmonary embolism. After that, she had atrial fibrillation and was started on Amiodarone and digoxin. In addition, she also developed hypotension with atrial fibrillation and treated with DC shock.

Two days later, echocardiogram was performed and it ruled-out any intracardiac thrombus with EF 65%. CT scan for the head was also done and showed minimal intraventicular hemorrhage with no active management by Neurosurgeon. Patient remained on ventilator, not obeying commands, and opened her eyes spontaneously with cough reflex. One week after, percutaneous tracheostomy was done and patient, then, became hemodynamically stable, weaned from noradrenaline and ventilator and allowed to breathe spontaneously with HME.

DISCUSSION

Hyperparathyroidism is a condition in which parathyroid glands become overactive and secret abnormal levels of parathyroid hormones. In the case of primary hyperparathyroidism, the cause of this overactivity is due to an enlargement in the gland itself due to adenoma, hyperplasia, carcinoma etc. Parathyroid gland play central role in maintaining the balance of serum calcium and phosphorus levels. Therefore, once the gland become hyperactive, several complications could be expected. These complications include but not limited to osteoporosis, kidney stones, clinical hypercalcemia and multiple cardiovascular problems. In the last two decades, studies have focused on exploring the association between primary hyperparathyroidism and heart diseases [12].

The main mechanism by which the cardiac abnormalities are established is through the followings. At the beginning, parathyroid hormones bind to PTH/PTHrp (receptor protein) on cardiomyocytes. This cause an increase in intracellular calcium levels which results in the activation of protein kinase C cascade. This activation causes a hypertrophic process to be initiated on the cardiac myocytes beside some other metabolic effects [13]. In addition to the hypertrophic effect, parathyroid hormones increase the chornotropic effect on cardiac cells and decrease the energy utilization which results in myocardial and valvular calcifications, diastolic dysfunction and cardiac hypertrophy [14]. Moreover, high calcium level along with high parathyroid hormones affects endothelial cell and cause a vascular wall dysfunction which consequently results in an increase in central blood pressure and worse the condition of hypertrophy [15].

These complications have been reported in different studies. A study by Symone et al, reported the occurrence of left ventricular hypertrophy in patients with high levels of parathyroid hormones and hypercalcemia [16]. Similarly, another blinded study found that around 82% of primary hyperparathyroidism patients experienced LV hypertrophy before the operation [17]. In the same study, the investigators found a regression of hypertrophy of interventricular septum and cardiac posterior wall by - 6% and - 19% ($P < 0.05$) after parathyroidectomy. The time takes for this regression to be completed is still uncertain. Some studies indicated that it takes not less than 2-3 months to get full hypertrophy regression [18, 19]. However, other studies indicated no difference in the hypertrophic levels until 6 to 24 hours after parathyroidectomy [20, 21]. In addition, studies found that patients with symptomatic primary hyperparathyroidism are at significant high risk of mortality before and even after parathyroidectomy [22, 23].

In our study, the patient experienced a rare complication of pHPT which is cardiac arrest. Although it is a rare case, some other case reports have also reported this incidence. One study reported a case for a patient who presented to emergency department unconscious and experienced cardiac arrest. Later, they found the reason for the arrest which is hyperparathyroidism that resulted in thyroid storm-induced cardiac arrest [24]. Another case for a 47-years old patient was discussed by another study [25]. This patient had hyperparathyroidism which leads to complete atrioventicular block and cardiac arrest despite the initiation of anti-thyroid medications. This was considered a rare case since the common cardiac manifestation of HPT is atrial fibrillation, atrial flutter, sinus tachycardia, angina pectoris, heart failure or cardiomyopathy. However, a complete atrioventicular block and cardiac arrest are very rare complications. The mechanism that leads to cardiac arrest has been demonstrated in several studies. Both elevated calcium level and PTH exert a hypertrophic effect on cardiac cells and subsequently leads to cardiac abnormalities and possibly cardiac arrest [17]. In addition, other reports have found a relation between the use of beta-blocker and cardiac arrest by inducing a poor ejection fraction. Therefore, it has been recommended to use short acting beta blocker like Esmolol for hemodynamic monitoring [25].

CONCLUSION

Hyperparathyroidism is characterized by abnormal levels of parathyroid hormones and calcium which causes different cardiac abnormalities. Although cardiac arrest is a rare complication, health care providers should be prepared to deal with such consequences.

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How to cite this article: Mahmood O, Saeed AG, Mohammed Z. Primary Hyperparathyroidism and Cardiac Arrest: Case report. Indian J Case Reports. 2016; 2(4): 121-124.

Conflict of interest: None stated, Funding: Nil