REUE | Review article

Adverse effects of energy drinks: A systematic review

Carmen Montserrat Rodríguez-Cabrera[1,2], Begoña García Calderón[2], Álvaro García-Muñoz[3], Dima Ibrahim-Achi[2,4], Aarati Vaswani-Bulchand[1,2], Guillermo Burillo-Putze[5,6]

BACKGROUND. Energy drinks (EDs) are beverages with a high caffeine content, whose consumption has increased exponentially worldwide, although their clinical side effects remain poorly understood.

OBJECTIVE. To review the adverse effects on various organ systems potentially associated with energy drink consumption, as reported in the scientific literature.

MATERIAL AND METHODS. We conducted a PubMed search using the MeSH term "energy drink." Inclusion criteria: case reports involving humans and published in English or Spanish, from January 2000 through December 2024.

RESULTS. Among 117 analyzed cases, 38.46 % involved the cardiovascular system, mainly ischemic events (24.45 %) and arrhythmias (22.23 %). Psychiatric disorders accounted for 19.13 %, followed by neurologic (13.04 %), GI (12.17 %), renal (8.69 %), hematologic (4.35 %), and other conditions (5.22 %), including ophthalmologic and dermatologic disorders.

CONCLUSIONS. Despite their widespread consumption, few cases of adverse events related to EDs have been reported. However, these beverages pose health risks—sometimes severe—primarily cardiovascular, neurologic, and psychiatric. Long-term effects remain poorly understood.

Keywords: Energy drink. Adverse effects. Health effects. High caffeine content.

Efectos adversos de las bebidas energéticas. Revisión sistemática

INTRODUCCIÓN. El consumo de bebidas energéticas (BE) se ha incrementado en la última década, sin embargo, sus efectos adversos son escasamente conocidos.

OBJETIVO. Describir los efectos adversos secundarios al consumo de BE publicados en la literatura científica y clasificarlos en función del órgano o sistema afectado.

MATERIAL Y MÉTODOS. Se realizó una revisión en el repositorio PubMed, utilizando como término MeSH "energy drink". Dentro de los criterios de elección de los artículos se incluyó: casos clínicos, referencia a humanos, e idiomas español o inglés. El periodo de búsqueda comprendió desde enero de 2000 hasta diciembre de 2024.

RESULTADOS. De los 117 casos analizados, el 38,46 % pertenecían a la esfera cardiovascular, siendo en su mayoría eventos isquémicos (24,45 %) o arritmias (22,23 %). La patología psiquiátrica constituyó el 19,13 % de los casos, seguida por la patología neurológica (13,04 %), la patología digestiva (12,17 %), patología nefrológica (8,69 %), patologías hematológicas (4,35 %) y, finalmente, otros entidades clínicas (5,22 %) donde se englobó patología oftalmológica y dermatológica.

CONCLUSIONES. En comparación con su volumen de consumo, existen pocos casos publicados sobre eventos adversos de las BE. No obstante, éstas presentan riesgos para la salud, en ocasiones graves, fundamentalmente de tipo cardiovascular, neurológico y psiquiátrico. Los efectos a largo plazo son poco conocidos.

Palabras clave: Bebida energética. Efectos adversos. Efectos sobre salud. Alto contenido en cafeína.

Introduction

The emergence of energy drinks (EDs) in the market began in 1960 in Japan and subsequently in Austria, with the creation of the brand *Red Bull* in 1987, where their popularity increased in Europe, and later in 1997 in the United States.[1,2]

As this is a commercial designation, there is currently no exact definition of EDs. Nevertheless, they can be defined as a type of non-alcoholic stimulant beverages, with a high caffeine content and other ingredients, whose purpose is to improve performance and enhance concentra-

Author Affiliations: [1]Programa de Doctorado en Ciencias de la Salud, Universidad de La Laguna, Tenerife, Spain. [2]Servicio de Urgencias, Hospital Universitario de Canarias, Tenerife, Spain. [3]Fundación Canaria Instituto de Investigación Sanitaria de Canarias, Tenerife, Spain. [4]Universidad Europea Canarias, La Orotava, Tenerife, Spain. [5]Red de Investigación en Atención Primaria de Adicciones (RIAPAD). [6]Departamento de Medicina Física y Farmacología, Universidad de La Laguna, Tenerife, Spain.
Corresponding Author: Carmen Montserrat Rodríguez Cabrera. Servicio de Urgencias. Hospital Universitario de Canarias. Carretera Ofra, s/n. 38320 La Laguna, Tenerife, Spain.
E-mail: montserodcab@gmail.com
Article Information: Received: 4-7-2025. Accepted: 7-10-2025. *Online:* 27-11-2025.
Editor in Charge: Montserrat Amigó Tadín.

tion capacity, especially in the academic setting.[4-6] For its part, the Plan Nacional sobre Drogas (PND) defines EDs as "non-alcoholic beverages, generally carbonated, composed mainly of caffeine and carbohydrates, amino acids, vitamins, minerals, plant extracts, accompanied by additives such as preservatives, flavorings, and colorants".[7]

Currently, there are more than 500 brands of EDs on the market,[8] with a business volume that has grown significantly, increasing from 12 billion dollars in 2012 to 45.8 billion in 2020.[1,9] In addition to their high caffeine content, these beverages contain guarana, taurine, ginseng, B-complex vitamins, L-carnitine, as well as sugar, among others, in different quantities and proportions.[10,11] They are mainly marketed as cans ranging from 250 to 710 mL, although in recent years the sale of so-called "energy shots" (bombs or "pelotazos") of approximately 60 mL has become popular.

According to the European Food Safety Authority (EFSA),[14-16] the recommended limits for daily caffeine intake are 400 mg for adults, 200 mg for pregnant women, and 100 mg (2.5 mg/kg/day) for adolescents and children. Common sources of caffeine include tea, with approximately 30 mg per 250 mL, cola soft drinks with 34.5 mg per can, and a cup of coffee containing around 40 mg of caffeine. EDs show wide variability in their caffeine content, with concentrations ranging from 80 to 505 mg per container. Considering that in many cases intake is not limited to a single unit per day, it is common to exceed the maximum recommended daily caffeine dose.

Regarding consumption patterns, in 2023 a systematic review including studies from 50 countries, including Spain, was published, showing that at least 54.7 % of the studied population had consumed EDs at some point, with 8.82 % daily consumption, and higher consumption in North America compared to Europe, especially among adolescents and young adults.[18]

At the national level, according to the ESTUDES 2023 survey of the PND, aimed at adolescents and young people aged 14 to 18 years, 47.7 % of respondents had consumed EDs in the last 30 days, with higher prevalence in males. Notably, 19.5 % reported co-ingestion with alcohol.[19] On the other hand, in the EDADES 2022 survey of the PND, aimed at individuals aged 15 to 64 years, the prevalence of consumption decreased to 14.2 %, with a higher consumption rate observed in the 15–24 age group.[20]

Aware of the usual consumption profile, ED manufacturers design advertising campaigns targeted at young audiences by promoting activities attractive at these ages, such as extreme sports, video games, and music events, using appealing images and messages.[23,24] Another aspect to consider is their accessibility on supermarket shelves, gas stations, and large retail stores, as well as their price, which is well adjusted to the economic capacity of young people, including promotions of multiple units at very low prices.[25,26]

To limit the consumption of these products among young populations, countries such as Lithuania (2014), Latvia (2016), and Turkey (2018) have banned the sale of EDs to individuals younger than 18 years of age, and in the case of Sweden, sales are restricted to those younger than 15 years. In Europe, since December 2014, all beverages containing > 150 mg of caffeine per liter must be labeled with the statement: "High caffeine content: Not recommended for children or pregnant or breastfeeding women".[27] Similarly, various pediatric scientific societies recommend limiting the sale of EDs to individuals younger than 12 years or even younger than 18 years.

The need to restrict ED consumption arises from evidence of their potential adverse effects (mainly cardiovascular and neurological),[29] which translates into an increase in visits to hospital emergency departments (HEDs). In this regard, the 2013 report from the Drug Abuse Warning Network (DAWN) indicated an increase in emergency visits secondary to adverse effects of EDs, rising from 6,996 cases in 2007 to 14,042 in 2011, and from 3,060 to 6,090 in relation to their combined use with illicit drugs or alcohol, with the most affected population being those aged between 18 and 39 years.[30]

The objective of this study is to perform a systematic review of acute adverse effects in different organs and systems associated with ED consumption reported in the scientific literature.

Material and methods

Following the PRISMA (Preferred Reporting Items for Systematic Review and Meta-Analyses) method,[31] we conducted a literature review in the PubMed repository using the MeSH term "energy drinks". The selection criteria included: case reports, effects on humans, and articles published in Spanish or English between January 2000 and December 2024.

A backward search based on the references of the initially obtained articles was also performed, as well as an additional search using the term "energy drinks" as free text (non-MeSH).

Two reviewers conducted the search, followed by reading of abstracts or full-text articles, excluding those that did not meet the inclusion criteria. Disagreements were resolved by consensus.

Sociodemographic data, amount consumed (number of cans), associated clinical features, as well as the organ or clinical domain involved were extracted.

The search using MeSH, free text, and backward strategy yielded a total of 276 articles, of which 121 were removed due to duplication, resulting in 155 manuscripts. In a second review, 2 articles were excluded as they were case reviews. Subsequently, 153 articles were assessed through abstract or full-text reading, excluding 55 articles for not meeting the objective and inclusion criteria. Finally, 98 articles were included in the review. Since most texts referred to clinical cases, the level of evidence was not assessed (Figure 1).

Results

From the 98 selected articles, the data presented in Table 1 were extracted. Data from 115 patients were ob-

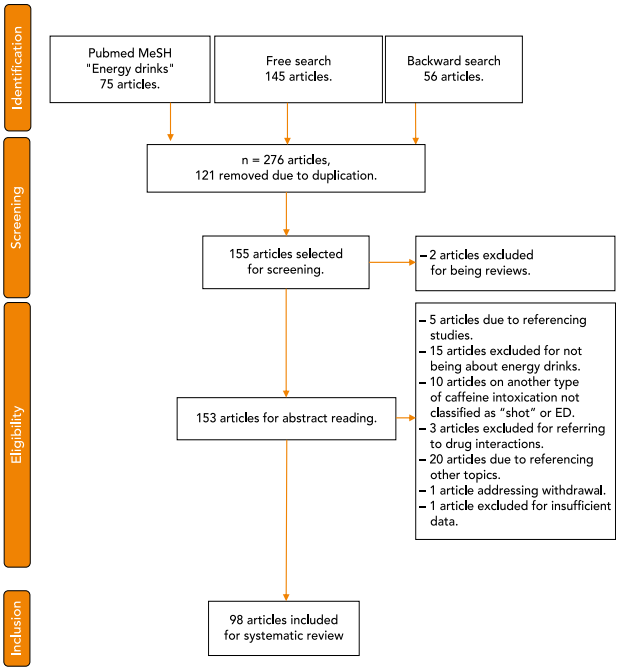

Figure 1. PRISMA flow diagram for article selection.

tained, since some articles included > 1 case, and a total of 117 identifiable disease were reported, as 2 patients presented conditions affecting different systems, which were counted separately.

The mean age of the published cases was 29.47 ± 12.26 years, ranging from an 8-year-old child (epileptic seizure)[188] to 76 years (a patient with hypercobalaminemia).[153] Males accounted for 80 % of cases.[18,19] The mean consumption was 5.5 cans.

Regarding affected organs and systems, cardiovascular effects were the most frequent (38.46 %), including cases of coronary ischemia[127,136,138,152,159,169,179,183,187,205,207] (24.45 %); arrhythmias such as ventricular fibrillation or ventricular tachycardia[116,122,137,140,142,148,155,178,193] and atrial fibrillation and flutter[126,134,139,171,181,197,207] (both 22.23 %); dilated cardiomyopathy[190,200,201,208] (8.89 %); and, less frequently, aortic dissection,[151] postural tachycardia syndrome,[121] supraventricular tachycardia,[120] long QT syndrome,[141,206] pediatric hypertension,[146] acute myocarditis,[198] and Takotsubo syndrome.[147]

Secondly, psychiatric disease accounted for 18.80 % of cases, with psychotic episodes[119,123,125,154,175,192,210] and manic episodes[115,128,158,160,168,182,184] being the most prevalent (40.9 % and 36.36 %, respectively), followed by anxiety and depressive symptoms (4.5 % each).

Neurological effects accounted for 12.82 %, with 53.34 % being seizure cases,[118,131,132,143,188] 26.67 % ischemic events,[150,165,202,209] and 20 % including aneurysm rupture,[135] reversible cerebral vasoconstriction syndrome,[166] and subarachnoid hemorrhage.[173]

Approximately 12 % of cases corresponded to digestive conditions, mainly hepatitis[129,130,161,174,191,196] and pancreatitis[163,185,186,194,204,210] (both 42.85 %). Additionally, 1 case of hypercobalaminemia[153] and 1 of atrophic gastritis with intestinal metaplasia[199] were described.

Renal signs accounted for 8.55 % of cases, mostly consisting of electrolyte disturbances[124,164,167,177] or acute kidney failure[133,162,192,196] (40 % each), and 20 % cases of rhabdomyolysis.[170,203] Hematological disorders were observed in 4.27 % of cases, with 40 % presenting hemorrhage[145] and 40 % thrombotic thrombocytopenic purpura,[195] and one case of acquired vitamin K deficiency.[180]

Finally, in the "others" category (5.13 %), 3 ophthalmological cases were grouped (bilateral scotoma due to vasoconstriction,[156] acute macular neuroretinopathy,[189] and intraretinal hemorrhage[176]), and three dermatological cases (allergic reaction,[157] erythema multiforme,[172] and eccrine chromhidrosis related to food coloring[212]).

Discussion

Currently, the mechanism by which these adverse effects of EDs occur is not entirely clear, although they appear to be due more to the mixture of ED components than to the effect of their ingredients separately;[32] therefore, the possible causes according to the affected system are outlined below.

Table 1. Adverse effects reported in the scientific literature after consumption of energy drinks, grouped by organ systems

	Total	Cardiology	Psychiatry	Neurology	Digestive System	Nephrology	Hematology	Others
N (%)	117	45 (38.46)	22 (18.80)	15 (12.82)	14 (11.97)	10 (8.55)	5 (4.27)	6 (5.13)
Age – years [mean (SD)] (range)	29.47 12.26 (8-76)	26.09 9.57 (13-54)	31.59 12.42 (17-69)	24.93 9.90 (8-44)	40.35 11.80 (16-76)	34.3 12.257 (17-62)	26.4 9.633 (18-41)	33 12.55 (19-48)
Male sex (%)	80	86.67	68.18	86.67	84.62	77.68	60	66.67
Cans consumed – mean (range)	5.49 (1-48)	6.23 (1-48)	6.58 (1-20)	3.6 (2-6)	3.82 (0.5-10)	6.17 (2-12)	2 (1.5-2.5)	3.17 (1-5.5)
Diagnoses (N)		AMI: 11 VF/VT: 10 AF/Flutter: 10 Dilated cardiomyopathy:4 Other: 10	Psychotic episode: 9 Manic episode: 8 Anxiety: 1 Depression:1 Other: 3	Seizure: 8 Stroke: 4 Other: 3	Hepatitis: 6 Pancreatitis:6 Other: 2	AKI: 4 Rhabdomyolysis:2 Electrolyte disturbances: 4	TTP: 2 Bleeding: 2 Other: 1	Ophthalmologic:3 Dermatologic: 3
Habitual consumption (%)	42.6	33.3	50	40	61.5	22.22	80	50

SD: standard deviation; AMI: acute myocardial infarction; VF: ventricular fibrillation; VT: ventricular tachycardia; AF: atrial fibrillation; AKI: acute kidney injury; TTP: thrombotic thrombocytopenic purpura.

Effects on the cardiovascular system

ED-related cardiovascular effects are, without doubt, the most studied and the most concerning because of their potential mortality, with caffeine being one of the components most strongly associated with this type of adverse reaction, especially considering that it is the most widely consumed psychostimulant substance in the world.[33]

Caffeine is rapidly absorbed and has an approximate half-life of 4.5 hours, which may increase in some individuals, in smokers, and with co-ingestion of alcohol. It is metabolized in the liver into a methylxanthine,[34-36] and its effects are related to an increase in catecholamines, an increase in calcium volume, and the inhibitory effects on adenosine A1 and A2A receptors, thereby producing an increase in blood pressure, an increase in heart rate, and an increase in lipid profile, among others.[37-40]

Caffeine does not appear to precipitate arrhythmias such as atrial fibrillation, and in some cases and depending on the dose, it acts as a protective factor against it and against other cardiovascular risk factors,[41-43] but these results should be interpreted with caution because of the different doses, habits, and dietary factors, as well as other external factors[44] or underlying diseases and individual-specific alterations.[45,46] In contrast, an increase in malignant arrhythmias has been demonstrated with high doses of caffeine in animal models, probably because very high doses and more invasive methods are used in those experiments than in human studies.[47,48]

Among the components of EDs is guarana, a plant from the Amazon that has been used medicinally in that region for centuries.[49]

Guarana contains more caffeine than coffee (up to 6 %),[50] so its consumption could increase caffeine levels above recommended amounts, in addition to interacting with different drugs and producing effects ranging from agitation and anxiety to arrhythmias,[51] together with other side effects attributable to guarana itself, such as coagulation abnormalities.

Moreover, taurine has been associated with the risk of cardiac disease, especially in combination with caffeine in Eds,[52,53] although studies evaluating taurine in isolation have shown beneficial effects.[54]

Effects in the psychiatric sphere

The effect of caffeine in relation to this type of disease is believed to be linked to blockade of both A1 and A2 receptors, which are closely related to the control of sleep, memory, and anxiety, thereby producing the opposite effect, with an increase in dopaminergic activity[65] that, along with other components of EDs such as taurine,[66] ginseng,[67] and inositol,[68] could be related to these symptoms.

Within psychiatric disease, caffeinism is found, which is caffeine intoxication including GI symptoms and others such as nervousness, excitation, hurried thinking, and pressured speech, which resemble anxiety attacks.[56,57] Furthermore, caffeine has been associated with anxiety disorders, sleep disorders, and eating disorders, in the same way that its role in worsening panic attacks and anxiety has been described when consumed at doses between 400 mg and 750 mg, both in patients with a past medical history of panic disorder and in those without such history.[58]

Overall, patients with psychiatric disease, especially those diagnosed with schizophrenia or bipolar disorder, have higher caffeine consumption,[55,56] a fact to be considered given that EDs may exacerbate the positive symptoms of these 2 clinical entities.[59-61] On the other hand, caffeine, like some antipsychotic drugs, is metabolized by cytochrome P450 1A2 (CYP1A2),[64] so its consumption may decrease the plasma levels of some drugs such as clozapine[62] and lithium.[63]

Neurological sphere

Regarding neurological effects, seizure episodes are the most frequent, and there are several theories and mechanisms by which EDs cause this type of disease, with caffeine acting as a triggering factor for epileptic seizures, both in animals[69] and humans, in whom an increase in catecholamine levels could produce a reduction in the anticonvulsant effect of adenosine.[70] Pharmacological interactions with anticonvulsant drugs, insomnia,[71,72] and hypokalemia should also be considered epileptogenic factors.[75]

On the other hand, taurine has more contradictory neurological effects. Although its neuroprotective properties have been demonstrated in relation to the reduction of free intracellular calcium concentration and its antioxidant capacity,[76] in an animal study conducted by El Idrissi *et al.*, after oral and IV administration of taurine, it was observed that mice given IV taurine had fewer seizure-related lesions, whereas those that had received oral taurine had more severe seizures.[77,78] Trials in humans have not shown adverse effects after consumption of 20-200 mg/day in the diet.[79]

The present study highlights the possible relationship between ED consumption and stroke, with 5 cases, probably related to the combined consumption of taurine and caffeine, producing cerebral vasospasm in patients with predisposing characteristics, such as structural heart disease or atherosclerosis, among others.[80] In the case of caffeine, catecholamine release and its ionotropic effect have been proposed as the pathophysiological mechanism and, in the case of taurine, its similar inotropic effects, as well as an increase in platelet aggregation.[81,127] Once again, the causes of this pathology and the use of these beverages remain unclear.

Digestive sphere

As shown in Table 1, most of the published cases within the digestive system corresponded to hepatitis. The effect of niacin (vitamin B3), an ingredient of this type of beverage, as well as of other over-the-counter vitamin supplements, as a cause of this condition is well known.[82] In contrast, niacin has shown a favorable effect in the management of dyslipidemia, reducing triglyceride levels, low-density lipoproteins (LDL), and very-low-density lipoproteins (VLDL), as well as increasing high-density lipoprotein (HDL) levels.[83]

Other side effects of vitamin B3[83-85] include flushing, acanthosis nigricans, metabolic effects such as increased glucose and uric acid levels, and gastrointestinal effects, ranging from minor symptoms such as abdominal discomfort, nausea, and vomiting to increased liver enzyme levels and hepatitis.[86] The latter entity has been associated with the dose consumed, occurring mainly with doses greater than 2,000 mg/day,[87] although it may occur at any dose and is also time-dependent,[88] being associated with the amidation reaction and subsequent degradation into nicotinamide and pyrimidine,[89] both hepatotoxic.

Pantothenic acid is a water-soluble vitamin present in EDs with potential gastrointestinal effects.[90]

Regarding pancreatitis, studies in animal models have demonstrated pancreatic involvement secondary to ED consumption.[91,92] All this suggests that individuals with a past medical history of liver transplantation, unknown mutations, or concomitant treatments should be aware of the risk of developing some of these digestive disorders in relation to ED consumption.[93,94]

Nephrological sphere

As already noted above, excessive caffeine consumption increases intracellular potassium concentration because of its effect on the Na^+/K^+ pump.[95-97] In addition to hypokalemia, its natriuretic and diuretic effect has also been reported,[98,99] probably in relation to its combination with taurine.[100]

Regarding the possible causes of rhabdomyolysis, caffeine can increase intracellular calcium volume, which over time may lead to sarcoplasmic damage,[101] although published cases are usually related to high ED consumption.[102]

On the other hand, a possible nephrotoxic effect of taurine due to altered renal flow has been postulated. However, other studies indicate a possible protective role of taurine in renal pathology,[103] although in patients on hemodialysis and in other chronic kidney disease patients, taurine may increase, which may negatively affect their health.[104]

Hematological sphere

In the literature, the main ED components producing hematological disorders are the different added supplements and not the principal components, with 2 having been mainly associated with both increased bleeding and thrombotic thrombocytopenic purpura (TTP). A relationship has been observed between ginseng and platelet inhibition, an increase in both prothrombin time and partial thromboplastin activity, as well as irreversible antiplatelet activity in humans.[105] As for guarana, apart from its high caffeine content, its effect on decreasing platelet aggregation appears to be related to decreased thromboxane.[106] Moreover, *ginko biloba* has been associated with hemorrhagic diathesis and platelet function abnormalities.[105,107] In addition, ED *per se* has demonstrated platelet and endothelial function alterations that could produce TTP,[81] a decrease in erythrocyte and platelet volume,[108] as well as a reduction in prothrombin time.[109]

Ophthalmological sphere

This study includes 3 cases within this area: transient vascular ischemia due to retinochoroidal vasoconstriction, acute macular neuroretinopathy, and intraretinal hemorrhage, which could be related to the effects of caffeine with respect to increased peripheral vascular resistance.[81,110,111] Cases of niacin-induced macular edema have also been published.[112]

Other clinical considerations

Most of the reviews we found focus on one specific organic sphere; however, when comparing our findings with other reviews, the results are similar. A 2015 review[113] had already shown that most adverse events were related to the cardiological sphere and, within it, to different types of arrhythmias, a result similar to that obtained in our review (52 %). On the other hand, it also discussed the neurological sphere and the possible relationship of EDs with epileptic seizures, which was also consistent with the frequency of cases found by us within this group (29 %). The aforementioned review did not include cases from the gastrointestinal sphere.

More recently, Constantino *et al*. conducted a review[114] in which, again and in line with our results and previous findings, cases of cardiovascular pathology were the most numerous (47.7 %), with arrhythmias ranking first. Within cardiac pathology, their classification differs from ours, since in our study we did not include death or cardiorespiratory arrest within the diagnosis, but rather the pathology that triggered that outcome, thus obtaining a greater number of ischemic events and malignant arrhythmias compared with their review.

On the other hand, our classification distinguishes between the psychiatric and neurological spheres, obtaining a higher number of cases in the former, whereas in that review, within the "neuropsychiatric" sphere, they reported a total of 22 cases (25.7 %) vs the 37 cases (30.77 %) we obtained in our review when the 2 specialties were combined. However, for both the GI and renal spheres, we obtained similar results.

Limitations

This review article has a series of limitations: first, the search was conducted using a single database (PubMed), which may have excluded some cases and series recorded only in other repositories. However, we consider that the highest-quality or most relevant cases are usually published in journals indexed in this repository. Second, the heterogeneity of each article when indicating the amount of ED consumed or the type of ED ingested may have overestimated or underestimated data related to usual consumption or the amount of beverage ingested when comparing them with one another. In fact, it is possible that the concentration of components and their proportion varied over the years covered by the search. Even so, we believe this does not diminish the value of the review, but rather allows its effects to be viewed from a temporal perspective, although in this regard an assessment of the temporal evolu-

tion of the published cases would be necessary. It is possible that, with the increase in consumption, mild cases or those occurring in healthcare settings where care for patients with these acute effects is very frequent are not published, as we have already indicated.

Finally, although in many of the published cases other causes that could have produced the described condition were ruled out, it is possible that in some cases the relationship between EDs and the clinical event cannot always be confirmed, nor can its possible relationship with the patient's previous condition.

What is striking in this review is the scarcity of studies on adverse effects of EDs published to date, especially considering their widespread consumption, which may be considered universal with respect to ethnic patterns, age, sex, socioeconomic level, etc. This is also notable in relation to the proliferation of new commercial brands and new ingredients, and the declared increase in their consumption in different national and European surveys. It is possible that either their consumption is not asked about in emergency care, their causal association is not considered relevant, or sufficient scientific importance is not attributed to it for publication. In this regard, we found no cases of metabolic type, such as hyperglycemia, despite the high glucose content of many of these EDs. Something similar in terms of underdiagnosis and lack of scientific publications may also be occurring with the possible chronic effects of their consumption, beyond the pediatric sphere.

Conclusions

EDs entail health risks, mainly cardiovascular, neurological, and psychiatric, based on the published cases, with special emphasis on more exhaustive monitoring of consumption in susceptible populations (children, adolescents, patients with pre-existing diseases) because of the adverse effects observed in the short term and the unknown effects in the long term.

In addition, young subjects should be informed of their risks, both when consumed alone and when co-ingested with other substances, including alcohol and other types of drugs, as well as the possible additive effects this may entail.

As clinicians, we must first investigate their consumption in emergency care in cases of clinical manifestations attributable to their composition and acute toxic-dose or chronic consumption and inform patients about the effects of their consumption as part of primary prevention. Similarly, we believe that it is necessary to continue investigating the possible causes of the different adverse effects and to develop large, prospective studies to determine their acute effects through active case finding.

ARTICLE INFORMATION

Conflict of Interest Disclosures: None reported.

Funding: The authors declare the non-existence of funding in relation to this article.

Ethical Responsibilities: The authors have confirmed the maintenance of confidentiality and respect for the patient rights, agreement of publication, and transfer of rights to Revista Española de Urgencias y Emergencias.

Data Availability: The data are available upon request to the corresponding author.

Author Contributions (CRediT): CMRC: Conceptualization, methodology, data collection, supervision, writing of the initial draft, review and editing of the final article, project administration, and validation thereof. BGC: data collection, review, and editing of the final article. DIA: Conceptualization, methodology, review, supervision, writing of the initial draft, and editing of the final article. AGM: data collection, review, and editing of the article. AVB: conceptualization, methodology, data collection, writing of the initial draft, and editing of the final article. GBP: Conceptualization, methodology, data collection, supervision, writing of the initial draft, review and editing of the final article, project administration, and validation thereof.

Use of Generative Artificial Intelligence Tools: The authors declare that they did not use AI tools in the preparation of this article.

Article not commissioned by the Editorial Board and with external peer review.

Note of the editors: This is a BOWMAN-generated English translation of the officially indexed Spanish-language article, which should be cited as Rev Esp Urg Emerg. 2026;5:112-133. In this translated version, the editors have supervised the process; however, it cannot be ruled out that some errors resulting from the artificial intelligence translation process may have gone unnoticed.

REFERENCES

1. Zucconi S, Volpatoa C, Adinolfia F, Gandini A, Gentile E, Loi A, et al. Gathering consumption data on specific consumer groups of energy drinks. EFSA Support Publ. 2013;10:1-10.
2. Richards G, Smith AP. A review of energy drinks and mental health, with a focus on stress, anxiety, and depression. J Caffeine Res. 2016;6:49-63.
3. Gheith I. Clinical Pathology of caffeinated and non-caffeinated energy drinks: Review. Life Sci J. 2017;14:21-36.
4. Bailey RL, Saldanha LG, Gahche JJ, Dwyer JT. Estimating caffeine intake from energy drinks and dietary supplements in the United States. Nutr Rev. 2014;72:9-13.
5. Lévy S, Santini L, Capucci A, Oto A, Santomauro M, Riganti C, et al. European Cardiac Arrhythmia Society Statement on the cardiovascular events associated with the use or abuse of energy drinks. J Interv Card Electrophysiol. 2019;56:99-115.
6. Lippi G, Cervellin G, Sanchis-Gomar F. Energy drinks and myocardial ischemia: A review of case reports. Cardiovasc Toxicol. 2016;16:207-12.
7. Portal Plan Nacional Sobre Drogas [Internet]. Spain: Ministerio de Sanidad. (Accessed 1 July 2025). Available at: https://pnsd.sanidad.gob.es/ciudadanos/informacion/bebidasenergeticas/home.htm
8. Reissig CJ, Strain EC, Griffiths RR. Caffeinated energy drinks—A growing problem. Drug Alcohol Depend. 2009;99:1-10.
9. Ajibo C, Van Griethuysen A, Visram S, Lake AA. Consumption of energy drinks by children and young people: a systematic review examining evidence of physical effects and consumer attitudes. Public Health. 2024;227:274-81.
10. Clauson KA, Shields KM, McQueen CE, Persad N. Safety issues associated with commercially available energy drinks. J Am Pharm Assoc. 2008;48:e55-e67.
11. Higgins JP, Tuttle TD, Higgins CL. Energy beverages: Content and safety. Mayo Clin Proc. 2010;85:1033-41.
12. Cao DX, Maiton K, Nasir JM, Estes NAM, Shah SA. Energy drink-associated electrophysiological and ischemic abnormalities: A narrative review. Front Cardiovasc Med. 2021;8:679105.
13. Oddy WH, O'Sullivan TA. Energy drinks for children and adolescents. BMJ. 2009;339:b5268-b5268.
14. Seifert SM, Seifert SA, Schaechter JL, Bronstein AC, Benson BE, Hershorin ER, et al. An analysis of energy-drink toxicity in the National Poison Data System. Clinical Toxicology. 2013;51:566-74.
15. Ehlers A, Marakis G, Lampen A, Hirsch-Ernst KI. Risk assessment of energy drinks with focus on cardiovascular parameters and energy drink consumption in Europe. Food Chem Toxicol. 2019;130:109-21.
16. Higgins JP, Yarlagadda S, Yang B. Cardiovascular Complications of Energy Drinks. Beverages. 2015;1:104-26.
17. Arria AM, Bugbee BA, Caldeira KM, Vincent KB. Energy drink use and high-risk behaviors: Research evidence and knowledge gaps. Nutr Rev. 2014;72:87-97.
18. Aonso-Diego G, Krotter A, García-Pérez A. Prevalence of energy drink consumption world-wide: A systematic review and meta-analysis. Adicction. 2024;119:438-63.

19. Observatorio Español de las Drogas y las Adicciones. Encuesta sobre uso de drogas en Enseñanzas Secundarias en Spain. ESTUDES 2023. (Accessed 27 January 2024). Available at: https://pnsd.sanidad.gob.es/profesionales/sistemasInformacion/sistemaInformacion/encuestas_ESTUDES.htm.
20. Observatorio Español de las Drogas y las Adicciones. Encuesta sobre Alcohol y Drogas en población general en Spain. EDADES 2022. (Accessed 27 January 2024). Available at: https://pnsd.sanidad.gob.es/profesionales/sistemasInformacion/sistemaInformacion/encuestas_EDADES.htm.
21. Heckman MA, Sherry K, De Mejia EG. Energy drinks: An assessment of their market size, consumer demographics, ingredient profile, functionality, and regulations in the United States. Compr Rev Food Sci Food Saf. 2010;9:303-17.
22. Visram S, Crossley SJ, Cheetham M, Lake A. Children and young people's perceptions of energy drinks: A qualitative study. PLoS One. 2017;12:e0188668.
23. Nordt P, Claudius I, Rangan C, Armijo E, Milano P, Yanger S, et al. Reasons for energy drink use and reported adverse effects among adolescent emergency department patients. Pediatr Emerg Care. 2017;33:770-3.
24. Ayalde J, Ta D, Adesanya O, Mandzufas J, Lombardi K, Trapp G. Awake and alert: Examining the portrayal of energy drinks on TikTok. J Adolesc Health. 2023;72:633-5.
25. Al-Shaar L, Vercammen K, Lu C, Richardson S, Tamez M, Mattei J. Health effects and public health concerns of energy drink consumption in the United States: A mini-review. Front. Public Health. 2017;5:225.
26. Mamiya H, Moodie EEM, Schmidt AM, Ma Y, Buckeridge DL. Price discounting as a hidden risk factor of energy drink consumption. Can J Public Health. 2021;112:638-46.
27. Regulation [EU] No 1169/2011 of the European Parliament and of the Council of 25 October 2011 on the provision of food information of consumers, amending Regulations [EC] No 1924/2006 and [EC] No 1925/2006 of the European Parliament and of the Council, and repealing Commission Directive 85/250/EEC, Council Directive 90/496/EEC, Comision Directive 1999/10/EC, Directive 200/13/EC of the European Parliament and of the Council, Commission Directives 2002/67/EC and 2008/5/EC and Commission Regulation [EC] No 608/2004;304. (Accessed 20 December 2023). Available at: https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:32011R1169&from=en.
28. Committee on Nutrition and the Council on Sports Medicine and Fitness. Sports drinks and energy drinks for children and adolescents: are they appropriate? Pediatrics. 2011;127:1182-9.
29. Hussain A, Jiji AK, Barke P, Biswas S, Tabrez SSM. Cardiovascular pathologies associated with excessive energy drink consumption: A review. Crit Rev Eukaryot Gene Expr. 2018;28:107-13.
30. Mattson ME. Update on emergency department visits involving energy drinks: A continuing public health concern. 2013. (Accessed 18 December 2023). Available at: https://pubmed.ncbi.nlm.nih.gov/27606410/.
31. Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. PLoS Med. 2009;6:e1000097.
32. Gutiérrez-Hellín J, Varillas-Delgado D. Energy Drinks and Sports Performance, Cardiovascular Risk, and Genetic Associations; Future Prospects. Nutrients. 2021;13:715.
33. Fredholm BB, Bättig K, Holmén J, Nehlig A, Zvartau EE. Actions of caffeine in the brain with special reference to factors that contribute to its widespread use. Pharmacol Rev. 1999;51:83-133.
34. Blanchard J, Sawers SJ. The absolute bioavailability of caffeine in man. Eur J Clin Pharmacol. 1983;24:93-8.
35. Cornish HH, Christman AA. A study of the metabolism of theobromine, theophylline, and caffeine in man. J Biol Chem. 1957;228:315-23.
36. Fligner CL, Opheim KE. Caffeine and its dimethylxanthine metabolites in two cases of caffeine overdose: a cause of falsely elevated theophylline concentrations in serum. J Anal Toxicol. 1988;12:339-43.
37. O'Keefe JH, Bhatti SK, Patil HR, DiNicolantonio JJ, Lucan SC, Lavie CJ. Effects of habitual coffee consumption on cardiometabolic disease, cardiovascular health, and all-cause mortality. J Am Coll Cardiol. 2013;62:1043-51.
38. Islam R, Ahmed M, Ullah W, Tahir YB, Gul S, Hussain N, et al. Islam H. Effect of Caffeine in Hypertension. Curr Probl Cardiol. 2023;48:101892.
39. De Giuseppe R, Di Napoli I, Granata F, Mottolese A, Cena H. Caffeine and blood pressure: a critical review perspective. Nutr Res Rev. 2019;32:169-75.
40. Godos J, Pluchinotta FR, Marventano S, Buscemi S, Li Volti G, Galvano F et al. Coffee components and cardiovascular risk: beneficial and detrimental effects. Int J Food Sci Nutr. 2014;65:925-36.
41. Ding M, Bhupathiraju SN, Satija A, van Dam RM, Hu FB. Long-term coffee consumption and risk of cardiovascular disease: a systematic review and a dose-response meta-analysis of prospective cohort studies. Circulation. 2014;129:643-59.
42. Mattioli AV. Caffeine and atrial fibrillation. En: Coffee in Health and Disease Prevention. Elsevier; 2015. p. 691-698.
43. Surma S, Romanczyk M, Filipiak KJ, Lip GYH. Coffee and cardiac arrhythmias: Up-date review of the literature and clinical studies. Cardiol J. 2023;30:654-67.
44. Khiali S, Agabalazadeh A, Sahrai H, Bannazadeh Baghi H, Rahbari Banaeian G, Entezari-Maleki T. Effect of Caffeine Consumption on Cardiovascular Disease: An Updated Review. Pharmaceut Med. 2023;37:139-51.
45. Dobmeyer DJ, Stine RA, Leier CV, Greenberg R, Schaal SF. The arrhythmogenic effects of caffeine in human beings. N Engl J Med. 1983;308:814-6.
46. Poole R, Kennedy OJ, Roderick P, Fallowfield JA, Hayes PC, Parkes J. Coffee consumption and health: umbrella review of meta-analyses of multiple health outcomes. BMJ. 2017;359:j5024.
47. Pelchovitz DJ, Goldberger JJ. Caffeine and cardiac arrhythmias: a review of the evidence. Am J Med. 2011;124:284-9.
48. Zuchinali P, Ribeiro PA, Pimentel M, da Rosa PR, Zimerman LI, Rohde LE. Effect of caffeine on ventricular arrhythmia: a systematic review and meta-analysis of experimental and clinical studies. Europace. 2016;18:257-66.
49. Schimpl FC, da Silva JF, Gonçalves JF, Mazzafera P. Guarana: revisiting a highly caffeinated plant from the Amazon. J Ethnopharmacol. 2013;150:14-31.
50. Heckman MA, Weil J, Gonzalez de Mejia E. Caffeine [1, 3, 7-trimethylxanthine] in foods: a comprehensive review on consumption, functionality, safety, and regulatory matters. J Food Sci. 2010;75:R77-R87.
51. Blankson KL, Thompson AM, Ahrendt DM, Patrick V. Energy drinks: what teenagers [and their doctors] should know. Pediatr Rev. 2013;34:55-62.
52. Kaur A, Yousuf H, Ramgobin-Marshall D, Jain R, Jain R. Energy drink consumption: a rising public health issue. Rev Cardiovasc Med. 2022;23:83.
53. Ragsdale FR, Gronli TD, Batool N, Haight N, Mehaffey A, McMahon EC, et al. Effect of Red Bull energy drink on cardiovascular and renal function. Amino Acids. 2010;38:1193-200.
54. Xu YJ, Arneja AS, Tappia PS, Dhalla NS. The potential health benefits of taurine in cardiovascular disease. Exp Clin Cardiol. 2008;13:57-65.
55. Lara DR. Caffeine, mental health, and psychiatric disorders. J Alzheimers Dis. 2010;Suppl 1:S239-48.
56. Winston AP, Hardwick E, Jaberi N. Neuropsychiatric effects of caffeine. Adv Psychiatr Treat. 2005;11:432-9.
57. Bradley JR, Petree A. Caffeine consumption, expectancies of caffeine-enhanced performance, and caffeinism symptoms among university students. J Drug Educ. 1990;20:319-28.
58. Klevebrant L, Frick A. Effects of caffeine on anxiety and panic attacks in patients with panic disorder: A systematic review and meta-analysis. Gen Hosp Psychiatry. 2022;74:22-31.
59. Lucas PB, Pickar D, Kelsoe J, Rapaport M, Pato C, Hommer D. Effects of the acute administration of caffeine in patients with schizophrenia. Biol Psychiatry. 1990;28:35-40.
60. Zaslove MO, Russell RL, Ross E. Effect of caffeine intake on psychotic in-patients. Br J Psychiatry. 1991;159:565-7.
61. Baethge C, Tondo L, Lepri B, Baldessarini RJ. Coffee and cigarette use: association with suicidal acts in 352 Sardinian bipolar disorder patients. Bipolar Disord. 2009;11:494-503.
62. Dratcu L, Grandison A, McKay G, Bamidele A, Vasudevan V. Clozapine-resistant psychosis, smoking, and caffeine: managing the neglected effects of substances that our patients consume every day. Am J Ther. 2007;14:314-8.
63. Kunitake Y, Mizoguchi Y, Sogawa R, Matsushima J, Kato TA, Kawashima T, et al. Effect of Excessive Coffee Consumption on the Clinical Course of a Patient With Bipolar Disorder: A Case Report and Literature Review. Clin Neuropharmacol. 2017;40:160-2.
64. Nehlig A. Interindividual Differences in Caffeine Metabolism and Factors Driving Caffeine Consumption. Pharmacol Rev. 2018;70:384-411.
65. Pardo Lozano R, Álvarez García Y, Barral Tafalla D, Farré Albaladejo M. Cafeína: un nutriente, un fármaco, o una droga de abuso. Adicciones. 2007;19:225-38.
66. Fekkes D, Pepplinkhuizen L, Verheij R, Bruinvels J. Abnormal plasma levels of serine, methionine, and taurine in transient acute polymorphic psychosis. Psychiatry Res. 1994;51:11-8.
67. Wong AH, Smith M, Boon HS. Herbal Remedies in Psychiatric Practice. Arch Gen Psychiatry. 1988;55:1033-44.
68. Atack JR. Inositol monophosphatase, the putative therapeutic target for lithium. Brain Res Rev. 1996;22:183-90.
69. Chu NS. Caffeine- and aminophylline-induced seizures. Epilepsia. 1981;22:85-94.
70. Cauli O, Morelli M. Caffeine and the dopaminergic system. Behav Pharmacol. 2005;16:63-77.
71. Asadi-Pooya AA, Zeraatpisheh Z, Rostaminejad M, Damabi NM. Caffeinated drinks, fruit juices, and epilepsy: A systematic review. Acta Neurol Scand. 2022;145:127-138.
72. Kaufman KR, Sachdeo RC. Caffeinated beverages and decreased seizure control. Seizure. 2003;12:519-21.
73. Mendez M, Radtke RA. Interactions between sleep and epilepsy. Journal of Clinical Neurophysiology. 2001;18:106-27.

74. Frucht MM, Quigg M, Schwaner C, Fountain NB. Distribution of seizure precipitants among epilepsy syndromes. Epilepsia. 2000;41:1534-9.
75. Tsuji T, Morita S, Saito T, Nakagawa Y, Inokuchi S. Serum potassium level as a biomarker for acute caffeine poisoning. Acute Med Surg. 2020;7:e568.
76. Wu JY, Prentice H. Role of taurine in the central nervous system. J Biomed Sci. 2010;17 Suppl 1:S1.
77. El Idrissi A, Messing J, Scalia J, Trenkner E. Prevention of epileptic seizures by taurine. Adv Exp Med Biol. 2003;526:515-25.
78. Eppler B, Patterson TA, Zhou W, Millard WJ, Dawson R Jr. Kainic acid [KA]-induced seizures in Sprague-Dawley rats and the effect of dietary taurine [TAU] supplementation or deficiency. Amino Acids. 1999;16:133-47.
79. Babu K, Church R, Lewander W. Energy drinks: The new eye opener for adolescents. Clin Pediatr Emerg. 2008;9:35-42.
80. Sanaei-Zadeh H. Overuse of energy drinks: why death? Am J Emerg Med. 2013;31:1713-4.
81. Worthley MI, Prabhu A, De Sciscio P, Schultz C, Sanders P, Willoughby SR. Detrimental effects of energy drink consumption on platelet and endothelial function. Am J Med. 2010;123:184-7.
82. Altschul R, Hoffer A, Stephen JD. Influence of nicotinic acid on serum cholesterol in man. Arch Biochem. 1955;54:558-9.
83. Al-Mohaissen MA, Pun SC, Frohlich JJ. Niacin: from mechanisms of action to therapeutic uses. Mini Rev Med Chem. 2010;10:204-17.
84. Capuzzi DM, Morgan JM, Brusco OA Jr, Intenzo CM. Niacin dosing: relationship to benefits and adverse effects. Curr Atheroscler Rep. 2000;2:64-71.
85. Backes JM, Padley RJ, Moriarty PM. Important considerations for treatment with dietary supplement versus prescription niacin products. Postgrad Med. 2011;123:70-83.
86. Coppola A, Brady PG, Nord HJ. Niacin-induced hepatotoxicity: unusual presentations. South Med J. 1994;87:30-2.
87. McKenney JM, Proctor JD, Harris S, Chinchili VM. A comparison of the efficacy and toxic effects of sustained- vs immediate-release niacin in hypercholesterolemic patients. JAMA. 1994;271:672-7.
88. Bhardwaj SS, Chalasani N. Lipid-lowering agents that cause drug-induced hepatotoxicity. Clin Liver Dis. 2007;11:597-613.
89. Pieper JA. Overview of niacin formulations: differences in pharmacokinetics, efficacy, and safety. Am J Health Syst Pharm. 2003;60:S9-S14.
90. Alhadeff L, Gualtieri CT, Lipton M. Toxic effects of water-soluble vitamins. Nutr Rev. 1984;42:33-40.
91. Ayuob N, ElBeshbeishy R. Impact of an energy drink on the structure of stomach and pancreas of albino rat: can omega-3 provide a protection? PLoS One. 2016;11:e0149191
92. Qassim AH, Alsammak MA, Ayoob AA. Histopathological changes in kidney and pancreas induced by energy drinks in adult male rats. Iraqi J Vet Sci. 2021;36:111-6.
93. Guyton JR, Bays HE. Safety considerations with niacin therapy. Am J Cardiol. 2007;99:22C-31C.
94. Muller N, Sarantitis I, Rouanet M, de Mestier L, Halloran C, Greenhalf W, et al. Natural history of SPINK1 germline mutation related-pancreatitis. EBioMedicine. 2019;48:581-91.
95. Passmore AP, Kondowe GB, Johnston GD. Caffeine and hypokalemia. Ann Intern Med. 1986;105:468.
96. Aizaki T, Osaka M, Hara H, Kurokawa S, Matsuyama K, Aoyama N, et al. Hypokalemia with syncope caused by habitual drinking of oolong tea. Intern Med. 1999;38:252-6.
97. Mudge DW, Johnson DW. Coca-Cola and kangaroos. Lancet. 2004;364:1190.
98. Shirley DG, Walter SJ, Noormohamed FH. Natriuretic effect of caffeine: assessment of segmental sodium reabsorption in humans. Clin Sci [Lond]. 2002;103:461-6.
99. Benowitz NL. Clinical pharmacology of caffeine. Annu Rev Med. 1990;41:277-88.
100. Schaffer SW, Shimada K, Jong CJ, Ito T, Azuma J, Takahashi K. Effect of taurine and potential interactions with caffeine on cardiovascular function. Amino Acids. 2014;46:1147-57.
101. Sung DJ, Choi EJ, Kim S, Kim J. Rhabdomyolysis from Resistance Exercise and Caffeine Intake. Iran J Public Health. 2018;47:138-9.
102. Campana C, Griffin PL, Simon EL. Caffeine overdose resulting in severe rhabdomyolysis and acute renal failure. Am J Emerg Med. 2014;32:111.e3-111.e4.
103. Chesney RW, Han X, Patters AB. Taurine and the renal system. J Biomed Sci. 2010;17:S4.
104. Suliman ME, Bárány P, Filho JC, Lindholm B, Bergström J. Accumulation of taurine in patients with renal failure. Nephrol Dial Transplant. 2002;17:528-9.
105. Ang-Lee MK, Moss J, Yuan CS. Herbal medicines and perioperative care. JAMA. 2001;286:208-16.
106. Bydlowski SP, Yunker RL, Subbiah MT. A novel property of an aqueous guaraná extract [Paullinia cupana]: inhibition of platelet aggregation in vitro and in vivo. Braz J Med Biol Res. 1988;21:535-8.
107. Sankararaman S, Syed W, Medici V, Sferra TJ. Impact of Energy Drinks on Health and Well-being. Curr Nutr Rep. 2018;7:121-30.
108. Khayyat LI, Essawy AE, Al Rawy MM, Sorour JM. Comparative study on the effect of energy drinks on haematopoietic system in Wistar albino rats. J Environ Biol. 2014;35:883-91.
109. Mirza F, Saleem Y, Qurat-ul-ain, Ahmed S. Effect of Energy Drink Consumption on Blood Glucose Level and Clotting Time; A Comparative Study on Healthy Male and Female Subjects. Int J Endorsing Health Sci Res. 2019;7:166-71.
110. Lotfi K, Grunwald JE. The effect of caffeine on human macularcirculation. Invest Ophthalmol Vis Sci. 1991;32:3028-32.
111. Karti O, Zengin MO, Kerci SG, Ayhan Z, Kusbeci T. Acute effect of caffeine on macular microcirculation in healthy subjects: an optical coherence tomography angiography study. Retina. 2019;39:964-71.
112. Makri OE, Georgalas I, Georgakopoulos CD. Drug-induced macular edema. Drugs. 2013;73:789-802.
113. Ali F, Rehman H, Babayan Z, Stapleton D, Joshi DD. Energy drinks and their adverse health effects: A systematic review of the current evidence. Postgrad Med. 2015;127:308-22.
114. Costantino A, Maiese A, Lazzari J, Casula C, Turillazzi E, Frati P, et al. The Dark Side of Energy Drinks: A Comprehensive Review of Their Impact on the Human Body. Nutrients. 2023;15:3922.
115. Machado-Vieira R, Viale CI, Kapczinski F. Mania associated with an energy drink: The possible role of caffeine, taurine, and inositol. Can J Psychiatry. 2001;46:454-555.
116. Cannon ME, Cooke CT, McCarthy JS. Caffeine-induced cardiac arrhythmia: an unrecognized danger of healthfood products. Med J Aust. 2001;174:520-1.
117. Berigan T. An anxiety disorder secondary to energy drinks: a case report. Psychiatry [Edgmont]. 2005;2:10.
118. Iyadurai SJ, Chung SS. New-onset seizures in adults: Possible association with consumption of popular energy drinks. Epilepsy Behav. 2007;10:504-8.
119. Chelben J, Piccone-Sapir A, Ianco I, Shoenfeld N, Kotler M, Strous D. Effects of amino acid energy drinks leading to hospitalization in individuals with mental illness. Gen Hosp Psychiatry. 2008;30:187-9.
120. Nagajothi N, Khraisat A, Velazquez-Cecena JLE, Arora R. Energy drink-related supraventricular tachycardia. Am J Med. 2008;121:e3-e4.
121. Terlizzi R, Rocchi C, Serra M, Solieri L, Cortelli P. Reversible postural tachycardia syndrome due to inadvertent overuse of Red Bull®. Clin Auton Res. 2008;18:221-3.
122. Berger AJ, Alford K. Cardiac arrest in a young man following excess consumption of caffeinated "energy drinks". Med J Aust. 2009;190:41-3.
123. Cerimele JM, Stern AP, Jutras-Aswad D. Psychosis following excessive ingestion of energy drinks in a patient with schizophrenia. Am J Psychiatry. 2010;167:353.
124. Ernest D, Chia M, Corallo CE. Profound hypokalaemia due to Nurofen Plus and Red Bull misuse. Crit Care Resusc. 2010;12:109-10.
125. Menkes DB. Transient psychotic relapse temporally related to ingestion of an "energy drink". Med J Aust. 2011;194:206.
126. Di Rocco JR, During A, Morelli PJ, Heyden M, Biancaniello TA. Atrial fibrillation in healthy adolescents after highly caffeinated beverage consumption: two case reports. J Med Case Rep. 2011;5:18.
127. Scott MJ, El-Hassan M, Khan AA. Myocardial infarction in a young adult following the consumption of a caffeinated energy drink. BMJ Case Reports. 2011;2011:bcr0220113854.
128. Rizkallah É, Bélanger M, Stavro K, Dussault M, Pampoulova T, Chiasson JP, et al. Could the use of energy drinks induce manic or depressive relapse among abstinent substance use disorder patients with comorbid bipolar spectrum disorder? Bipolar Disord. 2011;13:578-80.
129. Vivekanandarajah A, Ni S, Waked A. Acute hepatitis in a woman following excessive ingestion of an energy drink: a case report. J Med Case Rep. 2011;5:227.
130. Apestegui CA, Julliard O, Ciccarelli O, Ho Minh Duc DK, Lerut J. Energy drinks: Another "red flag" for the liver allograft. Liver Transpl. 2011;17:1117-8.
131. Babu KM, Zuckerman MD, Cherkes JK, Hack JB. First-Onset Seizure After Use of 5-hour ENERGY. Pediatr Emerg Care. 2011;27:539-40.
132. Trabulo D, Marques S, Pedroso E. Caffeinated energy drink intoxication. BMJ Case Rep. 2011;2011:bcr0920103322.
133. Schöffl I, Kothmann JF, Schöffl V, Rupprecht HD, Rupprecht T. "vodka energy": Too much for the adolescent nephron? Pediatrics. 2011;128:e227-e231.
134. Mugmon M. Atrial flutter with aberrant conduction in a patient taking amphetamine salts and caffeine. J Community Hosp Intern Med Perspect. 2012;1:10663.
135. Argano C, Colomba D, Di Chiara T, La Rocca E. Take the wind out your sails: relationship among energy drink abuse, hypertension, and break-up of cerebral aneurysm. Intern Emerg Med. 2012;7:S9-S10.
136. Benjo AM, Pineda AM, Nascimento FO, Zamora C, Lamas GA, Escolar E. Left main coronary artery acute thrombosis related to energy drink intake. Circulation. 2012;125:1447-8.
137. Hanan Israelit S, Strizevsky A, Raviv B. ST elevation myocardial infarction in a young patient after ingestion of caffeinated energy drink and ecstasy. World J Emerg Med. 2012;3:305-7.
138. Wilson RE, Kado HS, Samson R, Miller AB. A case of caffeine-induced coronary artery

vasospasm of a 17-year-old male. Cardiovasc Toxicol. 2012;12:175-9.
139. Izquierdo Fos I, Vázquez Gomis RM, Vázquez Gomis C, Piernas R, Climent Forner E, Llaguno Salvador MD, et al. Episodio de fibrilación auricular tras ingesta de bebida energética y práctica de deporte. An Pediatr [Barc]. 2012;77:417-9.
140. Rottlaender D, Motloch LJ, Reda S, Larbig R, Hoppe UC. Cardiac arrest due to long QT syndrome associated with excessive consumption of energy drinks. Int J Cardiol. 2012;158:e51-e52.
141. Dufendach KA, Horner JM, Cannon BC, Ackerman MJ. Congenital type 1 long QT syndrome unmasked by a highly caffeinated energy drink. Heart Rhythm. 2012;9:285-8.
142. Rutledge M, Witthed A, Khouzam RN. It took a RedBull to unmask Brugada syndrome. Int J Cardiol. 2012;161:e14-e15.
143. Calabrò RS, Italyno D, Gervasi G, Bramanti P. Single tonic-clonic seizure after energy drink abuse. Epilepsy Behav. 2012;23:384-5.
144. Szpak A, Allen D. A case of acute suicidality following excessive caffeine intake. J Psychopharmacol. 2012;26:1502-10.
145. Foran M, Strickland F, Perkins K, Smith JA. Excessive intraoperative bleeding with chronic energy drink consumption. J Oral Maxillofac Surg. 2012;70:1439-41.
146. Usman A, Jawaid A. Hypertension in a young boy: an energy drink effect. BMC Res Notes. 2012;5:591.
147. Kaoukis A, Panagopoulou V, Mojibian HR, Jacoby D. Reverse Takotsubo cardiomyopathy associated with the consumption of an energy drink. Circulation. 2012;125:1584-5.
148. Avcı S, Sarıkaya R, Büyükcam F. Death of a young man after overuse of energy drink. Am J Emerg Med. 2013;31:1624.e3-1624.e4.
149. Iglesias-Lepine ML, Epelde F, Espinosa J, Mariñosa M. Consumo de bebidas energizantes con alcohol, una mezcla arriesgada. Med Clin [Barc]. 2013;141:135-6.
150. Dikici S, Saritas A, Besir FH, Tasci AH, Kandis H. Do energy drinks cause epileptic seizure and ischemic stroke? Am J Emerg Med. 2013;31:274.e1-274.e4.
151. Jonjev ZS, Bala G. High-energy drinks may provoke aortic dissection. Coll Antropol. 2013;37 Suppl 2:227-9.
152. Polat N, Ardıç I, Akkoyun M, Vuruskan E. Spontaneous coronary artery dissection in a healthy adolescent following consumption of caffeinated "energy drinks". Turk Kardiyol Dern Ars. 2013;41:738-42.
153. Takahashi K, Tsukamoto S, Kakizaki Y, Saito K, Ohkohchi N, Hirayama K. Hypercobalaminemia induced by an energy drink after total gastrectomy: a case report. J Rural Med. 2013;8:181-5.
154. Görgülü Y, Tasdelen Ö, Sönmez MB, Köse Çinar R. A Case of Acute Psychosis Following Energy Drink Consumption. Noro Psikiyatr Ars. 2014;51:79-81.
155. Ward AE, Lipshultz SE, Fisher SD. Energy drink–induced near-fatal ventricular arrhythmia prevented by an intracardiac defibrillator decades after operative "repair" of tetralogy of fallot. Am J Cardiol. 2014;114:1124-5.
156. Asencio-Sánchez VM. Bebidas energéticas y salud visual. Arch Soc Esp Oftalmol. 2014;89:466-7.
157. Katsue H, Higashi Y, Baba N, Aoki M, Sakanoue M, Matsushita S, et al. Allergic reaction caused by acesulfame potassium in foods. Contact Dermatitis. 2014;71:251-2.
158. Cruzado L, Sánchez-Fernández M, Cortez-Vergara C, Rojas-Rojas G. Manía inducida por bebidas energéticas con alto contenido de cafeína. Actas Esp Psiquiatr. 2014;42:259-66.
159. Ünal S, Sensoy B, Yilmaz S, Ünal GG, Süleymanoglu M, Sen F, et al. Left main coronary artery thrombosis and acute anterior myocardial infarction related to energy drink. Int J Cardiol. 2015;179:66-7.
160. Krankl JT, Gitlin M. Caffeine-induced mania in a patient with caffeine use disorder: A case report. Am J Addict. 2015;24:289-91.
161. Huang B, Kunkel D, Kabany ME. Acute Liver Failure Following One Year of Daily Consumption of a Sugar-Free Energy Drink. ACG Case Rep J. 2014;1:214-6.
162. Greene E, Oman K, Lefler M. Energy drink-induced acute kidney injury. Ann Pharmacother. 2014;48:1366-70.
163. Shmelev A, Abdo A, Sachdev S, Shah U, Kowdley GC, Cunningham SC. Energetic etiologies of acute pancreatitis: A report of five cases. World J Gastrointest Pathophysiol. 2015;6:243-8.
164. Salanova-Villanueva L, Bernis-Carro C, Alberto-Blazquez L, Sanchez-Tomero JA. Arritmia cardiaca grave por hipopotasemia. Influencia de las sustancias diuréticas. Nefrologia. 2015;35:334-46.
165. Dikici S, Saritas A, Kilinc S, Guneysu S, Gunes H. Does an energy drink cause a transient ischemic attack? Am J Emerg Med. 2015;33:129.e5-129.e6.
166. Samanta D. Reversible cerebral vasoconstriction syndrome: another health hazard of energy drinks? Clin Pediatr [Phila]. 2015;54:186-7.
167. Mounir K, Mattous M, Atmani M. An unexplained hypokalemia: Consider energy drinks. Anaesth Crit Care Pain Med. 2015;34:303.
168. Wang HR, Woo YS, Bahk WM. Caffeine-induced psychiatric manifestations: a review. Int Clin Psychopharmacol. 2015;30:179-82.
169. Solomin D, Borron SW, Watts SH. STEMI Associated with Overuse of Energy Drinks. Case Rep Emerg Med. 2015;2015:1-3.
170. Iyer PS, Yelisetti R, Miriyala V, Siddiqui W, Kaji A. A remarkable case of rhabdomyolysis associated with ingestion of energy drink 'neon volt'. J Community Hosp Intern Med Perspect. 2016;6:32528.
171. Sattari M, Sattari A, Kazory A. Energy Drink Consumption and Cardiac Complications: A Case for Caution. J Addict Med. 2016;10:280-2.
172. Begolli Gerqari AM, Ferizi M, Halimi S, Daka A, Hapciu S, Begolli IM, et al. Erythema exsudativum multiforme induced by a taurine-containing energy drink. Acta Dermatovenerol Alp Pannonica Adriat. 2016;25:83-4.
173. Grant RA, Cord BJ, Kuzomunhu L, Sheth K, Gilmore E, Matouk CC. Aneurysmal subarachnoid hemorrhage and severe, catheter-induced vasospasm associated with excessive consumption of a caffeinated energy drink. Interv Neuroradiol. 2016;22:674-8.
174. Harb JN, Taylor ZA, Khullar V, Sattari M. Rare cause of acute hepatitis: a common energy drink. BMJ Case Rep. 2016;2016:bcr2016216612.
175. Hernandez-Huerta D, Martin-Larregola M, Gomez-Arnau J, Correas-Lauffer J, Dolengevich-Segal H. Psychopathology Related to Energy Drinks: A Psychosis Case Report. Case Rep Psychiatry. 2017;2017:1-5.
176. Pagano CW, Wu M, Wu L. Acute visual loss and intraretinal hemorrhages associated to energy drink consumption. Int Ophthalmol. 2017;37:1349-51.
177. Icin T, Medic-Stojanoska M, Ilic T, Kuzmanovic V, Vukovic B, Percic I, et al. Multiple Causes of Hyponatremia: A Case Report. Med Princ Pract. 2017;26:292-5.
178. Enriquez A, Frankel DS. Arrhythmogenic effects of energy drinks. J Cardiovasc Electrophysiol. 2017;28:711-7.
179. Gharacholou SM, Ijioma N, Banwart E, Munoz FD. ST-Segment Elevation Myocardial Infarction and Normal Coronary Arteries after Consuming Energy Drinks. Case Rep Cardiol. 2017;2017:1-5.
180. Zekavat OR, Fathpour G, Haghpanah S, Dehghani SJ, Zekavat M, Shakibazad N. Acquired Vitamin K Deficiency as Unusual Cause of Bleeding Tendency in Adults: A Case Report of a Nonhospitalized Student Presenting with Severe Menorrhagia. Case Rep Obstet Gynecol. 2017;2017:4239148.
181. Mattioli AV, Pennella S, Farinetti A, Manenti A. Energy Drinks and atrial fibrillation in young adults. Clin Nutr. 2018;37:1073-4.
182. Casas-Gómez C, Muñoz-Molero MJ, Guerrero-Sánchez R, Martínez-León F. Manía y bebidas energéticas. Actas Esp Psiquiatr. 2018;46:156-8.
183. Zacher J, May E, Horlitz M, Pingel S. Binge drinking alcohol with caffeinated "energy drinks", prolonged emesis and spontaneous coronary artery dissection: a case report, review of the literature and postulation of a pathomechanism. Clin Res Cardiol. 2018;107:975-9.
184. Quadri S, Harding L, Lillig M. An Energy Drink-Induced Manic Episode in an Adolescent. Prim Care Companion CNS Disord. 2018;20:18l02318.
185. Versha F, Young R, Ajumobi A. A Rare Cause of Acute Pancreatitis. Am J Gastroenterol. 2018;113:S762.
186. Al-Tamimi A, Kassim T, Chandra S. Recurrent acute pancreatitis with energy drinks consumption: Rare but definite association. Am J Gastroenterol. 2018;113:S828.
187. Wajih Ullah M, Lakhani S, Siddiq W, Handa A, Kahlon Y, Siddiqui T. Energy Drinks and Myocardial Infarction. Cureus. 2018;10:e2658.
188. Butragueño Laiseca L, Toledo del Castillo B, Miranda Herrero MC. Bebidas enérgicas como desencadenante de crisis convulsivas en pacientes pediátricos: a propósito de un caso. Neurologia. 2019;34:343-5.
189. Gupta N, Padidam S, Tewari A. Acute macular neuroretinopathy [AMN] related to energy drink consumption. BMJ Case Rep. 2019;12:e232144.
190. Belzile D, Cinq-Mars A, Bernier M, Leblanc MH, Bourgault C, Morin J, et al. Do Energy Drinks Really Give You Wings? Left Ventricular Assist Device Therapy as a Bridge to Recovery for an Energy Drink-Induced Cardiomyopathy. Can J Cardiol. 2020;36:317.e1-317.e3.
191. Uwaifo GI. Beware Energy Drinks: A Case of a Toxic Triad Syndrome in a Diabetic Patient with Nonalcoholic Fatty Liver Disease. Am J Med Sci. 2019;358:304-11.
192. Kelsey D, Berry AJ, Swain RA, Lorenz S. A case of psychosis and renal failure associated with excessive energy drink consumption. Case Rep Psychiatry. 2019;2019:1-5.
193. Osman H, Tabatabai S, Korashy M, Hussein M. Caffeinated Energy Drink Induced Ventricular Fibrillation: The Price for Overexcitement. Cureus. 2019;11:e6358.
194 Ibrahim AM, Ali M, Al-Bast B, Bhandari B, Kulkarni A. Energy drink consumption resulting in severe hypertriglyceridemia, hyperglycemia, and acute pancreatitis. J Am Coll Cardiol. 2019;73:2752.
195 Göçer M, Iltar U, Aykas F, Aslan V, Nizam I, Çelik M, et al. Thrombotic Thrombocytopenic Purpura Cases and Consumption of Large Quantities of Energy Drink: Related or Coincidence? Acta Haematol. 2020;143:176-180.
196. Al Yacoub R, Luczkiewicz D, Kerr C. Acute kidney injury and hepatitis associated with energy drink consumption: a case report. J Med Case Rep. 2020;14:23.
197. Hanif M, Saleem S, Naz S, Sundas F. Ener-

gy Drinks and Atrial Fibrillation: An Unusual Case of Caution. Cureus. 2020;12:e10807.
198. Baez-Ferrer N, Parra-Esquivel PC, Galvez-Rodríguez M, Burillo-Putze G, Abreu-Gonzalez P, Domínguez-Rodríguez A. Miocarditis relacionada con el uso de bebidas energéticas: a propósito de un caso. Emergencias. 2020;32:217-8.
199. Garg A, Rodriguez A, Lewis JT, Bansal R, Brahmbhatt B. Energy Drinks: A Reversible Risk Factor for Atrophic Gastritis and Gastric Intestinal Metaplasia. Cureus. 2020;12:e12298.
200. Fisk G, Hammond-Haley M, D'Silva A. Energy drink-induced cardiomyopathy. BMJ Case Rep. 2021;14:e239370.
201. Uyanik M, Gedikli O, Yildirim U. Energy Drink-Associated Cardiomyopathy after Excessive Consumption: A Case Report. J Tehran Heart Cent. 2021;16:119-22.
202. Staikoglou N, Polanagnostaki A, Lamprou V, Chartampilas E, Pavlou E, Tegos T, et al. Posterior cerebral artery dissection after excessive caffeine consumption in a teenager. Radiol Case Rep. 2022;17:2081-4.
203. Tinawi M. Severe Rhabdomyolysis Due to Strenuous Exercise with a Potential Role of a High-Caffeine Energy Drink. Cureus. 2022;14:e20867.
204. Randhawa N, Shah M, Spyratos T. The Pain of Staying Alert: A Case Report and Literature Review on Energy Drink-Induced Acute Pancreatitis. J Investig Med High Impact Case Rep. 2022;10:23247096221104468.
205. Pallangyo P, Bhalia SV, Komba M, Mkojera ZS, Swai HJ, Mayala HA, et al. Acute Myocardial Infarction Following the Consumption of Energy Drink in a 28-Year-Old Male: A Case Report. J Investig Med High Impact Case Rep. 2023;11:23247096231168811.
206. Yoshimine N, Oba N, Hasegawa C, Inoue N, Nagumo H, Arashiyama M, et al. Caffeine Intoxication, in Which the Theophylline Level Served as an Indicator of the Treatment Course and the Caffeine Level Could Be Measured. Intern Med. 2024;63:2157-61.
207. Othman MK, Yusof Z, Mohd Rossli NI, Mohd Fauzi MH, W Isa WYH. Adverse Cardiovascular Events Associated With Kopi Jantan. Cureus. 2024;16:e52344.
208. Báez-Ferrer N, Parra-Esquivel PC, Rodríguez-Cabrera CM, Burillo-Putze G, Avanzas P, Domínguez-Rodríguez A. Dilated cardiomyopathy associated with cardiotoxicity due to consumption of energy drinks. Rev Esp Cardiol (Engl Ed). 2024;77:699-700.
209. Sinha V, Lam L, Nguyen MV. High Doses of Caffeine-Induced Cerebral Infarction Leading to Partial Locked-In Syndrome in a Young Adult: A Novel Association? Case Rep Neurol. 2024;16:115-21.
210. McMillan C, Dower J, Ikeda I, Marhoffer E. Chronic Pancreatitis Pain Associated with Energy Drink Consumption: A Case Report. J Investig Med High Impact Case Rep. 2024;12:23247096241255806.
211. Mannix D, Mulholland K, Byrne F. Caffeine-Induced Psychosis: A Case Report and Review of Literature. Cureus. 2024;16:e66306.
212. Van Houtte L, Bulinckx A, Roquet-Gravy PP. Pink Eccrine Chromhidrosis Induced by Excessive Consumption of an Energy Drink. Cureus. 2024;16:e71854.

Annex 1. Articles included in the review

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Machado-Viera R, *et al.*[115]	2001/Spain	PSY	36/M	Euphoria, hyperactivity, insomnia, irritability, increased libido	Manic episode	3 cans/day (6 total)	Type I bipolar disorder	None	Not specified
Cannon ME, *et al.*[116]	2001/ Australia	CAR	25/F	Syncope	Ventricular fibrillation. Death	1 can (55 ml)	Mitral valve prolapse; palpitations	None	Occasional
Berigan T[117]	2005/USA	PSY	25/M	Anxiety, restlessness, irritability, difficulty concentrating, sleep-onset insomnia	Anxiety disorder	6–8 cans/day of 230 ml	No history	None	Habitual
Iyadurai SJP, *et al.*[118]	2006/USA	NEU	25/M	No symptoms reported. PE:SBP 150 mmHg, HR 120 bpm; remainder unremarkable. Lab tests:Leukocytosis. Imaging: No abnormalities	Tonic-clonic seizure	3–4 cans/ week of 474 ml; 2 cans of 700 ml 1 hour before	No history	None	Habitual
Iyadurai SJP, *et al.*[118]	2006/USA	NEU	19/M	No symptoms reported. PE:SBP 189 mmHg, HR 133 bpm. Lab tests: No abnormalities. Imaging: No abnormalities	Tonic-clonic seizure	Several 700 ml cans 2 hours before	Complex migraine	None	Occasional
Iyadurai SJP, *et al.*[118]	2006/USA	NEU	28/F	No symptoms reported. PE:SBP 145 mmHg, HR 99 bpm; remainder unremarkable. Lab tests: No abnormalities. Imaging: No abnormalities	Tonic-clonic seizure	700 ml	Migraine	Diet pills	Habitual
Iyadurai SJP, *et al.*[118]	2006/USA	NEU	26/M	No symptoms reported. Vital signs/PE: No abnormalities. Lab tests: No abnormalities. Imaging: No abnormalities	Tonic-clonic seizure	2 cans of 700 ml	No history	None	Not specified
Chelben J, *et al.*[119]	2007/Israel	PSY	41/F	Psychomotor agitation, hypervigilance, aggressiveness, impulsive behavior	Psychotic episode	5 cans/day	Cluster B personality disorder; suicide attempts	None	1 week
Chelben J, *et al.*[119]	2007/Israel	PSY	38/F	Psychomotor agitation, increased alertness, sleep-onset insomnia, impulsivity, self-harm	Psychotic episode	5–10 cans/day	Borderline personality disorder and bipolar disorder	Cannabis	1 month
Chelben J, *et al.*[119]	2007/Israel	PSY	25/M	Psychomotor agitation, hypervigilance, verbal aggression, thoughts of death	Psychotic episode	8–9 cans/day	Schizophrenia	None	1 month
Nagajothi N, *et al.*[120]	2008/USA	CAR	23/F	Palpitations, chest pain. PE:HR 219 bpm; remainder unremarkable. Lab tests: No abnormalities	Supraventricular tachycardia	Shot	No history	None	Not specified
Terlizzi R, *et al.*[121]	2008/Italy	CAR	16/M	Syncope, orthostatic intolerance in the prior 3 months. PE:No abnormalities. Imaging: No abnormalities	Postural tachycardia syndrome	4–5 cans daily	Non-professional athlete	None	3 months
Berger AJ, *et al.*[122]	2009/ Australia	CAR	28/M	Syncope with prior chest pain. PE: No abnormalities. Lab tests: TnI 0.24, peak 12.2 mmol/L; potassium 3.0 mmol/L. Imaging: No abnormalities. Invasive tests:No abnormalities	Ventricular fibrillation. Cardiac arrest. ST-elevation myocardial infarction	7–8 cans in the previous 7 hours	Motorcycling	Smoker, pack-year index 6	Not specified
Cerimele JM, *et al.*[123]	2009/USA	PSY	43/M	Paranoia, religious delusions, agitation	Psychotic episode	8–10 cans/day of 470 ml	Schizophrenia Former alcohol use; former drug use	None	8 weeks
Ernest D, *et al.*[124]	2010/ Australia	NEF	39/M	Weakness in all four limbs. PE: No abnormalities. Lab tests: Potassium 1.8 mmol/L, phosphorus 0.63 mmol/L, CK-NAC peak 46,298 IU/L	Severe hypokalemia	24 cans in 3 days of 250 ml	Tourette syndrome, OCD, anxiety, depression; alcohol abuse	Herbal preparation; ibuprofen	Cocaine

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Menkes DB[125]	2011/Nueva Zelanda	PSY	25/M	Irritability, paranoia, tachycardia, insomnia, emotional lability	Psychotic episode	3 shots	Obesity, schizophrenia; alcohol; cannabis	10 cups instant coffee	No
Di Rocco JR, *et al.*[126]	2011/USA	CAR	14/M	Palpitations. PE: Irregular pulse. Lab tests: No abnormalities. Imaging: No abnormalities	Atrial fibrillation	Not specified	No history	None	Cocaine
Di Rocco JR, *et al.*[126]	2011/USA	CAR	16/M	Alcohol intoxication, vomiting, head trauma. HR 160 bpm irregular. Lab tests: Blood ethanol 155 mg/dL. Imaging: No abnormalities	Atrial fibrillation	Not specified	ADHD, allergic asthma	Alcohol	No
Scott MJ, *et al.*[127]	2011/United Kingdom	CAR	19/M	Chest pain, dyspnea. PE:RR 16 rpm. Lab tests:TnI 34.67 ng/mL; D-dimer 375 ng/mL. Imaging: No abnormalities	STEMI	2–3 cans/day	Gastroesophageal reflux	None	Cocaine
Rizkallah É, *et al.*[128]	2011/ Canada	PSY	40/M	Elevated mood, irritability, grandiosity	Manic episode	6 cans/day	Bipolar disorder type I/former cocaine use	None	No
Rizkallah É, *et al.*[128]	2011/ Canada	PSY	30/F	Irritability, flight of ideas, decreased need for sleep, increased sexual desire	Manic episode	8 cans/day	Bipolar disorder type II/ former cocaine use	Cocaine	Cocaine
Rizkallah É, *et al.*[128]	2011/ Canada	PSY	36/M	Sleep disturbance, increased daytime somnolence, irritability, anxiety, depression	Depressive episode	9 cans/day	Bipolar disorder type I/marijuana, cocaine	None	No
Vivekanandarajah A, *et al.*[129]	2011/USA	DIG	22/F	Epigastric pain, vomiting, fever. PE:Mild epigastric tenderness. Lab tests:AST 7,709 U/L; ALT 7,533 U/L; total bilirubin 3.5 mg/dL (direct 1.9); albumin 3.8 g/ dL; GGT 29 U/L. Serology negative. Imaging: No abnormalities	Acute hepatitis	10 cans/day	No history	None	2 weeks
Apestegui CA, *et al.*[130]	2011/ Belgium	DIG	16/M	Jaundice. Lab tests:Bilirubin 10.7× ULN, AST 36× ULN, ALT 26× ULN, GGT 3× ULN. Biopsy:Perivenular hepatocellular necrosis, centrilobular and portal iand mild signs of cholangistis and endotheliitis	Toxic acute hepatitis (2 episodes)	15 cans/3 days (2 episodes; 3 cans/4 hours)	Liver transplant	Ibuprofen	Occasional
Babu KM, *et al.*[131]	2011/USA	NEU	15/M	Posticta state, vomiting. PE: Temp 38.1°C, BP 99/57 mmHg, HR 120 bpm, RR 40 rpm. Lab tests:Potassium 3.3 mEq/L. Imaging: No abnormalities	Tonic-clonic seizure	2 cans	No history	Coffee	Not specified
Trabulo D, *et al.*[132]	2011/ Portugal	NEU	28/M	Stupor (Glasgow 8). PE: BP 160/70 mmHg, HR 170 bpm, RR 30 rpm, temp 38.4°C. Lab tests: $PaCO_2$ 90 mmHg; PaO_2 40 mmHg; pH 6.8; lactate >15 mmol/L; leukocytes 23,200; glucose 220 mg/dL; creatinine 1.5 mg/dL; phosphorus 7.9 mg/dL; Mg 2.5 mg/dL. Imaging:Cardiomegaly; chronic vascular encephalopathy with corticosubcortical atrophy; TTE: severe mitral regurgitation, LV and atrial dilation, mild pulmonary hypertension	Tonic-clonic seizure. Ventricular tachycardia	6 cans/4 hours prior	Hepatitis C, postinfectious endocarditis, mitral insufficiency, prior stroke; Former user of heroin and cocaine	Coffee	Not specified

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Schöffl I, *et al.*[133]	2011/ Germany	NEF	17/M	Vomiting, dizziness. Lab tests: Creatinine 3.5 mg/dL initially, peak 6.9 mg/dL. Imaging:No abnormalities	Acute renal failure (acute tubular necrosis)	3 liters	No history; athlete	Alcohol (1 liter)	Not specified
Mugmon M[134]	2012/USA	CAR	26/M	Palpitations and dizziness during exercise. Lab tests: TnI peak 2.14 ng/mL. Imaging: No abnormalities	Atrial flutter with aberrant conduction	1 can/day	ADHD on amphetamine treatment	None	Daily
Argano C, *et al.*[135]	2012/Italy	NEU	38/M	Headache, vomiting, photophobia; hypertension. Lab tests:Increased norepinephrine and epinephrine. Imaging: MRI: ruptured aneurysm of anterior communicating artery	Ruptured aneurysm	5 cans/day	Not specified	None	3 weeks prior
Benjo AM, *et al.*[136]	2012/USA	CAR	24/M	Nausea, vomiting, palpitations, chest pain. PE:BP 138/94 mmHg, RR 18 rpm. Lab tests:TnI peak 38 ng/ mL. Imaging:TTE: apical hypokinesia; angiogram: 90 % occlusion of circumflex and left coronary artery	STEMI	Not specified	No history	Alcohol, cannabis; smoker	Not specified
Hanan Israelit S, *et al.*[137]	2012/Israel	CAR	24/M	Chest pain, nausea, vomiting. PE: BP 90/60 mmHg, HR 110 bpm, crackles. Death	STEMI. Ventricular fibrillation. Dead	20 cans	Obesity, mild untreated hypertension	MDMA.	Occasional
Wilson E, *et al.*[138]	2012/USA	CAR	17/M	Chest pain radiating to left arm. PE: BP 98/56 mmHg, HR 104 bpm, RR 22 rpm. Lab tests: Leukocytes 13,400/µL; potassium 2.6 mEq/L; HCO_2 18 mEq/L; CK peak 4,303 U/L; TnI peak 7.07. Imaging: TTE: EF 50 % with apical hypokinesia	STEMI	5-7 cans	Suspected prior pericarditis	None	Occasional
Izquierdo Fos I, *et al.*[139]	2012/Spain	CAR	13/M	Two syncopal episodes, palpitations, euphoria. PE: BP 132/68 mmHg, HR 100–180 bpm irregular, RR 45 rpm. Lab tests: No abnormalities. Imaging: No abnormalities	Atrial fibrillation	1 can (250 ml)	Soccer player; similar prior episodes after energy drink intake	None	Habitual
Rottlaender D, *et al.*[140]	2012/ Germany	CAR	22/F	No prior prodrome. Lab tests: Elevated TnI, CK, AST. Imaging: No abnormalities	Cardiac arrest: Torsades de pointes, ventricular fibrillation. Long QT syndrome	6 cans/4 hours	No history	None	Not specified
Dufendach KA, *et al.*[141]	2012/USA	CAR	13/F	Palpitations, chest pain, tremors, dizziness. ECG: QT/ QTc 464/622 ms	Long QT syndrome	1 can (470 mL)	Two prior syncopal episodes without energy drinks; citalopram treatment	None	2 weeks
Rutledge M, *et al.*[142]	2012/USA	CAR	24/M	Syncope. PE:No abnormalities. Lab tests:Potassium 2.7 mmol/L; HCO_2 17 mmol/L; Mg 1.7 mg/dL; PO_2 2 mg/ dL. Imaging: Chest X-ray: pulmonary edema	Cardiac arrest: ventricular fibrillation. Brugada syndrome	1 can	Prior palpitations; diarrhea	Alcohol	Not specified
Calabrò RS, *et al.*[143]	2012/Italy	NEU	20/M	Mild confusion. Lab tests: No abnormalities. Imaging: No abnormalities	Tonic-clonic seizure	4-6 cans/day	No history	None	5 months
Szpak A, *et al.*[144]	2012/United Kingdom	PSY	28/M	Aggressiveness, amnesia. Imaging: No abnormalities	Suicide attempt Amnesia	14 cans/48 hours	Family history of mental illness	None	Habitual

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Foran M, *et al.*[145]	2012/USA	HEM	31/M	Estimated bleeding: 5100 ml. Lab tests:Hematocrit drop from 40 to 30 g/dL; INR 2.1; prothrombin activity 24.9	Intraoperative bleeding	1–2 cans/day (470 ml)	Mandibular malformation	None	Habitual
Foran M, *et al.*[145]	2012/USA	HEM	18/M	Estimated bleeding: 2000 ml. Lab tests:Hematocrit drop from 39 to 24 g/dL	Intraoperative bleeding	2–3 cans/day (470 ml)	Mandibular malformation	None	Habitual
Usman A, *et al.*[146]	2012/ Pakistan	CAR	16/M	Palpitations for one week. PE:BP 150/95 mmHg, HR 110 bpm. Lab Test: No abnormalities	Arterial hypertension	3 cans/day	No history	None	2 weeks
Kaoukis A, *et al.*[147]	2012/USA	CAR	24/M	Chest pain, palpitations, dyspnea. Lab tests: Mild TnI elevation; NT-proBNP 8000 pg/mL. Imaging: Chest X-ray bilateral infiltrates; TTE: basal LV hypokinesia, EF 35 %	Takotsubo cardiomyopathy	Not specified	No history	None	Not specified
Avci S, *et al.*[148]	2013/Turkey	CAR	28/M	Palpitations, nausea. PE:Glasgow 3. Lab tests: pH 7.12; pCO_2 21.7 mmHg; pO_2 111.6 mmHg; HCO_2 15.3 mmol/L; TnI > 50 ng/mL; D-dimer 16,889; glucose 234 mg/dL. Imaging: TTE: left ventricular hypertrophy. Rest without alterations	Cardiac arrest: ventricular tachycardia. Death	3 cans/5 hours (250 ml)	No history	None	Habitual
Iglesias-Lepine ML, *et al.*[149]	2013/Spain	PSY	19/M	Nausea, vomiting, tremor of eyelids and hands. PE: BP 140/70 mmHg, HR 104 bpm; episodic tremor, epigastric tenderness. Lab test: no changes. Imaging; no changes	Catecholamine excess syndrome	2 cans	No history	Alcohol	Not specified
Iglesias-Lepine ML, *et al.*[149]	2013/Spain	PSY	18/M	Aggressive behavior. PE:BP 93/49 mmHg, HR 125 bpm, RR 28 rpm; pallor, diaphoresis, mydriasis, reflexes 2/5 Lab test: no changes. Imaging; no changes	Catecholamine excess syndrome	7 cans	Anxiety-depressive disorder	Alcohol	Habitual
Dikici S, *et al.*[150]	2013/Turkey	NEU	37/M	Tonic-clonic seizure. PE:Stuporous, bilateral Babinski, motor function 4/5 in all limbs, multidirectional nystagmus, diplopia. Lab tests: Hb 9.79 mg/dL. Imaging: MRI compatible with acute stroke	Ischemic stroke, epileptic seizure	3 cans (250 mL)	Iron-deficiency anemia	Smoker; alcohol	Occasional
Jonjev ZS, *et al.*[151]	2013/Serbia	CAR	54/M	Chest pain, fatigue, dyspnea. PE:BP 190/110 mmHg, HR 110 bpm. PE: Subacute aortic dissection DeBakey I Lab test: no changes.	Aortic dissection	4–5 cans/day	Severe hypertension, obesity	None	Occasional
Jonjev ZS, *et al.*[151]	2013/Serbia	CAR	26/M	Chest pain. PE:SBP 145 mmHg. Imaging:Aortic dissection DeBakey II Lab test: no changes.	Aortic dissection	5–6 cans	Bicuspid aortic valve; ascending aortic dilation	None	Not specified
Jonjev ZS, *et al.*[151]	2013/Serbia	CAR	48/M	Chest pain. PE:BP 145/95 mmHg. Imaging:Aortic dissection DeBakey I	Aortic dissection	Not specified	Family history of hypertension and myocardial infarction	None	Not specified
Polat N, *et al.*[152]	2013/Turkey	CAR	13/M	Chest pain. PE:RR 16 rpm; fourth heart sound (gallop). Lab tests: TnI peak 3.96 ng/mL. Imaging: TTE EF 54 % with apical hypokinesia	STEMI, coronary dissection	1 can	No history	None	First time

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Takahashi K, *et al.*[153]	2013/Japan	DIG	76/M	No symptoms reported. Lab tests: Vitamin B12 33,000 pg/mL	Hypercobalaminemia	½ can	Radical gastrectomy for gastric cancer (pT1N0M0)	None	Habitual
Görgülü Y, *et al.*[154]	2014/Turkey	PSY	21/M	Depression, crying, introversion, decreased concentration, aggressiveness, hallucinations	Psychotic episode	1–2 cans/day	No history	Occasional alcohol	Habitual
Ward AE, *et al.*[155]	2014/USA	CAR	45/M	Dizziness, lightheadedness	Ventricular tachycardia; ventricular fibrillation	3 cans in 3–4 hours	Tetralogy of Fallot; ICD	None	Habitual
Asensio-Sánchez VM[156]	2014/Spain	OFT	46/M	"Cloud" sensation affecting central vision	Bilateral central scotoma due to retinochoroidal vasoconstriction	5–6 cans/day (100 ml)	No history	None	Habitual
Katsue H, *et al.*[157]	2014/Japan	DER	32/F	Urticaria, odynophagia	Allergic reaction	1 can	No history	None	Habitual
Cruzado L, *et al.*[158]	2014/Peru	PSY	31/F	Irritability, hallucinations, insomnia, grandiosity, auditory hallucinations Lab test: no changes. Imaging; no changes. PE: no changes	Manic episode	10 cans/day	Sister with bipolar disorder	5 cups of coffee/day	Habitual
Ünal S, *et al.*[159]	2014/Turkey	CAR	32/M	Chest pain, palpitations, vomiting. PE:BP 178/92 mmHg, RR 22 rpm. Imaging:TEE anterior/apical hypokinesia; angiography: occlusion of left coronary and LAD	STEMI	5 cans	No history	None	Not specified
Krankl JT, *et al.*[160]	2014/USA	PSY	69/F	Insomnia, pressured speech, psychomotor agitation, distractibility, emotional lability, disorganized thinking	Manic episode	1/3 bottle daily	Bipolar disorder; hypothyroidism	2 cans Coca-Cola; 1000 mL coffee; pseudoephedrine	Habitual
Huang B, *et al.*[161]	2014/USA	DIG	36/M	Right upper quadrant pain, jaundice, fatigue, loss of appetite, 10 kg weight loss in one year. PE:Jaundice. Lab tests: AST peak 2,253; ALT peak 2,995; total bilirubin peak 23.1; creatinine 1.3; INR 3.2. Imaging: Biopsy: active hepatitis with bridging necrosis and lymphocytic infiltrate	Acute hepatitis	3 cans/day	No history	Alcohol: 10 beers in previous 3 hours	1 year
Greene E, *et al.*[162]	2014/USA	NEF	40/M	Fatigue, stiffness, dyspnea, hyperkinesia, agitation. PE:Hepatomegaly, respiratory effort; HR 109 bpm, RR 22 rpm. Lab tests:Creatinine 5.5 mg/dL, creatinine clearance 15 ml/min; sodium 130; potassium 5.6. Imaging: Not performed	Acute renal failure	5–6 cans/day (590 mL)	Type 2 diabetes mellitus, hypertension, anxiety, depression, PTSD, COPD, sleep apnea, pancreatitis; Former alcohol use	None	Several weeks
Shmelev A, *et al.*[163]	2015/USA	DIG	40/M	No symptoms reported	Acute pancreatitis	Not specified	Alcoholic pancreatitis; former alcohol use	None	Not reported

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Salanova-Villanueva L, *et al.*[164]	2015/Spain	NEF	25/F	Headache, tachycardia. PE:No abnormalities. Lab tests:Potassium 1.73 mEq/L; sodium 134; chloride 85; pH 7.58; PCO_2 46 mmHg; PO_2 86 mmHg; HCO_3 43.1 mmol/L; anion gap 5.9 mEq/L; urine chloride 22.2 mEq/L; urine sodium 210 mmol/L	Hypokalemia, prolonged QT, ventricular tachycardia	500–750 ml	No history	1 liter Coca-Cola daily	Habitual
Dikici S, *et al.*[165]	2015/Turkey	NEU	26/M	Unilateral vision loss with spontaneous resolution in 4 hours. Lab tests:No abnormalities. Imaging: No abnormalities	TIA	2 cans (250 ml)	No history	None	Occasional
Samantha D[166]	2015/USA	NEU	16/M	Headache, vomiting, gait disturbance, paresthesia in left lower limb. PE:Hyperreflexia, sensory deficit, positive Babinski, proprioception impairment. Lab tests: No abnormalities. Imaging: MRI with cortical/ subcortical diffusion abnormalities; angiography with irregularities in ACA, MCA, PCA	Reversible cerebral vasoconstriction syndrome	4 cans (236 mL)	No history	None	Occasional
Mournir K, *et al.*[167]	2015/ Morocco	NEF	28/M	Thirst, sweating, nausea. PE: BP 119/73 mmHg; HR 42 bpm; RR 17 rpm. Lab tests:Potassium 2.12 mmol/L	Hypokalemia	4 cans (1000 mL)	No history	None	Occasional
Wang HR, *et al.*[168]	2015/USA	PSY	32/M	Pressured speech, decreased need for sleep, increased daily activity, elevated mood	Manic episode	3–5 cans/day	No history	7–9 cups of coffee/day	2 weeks
Solomin D, *et al.*[169]	2015/USA	CAR	26/M	Chest pain. PE: Distress and diaphoresis. Lab tests: TnI 0.02 µg/L; CK-MB 160.1 µmol/L; altered lipid profile. Imaging:Catheterization with 100 % occlusion of left circumflex artery	STEMI	8–10 cans/day (473 ml)	No history	Smoker (1 pack/day)	Habitual
Iyer PS, *et al.*[170]	2016/USA	NEF	35/M	Dark urine, myalgias. PE:Muscle tenderness, dry oral mucosa. Lab tests: CK peak 100,290 U/L; AST 581 U/L; ALT 229 U/L. Other:Muscle biopsy with inflammation and necrosis	Rhabdomyolysis	2 bottles	Recent physical exercise	Cannabis.	Occasional
Sattari M, *et al.*[171]	2016/USA	CAR	28/M	Palpitations, bloody vomiting. PE:Irregular pulse 130 bpm. Imaging: No abnormalities. Other: Esophageal mucosal lacerations	Atrial fibrillation, Mallory-Weiss syndrome	2 cans/day	Obesity	Daily alcohol (2–3 beers), chewing tobacco	Habitual
Begolli AM G, *et al.*[172]	2016/USA	DER	19/M	Pruritus, increased local temperature, discomfort. PE: Pruritic macular lesions, confluent, several cm. Lab tests: No abnormalities	Exudative erythema	Not specified	Previous episode	None	Not specified
Grant RA, *et al.*[173]	2016/USA	NEU	44/F	Severe headache, neck stiffness, photophobia. Lab tests: No abnormalities. Imaging: CT: subarachnoid hemorrhage; angiogram: aneurysm	Subarachnoid hemorrhage with vasospasm	5 cans/day (470 mL)	No history	None	Habitual

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Harb JN, *et al.*[174]	2016/USA	DIG	50/M	Malaise, anorexia, abdominal pain, jaundice, vomiting, dark urine. PE:Jaundice, LUQ tenderness. Lab tests:AST 1,802 U/L; ALT 1,203 U/L; GGT 301 U/L; total bilirubin 10.3 mg/dL (direct 7.7); HCV antibodies positive. Imaging: US: gallbladder wall thickening. Biopsy: acute hepatitis with bridging necrosis and cholestasis	Acute hepatitis	4-5 cans/day	No history	None	3 weeks
Hernández-Huerta D, *et al.*[175]	2016/Spain	PSY	18/M	Insomnia, persecutory delusions, anxiety, agitation	Acute psychotic episode	6 cans/day	No history	Smoker (20 cigarettes/ day). Cannabis	7 days
Pagano CW, *et al.*[176]	2017/Costa Rica	OFT	48/M	Acute unilateral vision loss, malaise, palpitations, headache. PE: BP 180/120 mmHg; HR 100 bpm. Lab tests: No abnormalities. Imaging:Fundoscopy: intraretinal hemorrhage	Intraretinal hemorrhage	3 cans of 500 ml	Hypertension	None	Not specified
Icin T, *et al.*[177]	2017/Serbia	NEF	27/M	Nausea, vomiting, diarrhea, facial edema, dark urine. Lab tests:Potassium 2.8 mmol/L, sodium <100 mmol/L, lactate 11.4, leukocytes 21,000, creatinine 286 µmol/L. Imaging: No abnormalities	Severe hyponatremia	2 liters	Group A Streptococcus positive	Beer (5,000 mL), liquor (400–500 mL)	Occasional
Enríquez A, *et al.*[178]	2017/USA	CAR	19/M	Not reported. Imaging: No abnormalities	Cardiac arrest, ventricular fibrillation	3 cans of 300 mL	No history	None	Not specified
Enríquez A, *et al.*[178]	2017/USA	CAR	23/F	Syncope	Ventricular fibrillation	1 can	Peripartum cardiomyopathy (EF 20 %), ICD carrier	None	Occasional
Gharacholou SM, *et al.*[179]	2017/USA	CAR	27/M	Chest pain, dyspnea, diaphoresis. PE: BP 155/110 mmHg; RR 18 rpm. Lab tests:Troponin T 0.41; CK-MB 213.4 ng/ml. Imaging:TTE: LV apical akinesia; Cardiac MRI: LV apical akinesia	STEMI	4–5 cans	Familia (tío) con revascularización coronaria.	Fumador. En tratamiento con AINES.	Habitual
Zekavat O, *et al.*[180]	2017/USA	HEM	23/F	Menorrhagia. PE: Pallor. Lab tests: Hb 10.9 g/dL; bleeding time 9 s; PTT 59.2 s; PT 33.1 s; INR 5.97; factor X deficiency 8 %	Acquired vitamin K deficiency	Not specified	No history	Paracetamol.	6 months
Matiolli AV, *et al.*[181]	2018/Italy	CAR	23/M	Palpitations. PE: Irregular pulse, HR 150 bpm. Lab tests: No abnormalities. Imaging: No abnormalities	Atrial fibrillation	600 ml	No history	None	Not specified
Matiolli AV, *et al.*[181]	2018/Italy	CAR	22/M	Palpitations, chest pain, nausea. PE:Irregular pulse, HR 135 bpm. Lab tests: No abnormalities. Imaging: No abnormalities	Atrial fibrillation	750 ml	No history	None	Not specified
Matiolli AV, *et al.*[181]	2018/Italy	CAR	26/M	Palpitations, chest discomfort, anxiety, nausea. PE:Irregular pulse, HR 170 bpm. Lab tests: No abnormalities. Imaging: No abnormalities	Atrial fibrillation	600 ml	No history	Alcohol.	Not specified

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Casas-Gómez, *et al.*[182]	2018/Spain	PSY	22/M	Insomnia, anxiety, pressured speech, hypertimic mood, elevated self-esteem, loss of behavioral limits. PE:No abnormalitiesEF: Sin alteraciones.	Manic episode	20 cans of 250 mL	No history	None	Occasional
Zacher J, *et al.*[183]	2018/ Germany	CAR	25/M	Chest pain, vomiting. Lab tests:Leukocytosis, LDH, AST, ALT; CK 4,192 U/L; CK-MB 730; TnI 50 ng/ml. Imaging: TTE: reduced LV function with apical akinesia, EF 45 %; Coronary angiography: proximal dissection of left anterior descending coronary artery	STEMI	8 cans	Athlete; hypertension; family history of coronary disease	Smoker; Alcohol	Not specified
Quadri S, *et al.*[184]	2018/USA	PSY	17/F	Elevated mood, tangential thinking, decreased need for sleep, grandiosity, hyperreligiosity, delusional thoughts. Lab tests: No abnormalities	Manic episode	1–2 cans/day (470 ml)	No history Cannabis, oxycodone, cocaine	None	One week
Versha F, *et al.*[185]	2018/USA	DIG	27/M	Epigastric pain, nausea, vomiting. Lab tests: Lipase 2,375 U/L; AST 121 U/L; ALT 180 U/L. Imaging: Bile duct dilation (10 mm)	Acute pancreatitis	6 cans/day	Cholecystitis; previous pancreatitis episode	Not specified	Habitual
Al-Tamimi, *et al.*[186]	2018/USA	DIG	19/M	Recurrent epigastric pain episodes. Lab tests: Lipase >10,000 U/L. Imaging: MRI: acute pancreatitis	Acute pancreatitis (up to 10 episodes)	Not specified	SPINK1 mutation	Not specified	Habitual
Wajih Ullah M, *et al.*[187]	2018/ Pakistan	CAR	25/M	Chest pain, dyspnea, nausea, vomiting. PE:BP 155/95 mmHg; HR 110 bpm; S4. Lab tests: TnI 32.22. Imaging: No abnormalities	NSTEMI	7–9 cans/day	No history	None	Habitual
Butragueño L, *et al.*[188]	2019/Spain	NEU	8/M	Paresthesias, clonic movements, deviation of mouth commissure. PE: No abnormalities. Lab tests: No abnormalities. Imaging: No abnormalities. Other: EEG: bihemispheric epileptiform activity	Seizure	Not specified	No history	None	Habitual
Gupta N, *et al.*[189]	2019/USA	OFT	34/F	Scotoma. Imaging: OCT: hyperreflective parafoveal band without retinal thinning	Acute macular neuroretino-pathy	Not specified	No history	None	Not specified
Belzile D, *et al.*[190]	2019/ Canada	CAR	26/F	Cough, dyspnea. PE:Tachycardia, tachypnea. Lab tests: No abnormalities. Imaging: CXR: pulmonary edema, increased cardiothoracic index; TTE: dilated cardiomyopathy with reduced EF; Cardiac MRI: biventricular cardiomyopathy	Dilated cardiomyopathy	1–2 liters/day	Focal epilepsy	None	Habitual
Uwaifo GI[191]	2019/USA	DIG	46/M	Abdominal discomfort, nausea, vomiting. PE:Obese, actinic keratosis, upper abdominal tenderness. Lab tests: GGT, ALT, AST. Imaging: No abnormalities	Hepatitis, gastritis, pancreatitis	2–3 cans/day (470 ml)	Type 2 diabetes, fatty liver, obesity, hypertension, hyperlipidemia, hyperuricemia, nephrolithiasis; renal cell carcinoma (resected)	None	4 months

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Kelsey D, *et al.*[192]	2019/United Kingdom	PSY NEF	30/M	Agitation, anxiety, persecutory delusions. Low back pain, acute abdominal pain, vomiting. Lab tests: Cr 1,305 µmol/L; eGFR 4 ml/min; CK 3,336 U/L; CRP 38.8 mg/L; pH 7.27; microhematuria. Imaging: CT: subcutaneous edema in abdomen and pelvis	Psychotic episode; acute renal failure	12 cans/day (250 ml)	Overdose; depression	Alcohol	Habitual
Osman H, *et al.*[193]	2019/Dubai	CAR	32/M	Insomnia, palpitations, syncope. Lab tests: CPK 118 U/L; CK-MB 17 U/L; TnT 30 ng/L. Imaging:No abnormalities	Cardiorespiratory arrest (CRA); ventricular fibrillation	48 cans (250 mL)	No history	Vodka-containing drink	3 days
Ibrahim AM, *et al.*[194]	2019/USA	DIG	46/M	Epigastric pain, nausea, vomiting. Lab tests:Triglycerides 4,081; glucose 249 mg/dL. Imaging: CT: uncomplicated acute pancreatitis	Acute pancreatitis	3–4 cans/day	Diabetes Mellitus	None	2 weeks
Göçer M, *et al.*[195]	2020/Turkey	HEM	41/M	Syncope, dysarthria. Lab tests: Hb 8.4 g/dL; platelets 13,000; LDH 2,661. Imaging: No abnormalities	TTP	4 liters/day	No history	None	4 months
Göçer M, *et al.*[195]	2020/Turkey	HEM	19/F	Headache, menorrhagia. Lab tests: Hb 5 g/dL; platelets 6,000; LDH 1,703	TTP	500–1,000 mL/day	No history	None	15 days
Al Yacoub R, *et al.*[196]	2020/USA	NEF DIG	62/F	Confusion, fatigue, reduced intake, insomnia, nausea, vomiting, diaphoresis, decreased level of consciousness, weakness, lethargy. Lab tests: AST 4,333 U/L; ALT 2,866 U/L; ammonia 149 µmol/L; Cr 4; GFR 11. Imaging: No abnormalities	Hepatitis; acute renal failure	5–6 cans/day (470 mL)	Small cell lung carcinoma	None	Habitual
Hanif M, *et al.*[197]	2020/ Pakistan	CAR	22/M	Dyspnea, vomiting. PE:Arrhythmic pulse; HR 150 bpm. Lab tests:No abnormalities. Imaging: No abnormalities	Atrial fibrillation	2 cans	No history	None	Occasional
Báez-Ferrer N, *et al.*[198]	2020/Spain	CAR	45/M	Dyspnea, orthopnea, bendopnea. PE:HR 110 bpm; RR 24 rpm; O_2 sat 92 %; S3, pulmonary crackles. Lab tests: CRP 100 mg/dL; TnI 4.050 ng/ml; NT-proBNP 2,650 pg/ml. Imaging: CTR 0.5 with interstitial edema; TTE: dilated LV, EF 25 %; Cardiac MRI: edema and late enhancement; ECG: atrial fibrillation at 110 bpm	Acute myocarditis	1–2 cans/day	No history	Smoker	6 months
Garg A, *et al.*[199]	2020/USA	DIG	34/F	Epigastric abdominal pain, dyspepsia, colicky right upper quadrant pain. Lab tests:No abnormalities. Imaging:Endoscopy: intestinal metaplasia; biopsy: atrophic gastritis and antral metaplasia	Atrophic gastritis and intestinal metaplasia	1–2 cans/day	No history	None	15 years

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Fisk G, *et al.*[200]	2021/United Kingdom	CAR	21/M	Dyspnea, abdominal distension, asthenia, orthopnea, weight loss, dyspepsia, tachycardia. Lab tests: Cr 562 µmol/L; urea 47.4. Imaging:CXR: cardiomegaly; TTE: LV dilation with EF 1 1 %, RV dilation, biatrial dilation, pericardial effusion 0.4 cm; US: severe hydronephrosis and ureteral dilation	Bilateral hydronephrosis; dilated cardiomyopathy	4 cans/day (500 mL)	No history; ex-smoker	None	2 years
Uyanik M, *et al.*[201]	2021/Turkey	CAR	24/M	Dyspnea, fatigue, orthopnea. PE:HR 110 bpm; edema, bilateral crackles. Lab tests: Not reported. Imaging:CXR: right consolidation; CT: pulmonary edema and effusion; TTE: EF 25 % with global LV hypokinesia and dilation	Dilated cardiomyopathy	8–10 cans/day	No history	None	2 weeks
Staikoglou N, *et al.*[202]	2021/Grecia	NEU	14/M	Dysarthria, headache, right-hand weakness, hypoesthesia, right visual field defect. PE: BP 190/120 mmHg; HR 116 bpm. Imaging:MRI: T2 hyperintensity with restricted diffusion in left thalamus, splenium, and occipital lobe	Ischemic stroke due to posterior cerebral artery dissection	2 liters	No history	None	Occasional
Tinawi M, *et al.*[203]	2022/USA	NEF	40/M	Dark urine, arthralgias, myalgias. PE:No abnormalities. Lab tests: CK 139,680	Rhabdomyolysis	355 mL	Athlete	None	Not specified
Randhawa N, *et al.*[204]	2022/USA	DIG	29/M	Nausea, vomiting, epigastric pain. PE: Epigastric tenderness. Lab tests:Lipase 3,122 U/L; AST 115; ALT 110. Imaging: US: acute pancreatitis without gallstones	Acute pancreatitis	5–6 cans/day	No history	None	Habitual
Pallangyo P, *et al.*[205]	2023/ Tanzania	CAR	28/M	Retrosternal chest pain, dyspnea. PE:No abnormalities. Lab tests:Troponin I 9.6; CK-MB 54.2 ng/ml. Imaging:Coronary angiography: 100 % occlusion of left anterior descending artery	STEMI	2–5 cans/day	No history	None	Habitual
Yoshimine N, *et al.*[206]	2024/Japan	CAR	51/M	Palpitations, dyspnea, vomiting. PE:BP 150/72 mmHg; HR 120 bpm; RR 29 rpm. Lab tests:Leukocytosis; AST 167 U/L; CK-NAC 7,159 U/L; potassium 1.7 mmol/L; phosphorus 1.5 mg/dL; lactate 47 mg/dL; caffeine 71.4 µg/ml. Imaging:Pneumobilia. ECG: Sinus tachycardia, prolonged QT interval	Caffeine intoxication with QT prolongation	6 bottles (8 hours prior)	Hypertension; cholelithiasis	2 cups of coffee	Not specified
Othman M, *et al.*[207]	2024/Malasia	CAR	33/M	Syncope, central chest discomfort, palpitations. PE: HR 120 bpm. Lab tests: No abnormalities. Imaging:TTE: preserved LVEF; ECG: atrial fibrillation	Atrial fibrillation	Not specified	No history	Coffee	Habitual

(Continue)

Annex 1. Articles included in the review (continued)

Article	Year/Country	Field	Age/ Sex	Symptoms	Diagnosis	Amount	Previous pathology	Substances / other accompanying use	Consumption
Othman M, *et al.*[207]	2024/Malasia	CAR	27/M	Chest pain, profuse sweating, vomiting. PE:No abnormalities. Lab tests:Leukocytosis, platelets 436,000, Troponin T 310 pg/L. Imaging:TTE: LVEF 25 % with anteroseptal, anterolateral, and lateral hypokinesia (LV); coronary angiography: non-obstructive stenosis. ECG: ST elevation in I, aVL, V2–V6	STEMI	Not specified	No history	No.	2 years
Baéz-Ferrer N, *et al.*[208]	2024/Spain	CAR	31/M	Dyspnea. PE:No abnormalities. Lab tests:Troponin T 56.50; GFR 55 ml/min; Cr 1.56 mg/dL; NT-proBNP 1,323 pg/ml. Imaging:Cardiac MRI: EF 29 %; LV end-diastolic diameter 62 mm; ECG: sinus tachycardia	Dilated cardiomyopathy; acute pulmonary edema	4 cans/day	No history	No.	3 years
Sinha V, *et al.*[209]	2024/USA	NEU	30/M	Headache, seizure. PE:No abnormalities. Imaging: CT: basilar artery thrombus with hemorrhagic conversion	Seizure; ischemic stroke	Not specified	No history	400 mg caffeine (pills); tetrahydrocannabinol	Habitual
McMillan C, *et al.*[210]	2024/USA	DIG	62/M	Abdominal pain, vomiting. PE:BP 176/102 mmHg; LLQ tenderness radiating to back. Lab tests: Lipase 48 U/L; amylase 130 U/L; AST 51 U/L; ALT 54 U/L; ALP 138 U/L. Imaging:MRI: diffuse dilation of main pancreatic duct (6.4 mm)	Acute exacerbation of chronic pancreatitis	1 can/day (470 ml)	Chronic pancreatitis, atrial fibrillation, non-ischemic cardiomyopathy, chronic renal failure, gastric bypass, cholecystectomy; former cocaine and alcohol use	Marijuana	Habitual
Mannix D, *et al.*[211]	2024/Irlanda	PSY	51/F	Auditory and visual hallucinations, persecutory delusions. PE:No abnormalities	Psychotic episode	2–8 cans/day	Generalized anxiety disorder	None	Not specified
Van Houtte, *et al.*[212]	2024/ Belgium	DER	19/M	Pink sweating, pink discoloration of nails. PE:Chromonychia of nails. Imaging:Biopsy and histology without abnormalities	Eccrine chromhidrosis related to food coloring	Not specified	No history	None	Habitual

°C: degrees Celsius; µg/ml: micrograms/milliliter; µL: microliter; µmol/L: micromole/liter; NSAIDs: non-steroidal anti-inflammatory drugs; TIA: transient ischemic attack; ALT: alanine aminotransferase; ALP: alkaline phosphatase; AP: prothrombin activity; AST: aspartate aminotransferase; HCV Ab: hepatitis C virus antibodies; ED: energy drink; Tbili: total bilirubin; Dbili: direct bilirubin; CAR: cardiology; CK NAC: creatine kinase; cm: centimeters; CPK: creatine phosphokinase; Cr: creatinine; ICD: implantable cardioverter defibrillator; DER: dermatology; DIG: digestive; EEG: electroencephalogram; USA: United States; PE: physical examination; ECG: electrocardiogram; COPD: chronic obstructive pulmonary disease; TEE: transesophageal echocardiography; TTE: transthoracic echocardiography; F: female; HR: heart rate; EF: ejection fraction; RR: respiratory rate; g/dL: grams/deciliter; GFR: glomerular filtration rate; GGT: gamma-glutamyl transferase; Hb: hemoglobin; HCO_3: bicarbonate; HEM: hematology; HTN: hypertension; MI: myocardial infarction; CTR: cardiothoracic ratio; INR: international normalized ratio; pack-years index; IU/L: international units/liter; LDH: lactate dehydrogenase; bpm: beats per minute; ULN: upper limit of normal; M: male; MDMA: 3,4-methylenedioxymethamphetamine; mEq/L: milliequivalents/liter; Mg: magnesium; mg/dL: milligrams/deciliter; mm: millimeters; mL: milliliters; ml/min: milliliters/minute; mmHg: millimeters of mercury; mmol/L: millimoles/liter; ms: milliseconds; NEPH: nephrology; ng/ml: nanograms/milliliter; NEU: neurology; NT-proBNP: natriuretic peptide; OCT: optical coherence tomography; OPH: ophthalmology; $PaCO_2$: partial pressure of carbon dioxide; PaO_2: partial pressure of oxygen; CRA: cardiorespiratory arrest; CRP: C-reactive protein; pg/mL: picograms/milliliter; pg/L: picograms/liter; pH: hydrogen potential; Imaging: imaging test; PO_4: phosphate; PSY: psychiatry; PTT: partial thromboplastin time; PT: prothrombin time; QTc: corrected QT; MRI: magnetic resonance imaging; rpm: respirations per minute; BP: blood pressure; SBP: systolic blood pressure; CT: computed tomography; TBI: traumatic brain injury; ADHD: attention deficit hyperactivity disorder; OCD: obsessive-compulsive disorder; TnI: troponin I; TNM: tumor staging system; U/L: units/liter; LV: left ventricle; RV: right ventricle. STEMI: ST-segment elevation myocardial infarction; NSTEMI: non-ST-segment elevation myocardial infarction.