OLGU SUNUMU – CASE REPORT

SEVERE CACHEXIA AND PROLONGED STAY IN THE INTENSIVE CARE UNIT FOLLOWING INGESTION OF CAIGUA

CAİGUA BESİN ZEHİRLENMESİNİ TAKİBEN GELİŞEN ŞİDDETLİ KAŞEKSİ VE UZAMIŞ YOĞUN BAKIM SÜRESİ

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SUMMARY

We aimed to present a case with severe cachexia in the early period after ingestion of caigua (cyclanthera pedata) which is a slender tropical vine indigenous to South America. A 15-year-old male patient was referred to our intensive care unit with acute nausea and vomiting, muscle weakness, metabolic acidosis, respiratory failure, and loss of consciousness. His past medical history was unremarkable. He had eaten caigua shortly before onset of symptoms. He had rapidly progressive cachexia and lost more than 20 percent of his weight over 1 month. "Acute disseminated encephalomyelitis (ADEM) syndrome" had been considered as one possible differential diagnosis, but there was no diagnostic feature shown on the magnetic resonance imaging (MRI). Caigua is being used especially to lose weight widely around the world and caigua poisoning should be kept in mind in patients having rapid onset of unexplained cachexia.

KEY WORDS: *Caigua; Cachexia; Intensive care; Ingestion; Acute disseminated encephalomyelitis*

ÖZET

Güney Amerika'da yaygın olarak yetişen ve kullanılan tropikal bir bitki olan Caigua (yabani salatalık) ile beslenme sonrasında erken dönemde görülen şiddetli kaşeksi olgusunu sunmak istedik. 15 yaşında erkek hasta hastanemiz yoğun bakım ünitesine bulantı-kusma, kas güçsüzlüğü, metabolik asidoz, solunum yetmezliği ve bilinç bulanıklığı ile kabul edildi. Hikayesinde bilinen bir özellik yoktu. Semptomları belirmeden kısa bir süre önce caigua ile beslenmişti. Hastada 1 ay içinde geliş kilosunun %20'sinden fazlasını hızla kaybettirecek şiddetli kaşeksi gözlemlendi. "Akut dissemine ensefalomyelit (ADEM) sendromu" ayırıcı tanıda düşünüldü ancak manyetik rezonans incelemesinde (MRI) bir bulguya rastlanmadı. Caigua özellikle kilo vermek için dünyada yaygın kullanılmakta olup, hızlı başlangıçlı açıklanamayan kaşeksi olgularında caigua zehirlenmesi akılda tutulmalıdır.

ANAHTAR KELİMELER: *Caigua; Kaşeksi; Yoğun bakım; Sindirim; Akut dissemine ensefalomiyelit*

INTRODUCTION

Caigua (cyclanthera pedata) is a slender tropical vine that is mostly indigenous to South America. It produces a green, semi-flattened fruit resembling cucumber (Figure 1). The fruits or leaves are extensively eaten either cooked or uncooked. It is consumed as a folk remedy for fast weight loss, control of high blood pressure, treatment of intestinal parasites, tonsillitis, gastrointestinal disorders, hyperglycemia and hyperlipidemia (1). It is available in various preparations like pills or powder. The antioxidant activity of caigua had been studied by measuring the free radical activity (2). Although there had been several local studies on caigua, no studies had been conducted in western countries which supported any of these traditional uses (3).

The term cachexia comes from the Greek root kakos hexis, which means "bad condition" (4). Cachexia is a major cause of mortality and it is thought to be the immediate cause of death in 15% of trauma and in 20% to 40% of cancer patients (5). Cytokines, testosterone, insulin-like growth factor 1, myostatin, and glucocorticoid play a role in the pathogenesis of cachexia. Myocardial infarction, congestive heart failure, chronic renal failure, chronic obstructive pulmonary disease, anorexia, rheumatoid arthritis, and AIDS are commonly associated with cachexia (6). There had been no case report on severe cachexia after caigua ingestion. We aimed to present a case with severe cachexia after caigua ingestion and discuss the pathophysiology of severe cachexia in general.

Figure 1. The fruit with the leaves

CASE REPORT

15-year-old boy was admitted to the emergency department of our hospital with acute severe nausea and vomiting, muscle weakness, metabolic acidosis, respiratory failure and loss of consciousness. His family history and past medical history were unremarkable. According to his parents, he had eaten approximately 1.2 kg of caigua 3 hours before arrival to the hospital. They had no idea if he had eaten the fruit with the leaves or not. The symptoms had begun one hour after caigua ingestion. The Glasgow Coma Scale score was 7 on arrival and the patient was admitted to the intensive care unit following orotracheal intubation. Toxic substance analysis was studied by using the toxicology panel. Ethyl alcohol, acetaminophen, salicylates, tricyclic antidepressants, digoxin, phenytoin, phenobarbital, valproic acid, and carbamazepine levels were studied in blood samples. Urine screening tests were performed to detect any poisoning of benzodiazepines and barbiturates.

Because of the possibility of herbal poisoning, a 16 F nasogastric tube was inserted and 50 g of activated charcoal was administered at a rate of 12.5 g h^{-1}. Total parenteral nutrition was started. The patient's daily calorie requirement was estimated 1602 calories per day by using the Harris-Benedict formula.

The nutritional assessment of the patient before and after admission was stated by questioning the patient's social, surgical, medical and dietary history, physical examination, body mass index, mid-arm skin fold thickness, mid-arm muscle circumference, laboratory and biochemical assessments like visceral proteins (albumin and transferrin) and daily energy requirement calculations. Thus, we determined the rising of the calorie need.

Prolonged invasive ventilation and hospital stay ensued. Progressive worsening of pulmonary functions and blood gas values were noted. Bilateral basilar pulmonary infiltrates were seen in subsequent chest X rays. Computed tomography (CT) examination revealed bilateral basal pleural effusion. Total daily energy consuming was calculated by using the ''Harris Benedict formula" during the following days. At the end of the first week, the patient's daily calorie requirement increased and supplementary enteric feeding through the nasogastric tube was started, but cachexia ensued. Acinetobacter was isolated in tracheal aspirate and blood cultures. Respiratory fatigue was noted afterwards and tracheostomy was performed. The patient lost a total of 20% of his weight. His body weight regressed to 52 kg from 65 kg. Finally, a percutaneous enterogastrostomy feeding tube was inserted for prolonged feeding at the end of two months. The patient demonstrated low level neurobehavioral responses. He was aware to environmental stimulations. His eye opening was spontaneous, he had no verbal response, but localized pain during the three months' treatment period. Acute disseminated encephalomyelitis (ADEM) was considered as a possible differential diagnosis. However, MRI did not reveal any specific signs such as multifocal central nervous system lesions. Laboratory tests revealed nonreactive for viral hepatitis, HIV, and syphilis serology. After the first month, the patient was still on supplementary oxygen in the intensive care unit (Figure 2). We planned to discharge the patient to home with Bilevel Positive Airway Pressure (BIPAP) machine. He began to gain weight by feeding with high calorie enteral nutrition.

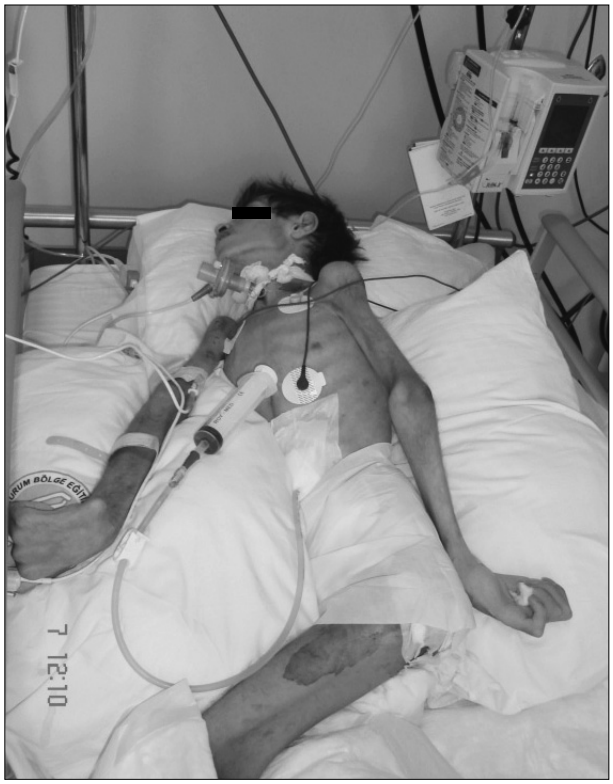

Figure 2. Cachexia following ingestion of caigua

DISCUSSION

All evaluated capsicum genus like cyclanthera pedata exhibit both hypoglicemic and hypotensive effects. The hypotensive effect is due to ''angiotensin 1 converting enzyme" inhibition. They had been used in treatment for hyperglicemia associated with type 2 diabetes mellitus (7). Liquid chromatography and mass spectrometry have been used in studies of fruits of Cyclanthera pedata scrabs (8).

We noted hypoglycemia and hypotension in this patient on arrival. This was managed by infusion of 5% Dextrose, colloid and vasopressor support. We believe that caigua ingestion was responsible for acute hypotension and hypoglicemia in this patient. Acute cachexia following caigua ingestion had not been reported before. The exact pathogenesis of acute cachexia after caigua intake is still unclear.

Caigua may cause dehydration due to diuresis. However, this cannot fully explain the onset of severe cachexia in this patient. Underlying malignancies should always be considered in patients with unexplained cachexia. Therefore whole-body CT screening was performed in this patient which was negative for malignancy. Van Heel et al (9) reported a case of acute painful diabetic neuropathy with cachexia. The patient presented with abdominal pain and severe weight loss which mimicked neoplastic disease. In our case, we had considered underlying malignancy but the whole body CT screening was negative. One the other hand, we could not diagnose ADEM in this patient.

In a clinical analysis of 125 patients, the most effective nutritional method for cancer cachexia was suggested to be a combination regimen (10). On the other hand, some special agents were found beneficial in cachexia treatment. Multiple studies demonstrated that ghrelin which is an endogenous ligand for the growth hormone receptor, has possible positive effects on nutritional management of several chronic wasting diseases such as diabetes mellitus, cancer or chronic heart failure (11). The European Society of Parenteral and Enteral Nutrition guidelines suggest that high-energy and high-protein enteral nutrition is effective for the management of cachexia. Nonetheless branched-chain amino acids like valine, leucine and isoleucine are useful (12). Therefore we closely observed the electrolyte imbalance and took precautions against glucose intolerance. Finally, we maintained frequent nutritional assessment with high-energy and high-protein enteral nutrition.

Common features in cachexia include decreases in voluntary movement, insulin resistance, anhedonia, and susceptibility to secondary infections (13-14). ADEM had been considered in this patient. In the case report by Summerfield et al (15), ADEM was noted as a paraneoplastic syndrome due to small cell lung cancer.

Different processes appear to play a role in cachexia. Weight loss and changes in body composition are closely related to acute and chronic inflammatory co-morbidities (16). Hypermetabolism, with an increase in proinflammatory cytokine turnover and reduced appetite, are seen in both cancer cachexia and cachexia due to chronic heart failure (17). Foster et al (18) reported a specific receptor subtype called ''melanocortin-4". This receptor plays a role in body weight regulation and acute antagonism of this receptor produces an increase in food intake and a decrease in metabolism. Caigua regulates the fat metabolism and decreases the levels of cholesterol in blood. Additionally, caigua seeds contain seven serine proteinase inhibitors and most of the serine proteinase inhibitors control proteolytic cascades. We concluded that proteolysis is the next step following fat metabolism during caigua food poisoning and finally cachexia appears.

This extremely rare case of caigua food poisoning with severe acute cachexia is the first report of caigua related cachexia in the literature to our knowledge. We concluded that cachexia may develop in critically ill patients after food poisoning. Therefore, this situation must be followed up closely in order to obtain an accurately management and best outcomes.

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