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Case Report
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Acute Adrenal Insufficiency Associated with Taurine Poisoning Complicated by Cardiogenic Shock: A Case Report

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Abstract

Background: Consumption of energy drinks has exploded over the last twenty years. The addition of psychoactive substances such as taurine are supposed to improve physical and mental performances.

Case Report: We report a case of a patient admitted to our medical ICU for a cardiogenic shock caused by acute adrenal insufficiency secondary to taurine intoxication. Corticosteroid therapy allowed norepinephrine and dobutamine withdrawal in 24 and 48 h, respectively.

Conclusion: Acute adrenal insufficiency is a rare cause of cardiogenic shock. Studies suggest that taurine interacts with the corticotropic axis. Very high blood levels of taurine could induce an acute adrenal deficiency indirectly inhibiting cortisol synthesis.

Keywords: Acute adrenal insufficiency; Cardiogenic shock; Taurine poisoning; Echocardiography

Abbreviations

ECAS: European Cardiac Arrhythmia Society; HPA: Hypothalamic Pituitary Adrenal; VTI LVOT: Velocity-Time Integral of Left Ventricular Outflow Tract; RAAS: Renin-Angiotensin-Aldosterone System

Introduction

Energy drinks consumption has exploded in these last decades. However, consumption is not exempt of risk [1]. Caffeine is the primary psychoactive ingredient but energy drinks usually contain other substances purported to increase performances, including taurine.

Taurine, or 2-aminoethanesulfonic acid, is an organic acid widely distributed in tissues (retina, skeletal and cardiac muscle tissue) [2]. Taurine has many biological roles: Conjugation of bile acids, osmoregulation, membrane stabilization, and modulation of calcium signaling [2]. Published relevant human clinical trials with oral taurine doses ranging from 500 mg/day to 10 g/day found no adverse effect but a significant decrease in blood pressure and serum catecholamine were observed [3].

Case Presentation

A 68-year-old patient was admitted to our medical ICU for cardiogenic shock. His medical history included a heart failure with mildly reduced (45%) ejection fraction without coronary artery disease, under triple therapy (angiotensin receptors blockers, beta-blocker, diuretics) and chronic adrenal insufficiency post-corticosteroid therapy with glucocorticoid replacement (hydrocortisone 10 mg × 2/day).

In the days preceding admission, the patient presented asthenia associated with bronchitis and diarrhea. He received an antibiotic treatment at home (amoxicillin and clavulanic acid) and consumed 2 L to 2.5 L of energy drink (Red Bull®) per day during that week. His dyspnea suddenly worsened with a clinical presentation of acute pulmonary edema. During out-of-hospital transport, the patient presented a severe circulatory failure secondary to rapid atrial fibrillation requiring an external electric shock and invasive mechanical ventilation support.

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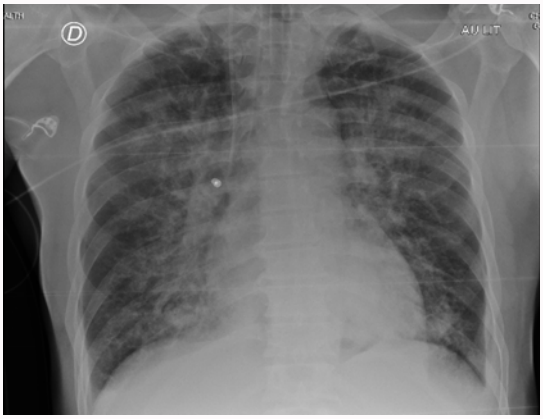

Figure 1A: Chest X-ray on patient admission.

On admission this 68-year-old patient, measuring 177 cm for 89 kg (BMI of 28), still presented signs of circulatory failure with diffuse mottling of both lower extremities, a blood pressure of 82/58 (63) mmHg and sinus tachycardia at 109 bpm, the atrial fibrillation having been reduced by the external electric shock.

The initial chest X-ray with signs of cardiac overload and the electrocardiogram showing sinus tachycardia rhythm without signs of myocardial ischemia are presented in Figure 1A, 1B.

A coronary angiography was performed and didn't show any acute coronary lesion eliminating cardiogenic shock of ischemic origin. The transthoracic echocardiography performed in ICU showed a severe and diffuse bi-ventricular hypokinesis with an estimated left ventricle ejection fraction of 10% to 15%, an increase of left ventricular filling pressures at the mitral level [elevated values of the ratio of early-to-late diastolic blood Doppler velocities and of the ratio of early blood Doppler to early tissue Doppler diastolic wave velocity (18)], a right ventricular function severely altered (peak of systolic tricuspid annulus tissue Doppler velocity of 8 cm/s). (Figures 2A-2C and Supplemental Video I). Inotrope and vasopressor supports were rapidly introduced to restore normal blood pressure. In the hypothesis of acute adrenal insufficiency secondary to a possible treatment interruption, serum cortisol and Adrenocorticotropic Hormone (ACTH) were sampled on Cath-lab per coronary angiography and a first dose of corticosteroid therapy was injected.

Initial laboratory tests didn't show any sign of biological inflammatory syndrome (CRP 5 mg/L, leukocyte counts 4,600 per cubic millimeter).

Neither the electrocardiogram nor the coronary angiography showed any sign of ischemic etiology, moreover the patient did not present any clinical or biological arguments for a sepsis, another etiology was sought.

The etiological assessment of this cardiogenic shock was a probable life-threatening adrenal crisis associated with taurine poisoning. Indeed, baseline serum cortisol level was 59 nmol/l (normal range: 170-740 nmol/l), serum ACTH level was abnormally normal 5 pmol/l (normal range: 2-11 pmol/l) and taurine urinary dosage was 354 µmol/mmol of creatinine (normal range: 16-150). The ACTH stimulation test performed the next day was in favor of corticotropic insufficiency: Cortisol level was 86 nmol/l after stimulation for a normal >550 nmol/l). Urinary catecholamines and cortisol were not measured. Moreover, the serum level of caffeine assessed at the same time was rather low: 1.6 mg/l (normal range: 8-20). There was no argument for another etiology for this severe acute heart failure, in particular no argument for a septic myocardial dysfunction.

Corticosteroid therapy allowed norepinephrine and dobutamine withdrawal within 24 and 48 h, respectively. After catecholamines weaning, myocardial dysfunction had quickly reversed and returned to baseline on check echocardiography (Figures 2D-2F and Supplemental Video I). The patient was able to be extubated at day 5 after diuretic depletion. Then, he was transferred to a cardiac rehabilitation center without recurrence of cardiac decompensation.

Discussion

Herein we report a case of cardiogenic shock caused by acute adrenal insufficiency secondary to a possible non-observance of corticosteroid substitution therapy and potentially amplified by taurine intoxication.

The literature demonstrates a substantial level of safety for supplemental taurine up to 10 g/day. In our clinical case report,

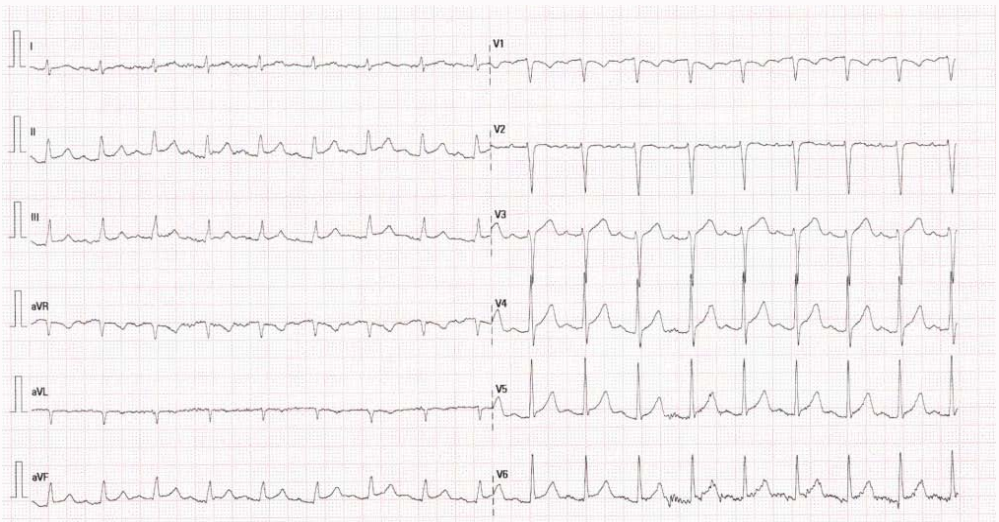

Figure 1B: Electrocardiogram on patient admission.

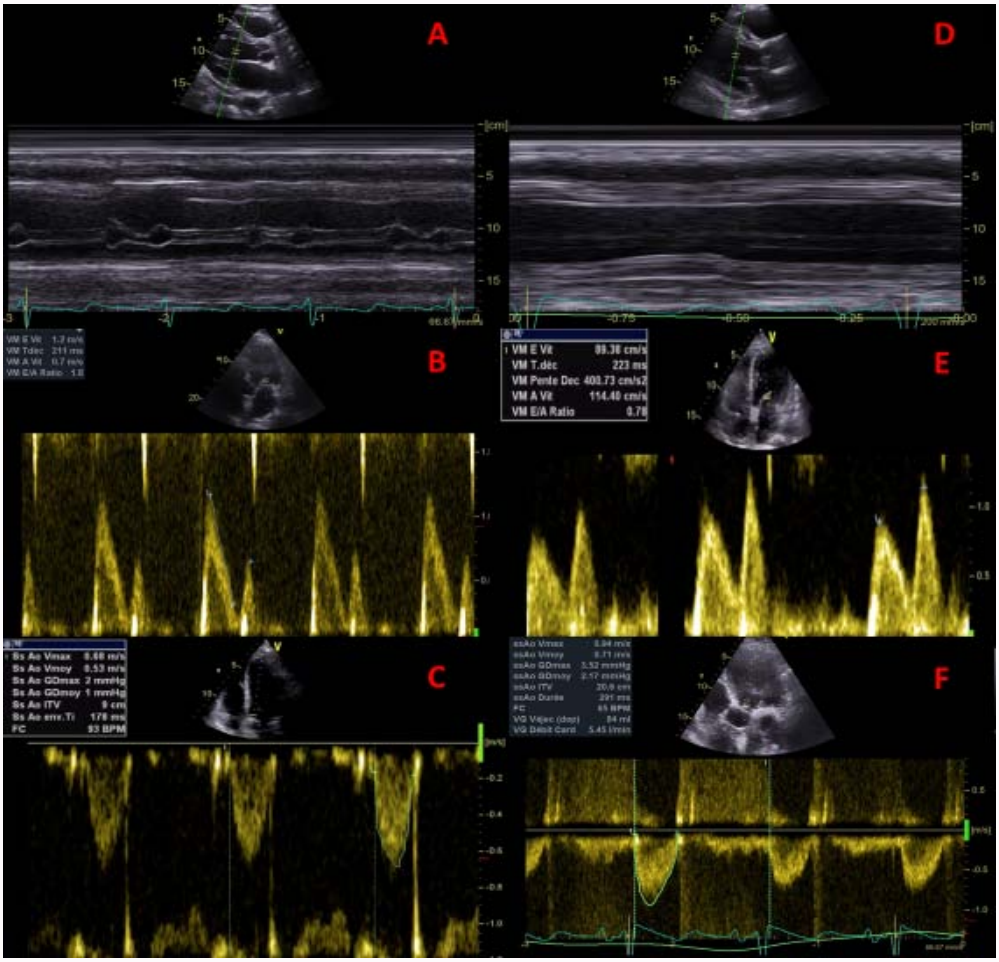

Figure 2: Echocardiographic assessments during cardiogenic shock and recovery.
A, D: Left ventricle profile in M-mode, performed in the parasternal long-axis view, during cardiogenic shock (D1) and recovery (D5).
B, E: Diastolic mitral inflow in pulsed-wave Doppler performed in apical four-chamber view, with elevated E/A ratio (1.8) during cardiogenic shock (D1) and normalized (0.8) at recovery (D5).
C, F: Velocity-time integral of left ventricular outflow tract (VTI LVOT), during cardiogenic shock (VTI LOVT =9 cm at D1) and recovery (VTI LOVT =20 cm at D5).

the patient ingested more than 10 g of taurine per day on several consecutive days.

Acute adrenal insufficiency is a rare but already described cause of cardiogenic shock [4-6]. There is a close link between cardiac function and adrenal function [7]. Several animal studies suggest that taurine interacts with the corticotropic axis [1,8,9].

Lv et al. published in 2015 a study about the effects of taurine on the Hypothalamic Pituitary Adrenal (HPA) axis in stress-induced hypertensive rats [1]. They demonstrated that the anti-hypertensive actions of taurine are related to an intervention in the classical kidney Renin-Angiotensin-Aldosterone System (RAAS), likely through elevations in angiotensin converting enzyme 2 inhibition of angiotensin 1 [9]. This interaction with the RAAS induces a decrease in glucocorticoid production from the adrenal cortex by negative feedback on the HPA axis.

In 2019, the European Cardiac Arrhythmia Society (ECAS) has undertaken a systematic and critical review of published data on twenty-two cardiovascular events related to energy drinks consumption [10]. They considered that these events were worrisome enough to raise a serious concern and to issue recommendations for better prevention.

We suggest that taurine partially inhibits cortisol synthesis but this clinical case does not allow determining a definitive causal link. More prospective studies are needed to explore this possible association.

Learning Objective

Consumption of energy drinks containing Taurine has exploded over the last twenty years. Taurine interacts with the corticotropic axis. Acute adrenal insufficiency is a rare but already described cause of cardiogenic shock.

A taurine poisoning could induce a life-threatening adrenal crisis responsible of cardiogenic shock.

Authors' Contribution

FD: Collected data, wrote the case report, FB: Performed the transthoracic echocardiography, PM: Had corrections to the manuscript, AMD: Had corrections to the manuscript. All authors have read and approved the manuscript.

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