OLGU SUNUMU CASE REPORT

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A Female Patient with Recurrent Ventricular Fibrillation Attacks Due to Severe Hypokalemia Induced by Taraxacum Plant Intake

Kadın Hastada Karahindiba Kullanımı ile Tetiklenen Şiddetli Hipokalemi Nedeniyle Tekrarlayan Ventriküler Fibrilasyon Atakları Gelişimi

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ABSTRACT Taraxacum is used by patients due to strengthening the immune system or diuretic effects. A 33-year old female patient with acute myeloid leukemia (AML) diagnosis was taken to the intensive care unit (ICU) due to sudden cardiac arrest. Initially, serum values identified severe hypokalemia (1.5 mmol/L). Despite replacement potassium, ventricular fibrillation (VF) attacks recurred. History has long-term and excessive consumption of dandelion plants to strengthen the immune system. There is no evidence for hypokalemia except taraxacum because she hadn't received chemotheraphy agent and hadn't had diarrhoea during last 2 months. We thought that the recurrent VF attacks and previous cardiac arrest were due to hypokalemia linked to severe diuresis. After patient's potassium were above 3 mmol/L, no ventricular fibrillation attacks were observed.

Keywords: Taraxacum; hypokalemia; ventricular fibrillation

güçlendirici ve idrar söktürücü etkisi nedeniyle hastaların kullandığı bir bitkidir. 33 yaşında akut myeloid lösemi (AML) tanılı, bayan hasta ani gelişen kardiyak arrest nedeni ile yoğun bakım ünitesine (YBÜ) alındı. Geliş serum değerlerinde şiddetli hipokalemi (1,5 mmol/L) saptandı. Uygulanan potasyum replasman tedavilerine rağmen tekrarlayan ventriküler fibrilasyon (VF) atakları gelişti. Hastanın öyküsünde bağışıklık sistemini güçlendirmek için verilen karahindiba bitkisinden uzun süreli ve fazla miktarda tüketim olduğu görüldü. Hastanın son 2 aydır kemoterapi ajanları almadığı ve diyaresi olmadığından hipokalemi sebebi için karahindiba kullanımı dışında kanıt bulunamadı. Biz tekrarlayan VF ataklarının ve geçirilmiş kardiyak arrest sebebinin şiddetli düreze bağlı hipokalemiden kaynaklandığını düşündük. Hastanın potasyum değerlerinin 3 mmol/L değerinin üzerine çıkması sağlandıktan sonra ventriküler fibrilasyon atağı görülmedi.

ÖZET Karahindiba (taraxacum officinale) bitkisi bağısıklık sistemini

Anahtar Kelimeler: Karahindiba; hipokalemi; ventriküler fibrilasyon

The use of plants for medical aims is becoming more common in Europe, America and developing countries. Taraxacum species have been used in traditional Chinese medicine as a diuretic for 2000 years.¹ Dandelion varieties are found commonly everywhere and ensure elimination of fluids through the kidneys in urinary and renal diseases.² Due to containing potassium, they reduce serum potassium values by a lower amount compared to other medical diuretics and even may elevate them.^{2,3} A 33-year old female patient admitted to the intensive care unit (ICU) with cardiac arrest had severe hypokalemia (1.5 mmol/L) observed in blood values. The patient with three-year AML diagnosis had received chemotherapy (CT) 2 months previously due to recurrence. History obtained from the family revealed that she had consumed 10-15 cup of herb tea daily with dandelion plants (taraxacum officiale) for 2 months to strengthen her immune system. In this knowledge, we thought that hypokalemia developing

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due to excessive diuresis was considered to cause cardiac arrest and recurrent VF attacks.

This study highlights the utility, severe hypokalemia because of consuming large amounts of dandelion plants which cause recurrent ventricular fibrillation. Written consent had been obtained from the patient's relative for publication of this case report.

CASE REPORT

A 33-year old female patient with body mass index of 13.8 was admitted to the ICU due to sudden cardiac arrest. The patient's history included 3-year AML diagnosis, recurrence 2 months previously and 2 doses of CT two months ago. At that time serum biochemical values were WBC: 4.2 mm³/10³, neu: 3800/mm³, Hgb: 6.4 mg/dl, plt: 19000 10³/mm³, BUN:15 mg/dl, creatinine: 0,34 mg/dl, C-reactive protein (CRP): 21.3. Medical treatment included Mikofenalat Mofetil (MMF) 1 gr 2x1, prednizolon 1 mg/kg/day and cyclosporine-A (CsA) and acyclovir 800 mg 2x1 for recurrence. Unfortunately, all these medications have similar side effects like leukopenia, thrombocytopenia, immunosuppression, renal and hepatic failure. Most frequent side effects are nausea, vomitting and allergy. Our patient had severe nausea attacks, episodic vomitting, rarely diarrhea and no allergy when she had chemotherapy agents two monts ago. During the last two months, there were no symptoms like nausea or diarrhea but she felt weakness. Before admitting ICU serum potassium was 2,2 mmol/L and she had given 8 ampuls of %10 potassium chloride (KCL) infusion for 10 hours. Despite these medications she had cardiac arrest suddenly. After successful resuscitation, she was admitted to ICU. Tests revealed potassium 1.5 mmol/L, magnesium 1.42 mmol/L, calcium 8.9 mg/dl and CRP was 66.3 mg/L. Renal functions (BUN 18 mg/dl and creatinine 0.68 mg/dl) were normal, with moderate increase in liver functions (AST: 70 U/L). Hemoglobin was 10.5 mg/dl, platelets were 57,400/mm³. Initial electrocardiography (ECG) of the patient showed sinus rhythm, flattened T waves and U wave (Figure 1).

Potassium support was begun with 10% KCL at 10 mEq in the first 5 minutes and 40 mEq/h infusion with continuous ECG monitoring.⁴ Potassium increased to 2.6 mmol/L. In the first 12 hours, the patient had two recurrent VF attacks despite 32 ampuls of 10% KCL treatment. She was defibrillated and returned to sinus rhythm. After defibrillation, the patient medicated with amiodaron 200 mg tb 3x1, magnesium sulfate (15%, total dose 3g), klopidogrel 75 mg tb 1x1 and atorvastatin 40 mg tb 1x1. Patient's urine output was 150-200 ml per hour without diuretic medication. On the 2nd day, when potassium was 2.5 mmol/L, the patient was extubated. Urine output was observed to be 200 ml per hour without diuretic treatment from admission onward. Replacement with KCL continiued 20-40 mEq/h 10% KCL daily. Despite the replacement, potassium was 2.5 mmol/L. While replacement continued, sudden VF attack recurred. The patient was intubated again. With VF attacks recurring twice, the patient was defibrillated and returned to sinus rhythm. After second



FIGURE 1: Initial ECG flattened T waves and U wave observed.



FIGURE 2: After supplement of potassium chloride: T wave depression reduced and QT intervals lengthened.

fibrillation attack we thought that sufficient elevation may not be obtained in spite of potassium replacement. For this reason, magnesium replacement was added to treatment. 15% magnesium sulfate with total dose of 3 gr was added on potassium replacement daily. On checks, potassium values remained above 3 mmol/L. Repeated ECG showed that T wave depression reduced and QT intervals lengthened (Figure 2). This showed that our treatment was effective. The patient was extubated on the 10th day and VF attacks did not recur. As from the first day, the patient was fed with 20 ml/kg Oliclinomel-N4 (1000 ml) which contains 0.119 gr KCL and 0.045 MgCL/100 ml. Urine output rate regressed to 50 ml per hour. On the 14th day, the patient was transferred to the ward for observation and discharged.

History obtained from the family revealed that the patient had been continuously using taraxacum officinale (TO) for the last 2 months. It is considered that severe hypokalemia developed linked to increased diuresis due to TO use and linked to this the VF and cardiac arrest formed.

DISCUSSION

One of the most commonly known causes of hypokalemia is diuretic use. Due to ethnobotanical research, taraxacum species are used to increase elimination of fluids in urinary and renal diseases.² Apart from this effect, it is known to be a strong nutritional supplement.⁵ Compared to other botanic diuretics, it contains three times more potassium.² Some publications have emphasized that TO causes

hypokalemia.⁶ Additionally, most importantly it causes hypokalemia and hypomagnesemia due to excessive diuretic effects.² Pharmacological diuretics cause less magnesium loss.⁷

Arrhythmia incidence linked to degree of hypokalemia is a known reality. Hypokalemia increases the resting cardiac membrane potential, and lengthens the action potential duration and refractory period duration.8 These physiological changes increase excitability of the heart and cause dysrhythmia. Pezhouman et al. researched molecular basis of hypokalemia induced VF and they explained that arrhythmias about inhibition of Na-K pump.9 These findings of hypokalemia develop in 80% of cases with serum potassium concentration <2.5 mEq/L.¹⁰ Our case had initial serum potassium values of 1.5 mmol/L measured on admission to the ICU. It was considered that severe hypokalemia may have caused VF and cardiac arrest. In the process of elevating values to 2.6 mmol/L, VF attacks developed twice. Potassium replacement alone may not be sufficient to prevent arrhythmia linked to hypokalemia. Anderson et al. emphasized the necessity to use calcium channel blockers for arrhythmia.¹¹ Yang et al. recommended the use of a Class-III antiarrhythmic agent in the presence of arrhythmia with hypokalemia.¹² The other journal identified that in the presence of dofetilide (class III antiarrythmic agent and potassium channel blocker) the arrhythmia risk formed with serum potassium values below 3.3 mmol/L, with arrhythmia identified at 2.7 mmol/L values when antiarrhythmic agents were not used.9 In our patient we

added the antiarrhythmic cordorone 200 mg 3x1 to treatment while administering potassium replacement.

As urine output in the patient was 5 ml/kg/h, fluid hydration and potassium replacement treatment continued. While administering replacement, when serum potassium values were 2.5 mmol/L, the patient developed VF attacks twice. In resistant hypokalemia cases, sufficient elevation may not be obtained in spite of potassium replacement. In this situation, magnesium replacement may be added to treatment.¹³ As a result, we administered 15% magnesium sulfate with total dose of 3 g in addition to potassium replacement in our patient. Monitoring showed that we were able to keep potassium values above 3 mmol/L.

Apart from the diuretic effect, taxacarum officinale is used by patients due to antioxidant properties and for vitamin and mineral supplementation.⁵ The patient used it to support the immune system due to recurrence of AML and CT administration in the last 2 months. Histopathological changes occurring linked to renal toxicity of plant extracts have been shown to have regulative and supportive effects.¹⁴ Additionally, due to effects of hypokalemia, hypomagnesemia and inducing renal oxidative injury, long term use may cause harm as with other diuretics.¹⁵ Our case had intake duration of 2 months and took excessive amounts causing severe hypokalemia and later ventricular fibrillation. Consequently, taxacarum species are plants commonly used as diuretics and nutritional supplements in the population. Due to containing potassium, they cause less potassium loss than many diuretic treatments. Additionally, as long-term use may change the electrolyte values of patients, it may lead to electrolyte imbalance and dysrhythmia. Just as for all diuretic agents, it should be used by taking care of duration and frequency of use. As there are few human studies related to taxacarum, the need for advanced research on this topic should not be forgotten.

Source of Finance

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: Meltem Genez; Control/Supervision: Meltem Genez; Literature Review: Meltem Genez; Writing the Article: Meltem Genez.

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