Refractory alkalosis developed after cardiac arrest: Apathetic thyrotoxicosis

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Özet

Kardiyak arrest sonrası tedaviye dirençli alkalozis gelişmesi: Apatetik tirotoksikoz
Hipertiroidi varolan kalp hastalığını arttırabilir, atrial fibrilasyon ve kronik kalp yetmezliğine neden olabilir. Kardiak arrest sonrasında tedaviye dirençli alkaloz gelişimesinin nedeni bu olgumuzda olduğu gibi 'apatetik tirotoksikoz' olabilir. Bu nedenle atrial fibrilasyonlu yaşlı olgularda subklinik hipertiroidizm olasılığı akılda tutulmalı ve araştırılmalıdır. Hipertiroidik yaşlı hastalarda plazma tiroid hormonlarındaki artış genellikle orta düzeydedir ve hipertiroidiye bağlı gerçek klinik durum atrial fibrilasyon ve kalp yetmezliğinin semptomları nedeniyle maskelenmiş olabilir

Anahtar kelimeler: solunumsal alkaloz, kardiyak arrest, apatetik tirotoksikoz.

Abstract

The increase of plasma thyroid hormones in elderly patients is generally with a mild elevation and the real clinical scheme due to hyperthyroid is hidden because of the symptoms of heart failure and atrial fibrillation. A woman patient at 71 years old made an application to the emergency service with the complaints of abdominal distention and a period of twenty days without defecation. An atrial fibrillation with high ventricular pulse was found in the first examination. After the resolution of ileus the patient had a cardiac arrest. It was observed that alkalosis which was resistance to therapy developed after cardiac arrest. We investigated the reasons of the alkalosis which was resistant to therapy. The results of the thyroid functions tests showed that the patient had an undiagnosed hyperthyroidism before.
Thyrotoxicosis can increase the existing heart disease, atrial fibrillation and chronic heart failure. Alkalosis which is resistance to therapy develops after cardiac arrest, the reason might be the apathetic thyrotoxicosis. Thus, the elderly patients with atrial fibrillation, subclinical hyperthyroidism should be investigated.

Keywords: Respiratory alkalosis, cardiac arrest, apathetic thyrotoxicosis.

Introduction

Hyperthyroidism, especially in elderly patients the increase in the plasma thyroid hormones is at mild levels. The symptoms due to hyperthyroidism can be masked by the atrial fibrillation or by hearth failure with high cardiac output. These patients are resistant to inotropic agents. Although inotropic agents are used, it is impossible to eliminate tachycardia. Decreasing the thyroid hormone levels can only control these symptoms and then inotropic can be effective (1). This type of cardiac failure is called 'apathetic thyrotoxicosis' (2).
In this case, after cardiac arrest, respiratory and metabolic alkalosis resistant to medical therapy occurred, and while investigating the reason, apathetic thyrotoxicosis determinated.

Case Report

A 71 years old woman was admitted to the S.D.U. emergency department with the complaints of abdominal distension and lack of defecation for 20 days. In the initial examination of the patient; arterial blood pressure: 110/70 mmHg, pulse rate: 100 beat/min, bilateral pretibial edema (+), presacral edema (+). Atrial fibrillation with high ventricular rate was found in ECG recording with arrhythmia in auscultation of the heart. X-Ray examination of the lungs showed that elevation of right diaphragm and bilateral hiler fullness and increased cardio-thoracic ratio. The results of biochemical exam were shown at the table.

Table : The patient's biochemical exam.

Test	Level	Test	Level
Serum glucose	158 mg/dl	Na^+	137 mEq/L
BUN	24 mg/dl	K^+	3.9 mEq/L
Creatine	1.0 mg/dl	Ca^{++}	7.9 mEq/L
AST	1060 IU/L	Cl^-	91 mEq/L
ALT	818 IU/L	Urine density	1022
LDH	5780 IU/L	Urine pH	5.5
CK-MB	22 mg/dl	Urine acetone	(-)
Htc	38.2 %	Urine protein	(+)
Hb	12.6 g/dl	Urine bilirubin	(-)
WBC	9900/mm^3	Urobilinogene	(+/-)
Platelets	257.000 mm^3		
Microscopic urine examination: 4-5 Erythrocyte and 5-6 Leukocyte			

After the enema application the intestinal obstruction was resolved. While performing v. subclavian catheterization for further therapy cardiac arrest had occurred. Because there was no spontaneous respiration and mental activity the patient was admitted to intensive care unit. Mechanical respirator initiated by Bird 8400 at SIMV mode and FiO2: 90%. The blood gas analysis was as follow: pH: 7.51, pO2: 343.6 mmHg, pCO2: 26.4 mmHg, HCO3: 21.3 mmol/L, Na+: 133.4 mmol/L, Ca++: 0.946 mmol/L. There was no acidosis, so any NaHCO3 therapy applied during resuscitation. Arterial blood pressure was 140/80 mmHg and heart rate 60 beat/min at the beginning but hearth rate began to increase after an hour. Two hours later heart rate was 135 beat/min and midazolam-atracurium infusion started for sedation. Digoxin 0.25 mg/day and furosemide 40 mg/day added to the therapy. Next day, following the arrest respiratory alkalosis was dominated at the blood gas analysis. pH: 7.55, pCO2: 48.1 mmHg, pO2: 105.8 mmHg, HCO3: 42.2 mmol/L. Heart rate was between 120-140 beat/min. Metoprolol tartarate 5 mg/day was added to the therapy, because of the existing atrial fibrillation and heart failure. Dopamine 5 gr/kg, dobutamine 5 gr/kg were added to the therapy to improve the blood pressure and cardiac output. Enoxaparine sodium 40 mg/day, verapamil HCl 5 mg/day were also added. At the blood biochemistry a serum electrolytes (Na+: 132 mmol/L., K+: 2.9 mmol/L, Cl-: 93 mmol/L, Ca++: 6.7 mmol/L, Mg++: 1.4 mmol/L) and creatine levels were found lower when blood urea levels were normal. At the 4th day after the arrest the serum electrolytes improved but alkalosis scheme was persistence. The thyroid hormone levels that we wanted to determine the reason of this refractory alkalosis, showed us a hyperthyroid not diagnosed before (Free T3: 4.85 pg/ml (2,3-4,2), free T4: 2.01 pg/ml (0.89-1.8), TSH 0.291 mIU/ml (0.35-5.5)). Unfortunately, before we began the therapy, barotraumas, one of the complications of the mechanical respirator occurred and the patient was lost.

Discussion

Metabolic acidosis develops due to hypoxia and insufficient blood circulation after cardiac arrest; water and sodium enters to the cell and potassium leaves. On the other hand metabolic alkalosis mostly develops after iatrogenic replacement of excessive alkali, the loss of gastric content or after the diuretic therapy. The reason of the respiratory alkalosis is hyperventilation of any reason. Hyperventilation can be developed by stimulation of respiratory center in the brain by biochemical or hormonally. Respiratory alkalosis due to hyperthyroidism is seen very rarely (2). In this case, acute respiratory alkalosis developed instead of metabolic or respiratory acidosis after cardiac arrest. Acute respiratory alkalosis is always decompansations. Respiratory alkalosis is determinated as decrease in PaCO2 and increase in blood pH due to hyperventilation. Renal compensation of respiratory alkalosis develops slowly. In chronic respiratory alkalosis, HCO3- excrete from kidneys increased and also BE increased. In this case, observation of blood gas results at the day after cardiac arrest, chronic respiratory and metabolic alkalosis developed, and it was refractor to therapy. In combined alkalosis, myocard

depression, vasoconstriction develops and O2 deliver to tissues decreased. In alkalosis, even though we approved the electrolyte disturbances by sufficient replacement therapy, serum potassium levels decreased, carpopedal spasm, tetani due to ionized calcium occurred.

We wanted to determine the reason of this refractory alkalosis. Also anoxic encephalopathia can be the reason of alkalosis. In hypoxemia, by the peripheral chemoreceptors, respiratory center was stimulated. However, in this case by immediate resuscitation, CNS was prevented from anoxia. The thyroid hormone levels that we wanted to determine the reason of this refractory alkalosis showed us a hyperthyroidy which is not diagnosed before.

It is known that hyperthyroid causes important cardiovascular changes. Especially in elderly patients hyperthyroidism can be seen with atypical symptoms as the same as apathetic hyperthyroidism (3). Hyperthyroidism can augment chronic diseases, especially cardiovascular situations. A disorder of the cardiovascular function and the exercises capacity is more important for elderly patients. When the patient becomes euthyroid most of the cardiovascular abnormalities vanish and the atrial fibrillation can still exist lightly. For optimal antithyroid therapy is to be applicated immediately and absolutely. Patients older than 50 years having atrial fibrillation and hyperthyroid especially with the risk of emboli, and having ECG symptoms about hypertension or left atrium enlargement is advised to use an anticoagulant therapy (4). At a study about the hyperthyroid patients, it was found that the most seen arrhythmia is atrial fibrillation (54.7%). At the same study, the frequency of atrial fibrillation was found to be 51.4 % (5).

Thyrotoxicosis can increase the existing heart disease, atrial fibrillation and chronic heart failure (4). Alkalosis which is resistance to therapy develops after cardiac arrest, the reason might be the apathetic thyrotoxicosis. Thus, the elderly patients with atrial fibrillation, subclinical hyperthyroidism should be investigated.

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