Sudden cardiac arrest as a rare presentation of myxedema coma: case report

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1. Case report

A 62-year-old male with past medical history of hypertension, diabetes mellitus, and hepatitis C was brought to the emergency department (ED) by Emergency Medical Service (EMS) after initial resuscitation of cardiac arrest at his home. He was found in asystole by EMS and return of spontaneous circulation (ROSC) was achieved after one dose of epinephrine. No further history was available. In ED patient was found intubated and unresponsive to painful stimuli. Patient was hypothermic (93°F) and physical examination revealed a puffy face and dry skin; the rest of the examination was unremarkable. EKG revealed sinus bradycardia and low voltage QRS with left axis deviation (Figure 1). He was admitted to the intensive care unit (ICU) in view of status post cardiac arrest of unknown etiology. Work up was done to rule out most common etiologies of sudden cardiac death. Chest x-ray was negative for any acute pulmonary pathology. Echocardiogram revealed normal left ventricular function with no regional wall motion abnormality and no pericardial effusion. Head CT was unremarkable. Perfusion scan was not suggestive of pulmonary embolism. Electrolytes were normal on presentation, with no evidence of hyponatremia or hypoglycemia. Later, son revealed that patient had history of forgetfulness a week prior to presentation but he ascribed those symptoms to dementia. Thyroid profile revealed normal TSH level of 43.730 uIU/ml and free T4 level of <0.11 ng/dl and total T4 < 0.5 mcg/dl; the patient was diagnosed with myxedema coma with a score of more than 60. The patient was then started on intravenous levothyroxine and hydrocortisone. Adrenal insufficiency was ruled out as the serum cortisol am was 19.8 ug/dl. The patient was managed in the ICU and he continued to have episodes of bradycardia. The hospital course was complicated by another episode of cardiac arrest but he was successfully resuscitated; it was preceded by prolonged QT and bradycardia, leading to third degree heart block (Figure 2). Because of persistent bradycardia the patient’s levothyroxine dose was increased. He was also started on T3 a week after treatment with levothyroxine. The patient showed gradual improvement in hemodynamic status and was able to be taken off pressor support and was successfully extubated. The hospital course was complicated by another episode of cardiac arrest but he was successfully resuscitated; it was preceded by prolonged QT and bradycardia, leading to third degree heart block (Figure 2). Because of persistent bradycardia the patient’s levothyroxine dose was increased. He was also started on T3 a week after treatment with levothyroxine. The patient showed gradual improvement in hemodynamic status and was able to be taken off pressor support and was successfully extubated. The patient showed gradual improvement in hemodynamic status and was able to be taken off pressor support and was successfully extubated. Eventually, the patient improved and was discharged home.

2. Discussion

Hypothyroidism mostly affects females and an overt hypothyroidism is present in approximately 3% of an adult female population. Cardiovascular manifestations which are frequently seen in hypothyroid state include decrease in cardiac output, heart rate, stroke volume, and myocardial contractility and an increase in systemic vascular resistance. Although the most common cardiac arrhythmias seen in hypothyroidism are sinus bradycardia, low voltage QRS complexes and prolongation of QT interval, ventricular tachycardia, torsades de pointes, and ventricular fibrillation have been reported in a few cases [1]. Sudden cardiac arrest (SCA) is uncommon in profound hypothyroidism. Only one case of SCA has been reported to our knowledge [2].
reported so far and that was also in an elderly female [2]. Our case is unique because it reports SCA as a first presentation of severe hypothyroidism in an adult male.

Thyroid hormone is an important regulator of cardiac gene expression and many of the cardiac manifestations of thyroid dysfunction are associated with alterations in T3-mediated gene expression. Thyroid hormone affects the action potential duration and repolarization currents in cardiac myocytes through both genomic and non-genomic mechanisms [1,3]. The pacemaker regulated genes, hyperpolarization activated cyclic nucleotide-gated channels 2 and 4, are transcriptionally regulated by thyroid hormone [4]. Thyroid hormone regulates several enzymes involved in calcium fluxes in the heart including the calcium-dependent adenosine triphosphates and phospholamban [5,6].

Overall, hypothyroidism is believed to induce a sympathovagal imbalance, characterized by decreased cardiovascular sympathetic and vagal modulation. However, the sympathetic influence is believed to predominate. The sympathovagal imbalance and increased inhomogeneity of ventricular recovery times can both predispose to potentially life-threatening arrhythmias. High plasma norepinephrine levels have been seen in hypothyroidism, but the responsiveness to endogenous catecholamine is decreased, because of a decrease in the number of beta adrenergic receptors and their desensitization to the effect of catecholamine. This autonomic dysfunction can be partly restored after replacement treatment with levothyroxine [7].

Uncontrolled hypothyroidism leads to decreased expression of tri-iodothyronine (T3) in the heart cells which can cause worsening of

Figure 1. EKG showing low voltage and sinus bradycardia.

Figure 2. EKG showing prolonged QT.
cardiac contractility, decreasing heart rate, and a slowing of the conduction of electrical stimuli in the heart muscle [8]. This may be the reason for bradycardia and prolongation of the QT interval and, in consequence, life-threatening arrhythmias may occur. This explains why our patient had a second episode of SCA in the hospital which was preceded by complete heart block (CHB). After starting T3, it was resolved and patient did not develop CHB or any arrhythmias during further hospital stay.

In review of the literature, severe hypothyroidism has presented with syncope secondary to arrhythmias, complete heart block. However, it is extremely rare for hypothyroidism to present first time as SCA [8]. Our patient was not known to have hypothyroidism and presented with SCA. He was found to have prolonged QT which might have triggered ventricular arrhythmias and subsequently led to SCA. No other focus of arrhythmias was found and it resolved after starting levothyroxine. Hence, undiagnosed hypothyroidism was the attributable cause of sudden cardiac arrest in this case.

2.1. Conclusion

Determining the underlying cause of sudden cardiac arrest as early as possible plays a crucial role in management. That’s why we report this case to consider myxedema coma as a potential reversible cause in the differential diagnoses of SCA.

Disclosure statement

No potential conflict of interest was reported by the authors.

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