LETTERS TO THE EDITOR

Serotonin syndrome during a heat wave

Síndrome serotoninérgico durante una ola de calor

To the editor:

Serotonin syndrome is an adverse drug reaction characterized by dysfunction of the autonomic nervous system, with alterations in mental state and muscle tone, which in severe cases occurs with hyperthermia[1]. Advanced age, female sex and some pharmacogenetic alterations of monoamine oxidase (MAO) or metabolizing enzymes of antidepressant drugs can act as predisposing factors[2,3]. Extreme environmental conditions can also increase the susceptibility of the elderly to the effects of antidepressant, neuroleptic or anticholinergic drugs, by interfering with thermoregulation mechanisms[4,5]. During the first week of July 2015, four patients between the ages of 75 and 81 contacted the emergency department for high body temperature, tachycardia, hypertension and decreased consciousness. All were diagnosed with serotonin syndrome. The main characteristics of the cases are detailed below.

Case 1

A 75-year-old woman admitted on July 2, 2015 (maximum ambient temperature of the symptom onset day: 33ºC/100% humidity). Her pathological history included diabetes mellitus type II, hypertension (HT), dyslipidemia and major depression. The number of simultaneous chronic drugs at baseline was 15 [omeprazole, metformin, enalapril, hydrochlorothiazide, ezetimibe, acetylsalicylic acid (ASA), diltiazem, propranolol, solifenacin, paracetamol, trazodone, duloxetine, clomipramine, sulpiride, levosulpiride]. She had a temperature of 40ºC, hypertension, tachypnea, tachycardia, poor distal perfusion, decreased consciousness, hypotonia, myoclonus and lactacidemia. Suspected drugs were trazodone 100 mg/24 h, duloxetine 60 mg/24 h, clomipramine 50 mg/24 h, sulpiride 50 mg/24 h and levosulpiride 25 mg/8 h. Trazodone had started one month before admission, and the rest of the chronic medication had not changed in recent months. In the emergency room, antidepressant treatment was discontinued, support measures were started, cyproheptadine 4 mg/8 h and benzodiazepines. The patient persisted with serious sequelae: cerebellar syndrome and functional dependence. Causality (temporal sequence, plausibility, alternative causes, improvement upon withdrawal, re-exposure) was considered probable (yes/yes/no/yes/ not assessable).

Case 2

An 81-year-old male admitted on July 6 and 8, 2015 (maximum ambient temperature of the symptom onset day: 39ºC/50% humidity). The relevant pathological antecedents were diabetes mellitus type II, hypertension, atrial fibrillation, cerebrovascular accident (CVA) ischemic, recent comical episode, depression, moderate cognitive deterioration, malignant prostate cancer and diverticulitis. The number of simultaneous chronic drugs at admission was 13 (omeprazole, enalapril, atorvastatin, ASA, digoxin, dabigatran, bisoprolol, fesoterodine, calcifediol, calcium carbonate, levetiracetam, duloxetine, haloperidol). The patient had high fever, hypertension, tachycardia, tachypnea, diaphoresis, stiffness, clonic tonic movements, acute renal failure, leukocytosis and lactacidemia. The suspect drugs were duloxetine 60 mg/12 h and haloperidol 2 mg/ml 8 drops/24 h. Treatment with duloxetine had begun more than 3 years ago. Treatment with haloperidol, irregular, had begun more than 12 months ago. The acenocoumarol had been changed to dabigatran 1 month ago. Because of the onset of fever, he had started treatment with amoxicillin-clavulanic acid and nystatin. The rest of the chronic medication had not changed in the last 12 months. Antidepressant treatment was stopped, supportive measures were initiated and 4 mg/8 h of cyproheptadine was administered. The patient died. Causality was considered probable (yes/yes/no/yes/no assessable).

Case 3

An 81-year-old woman admitted on July 6, 2015 (maximum ambient temperature of the symptom onset day: 38ºC/40% humidity). As a relevant pathological background, she presented low-grade splenic lymphoma treated with rituximab and subsequent splenectomy in 2013, and severe osteoarthritis. The number of simultaneous chronic drugs was 7 [omeprazole, tolterodine, megestrol, latanoprost (eye drops), lormetazepam, citalopram and tramadol]. The patient's temperature was 41ºC, hypertension, tachycardia, disorientation, decreased consciousness and myoclonus. The suspect drugs were citalopram 20 mg/12 h and tramadol 50 mg/8 h. Treatment with citalopram had started 2 months earlier, and tolterodine had started the previous month. The rest of the medication had not been modified for more than 12 months. Antidepressant treatment was discontinued, supportive measures and levetiracetam were initiated; cyproheptadine 4 mg/8 h was administered. The patient presented severe sequelae with functional deterioration with respect to the situation prior to admission, residual confusional syndrome and post-extubation aphonia. The causality was probable (yes/yes/no/yes/no assessable).

Case 4

A 79-year-old male admitted on July 8, 2015 (maximum ambient temperature of the symptom onset day: 38ºC/45% humidity). He had a history of type II diabetes mellitus, hypertension, dyslipidemia, atrial fibrillation, heart failure, chronic bronchitis, low-grade MALT lymphoma, tuberculosis, pneumonia, and suprarenal insufficiency. The number of simultaneous chronic drugs at admission was 13 (omeprazole, metformin, glibenclamide, enalapril, hydrochlorothiazide, furosemide, simvastatin, spironolactone, ASA, tamsulosin, calcium carbonate/colecalciferol, hydrocortisone, trazodone, risperidone flas). The patient's temperature was 41ºC, decreased consciousness, hypotonia, myoclonus, and the suspected drugs were trazodone 100 mg/24 h and risperidone 0.5 mg/24 h. Both trazodone and risperidone had been started 4 months before, spironolactone 3 months ago, and the rest of the chronic medication had not changed for more than 12 months. Antidepressant treatment was discontinued and supportive measures were initiated. The patient died. The causality was probable (yes/yes/no/yes/no assessable).

The cases described are not typical in that none were preceded by a recent change in treatment[3,6]. In all of them neurological, vascular and infectious causes were excluded; and the presence of myoclonus and autonomic alterations in the context of chronic use of serotonergic agonists were decisive in establishing the diagnosis[3,6]. The four cases were presented coinciding with a heat wave that maintained constant high temperatures and humidity, with values around 40ºC and 100%[7], respectively. An environmental temperature higher than the normal temperature of the organism can compromise the mechanisms of heat dissipation

by radiation, conduction and convection; and a high environmental humidity (> 75%) makes the loss of heat through sweating also ineffective, making thermoregulation difficult. Elderly people may have reduced some of the mechanisms of thermoregulation, for example by a lower cutaneous hydration, a lower vasodilatation capacity of the skin and a lower autonomic reactivity, which in turn may be affected by various comorbidities[8]. Diabetes mellitus is frequently associated with autonomic neuropathy, which may compromise distal sweating function, and chronic hypertension could potentiate the vasoconstrictive response to serotonin[9]. Likewisesome say that psychiatric disorders, neurological disorders and anhidrosis are risk factors for hyperthermia in the face of a heat wave[9]. On the other hand, along with profuse sweating, diuretics can compromise hydration and precipitate prerenal renal failure, with increased plasma concentrations of drugs eliminated by filtration. Angiotensin-converting enzyme (ACE) inhibitors, beta-adrenergic blockers, and digoxin may compromise the pressor, chronotropic, and inotropic responses necessary to counteract the increase in temperature. Antimuscarinics can hinder sweating, and phenothiazines can hinder central thermoregulation; both have been associated with an increased risk of mortality due to an increase in temperature[8]. Three of the four cases described had a history of diabetes mellitus and hypertension, and all were receiving drugs that could have increased the risk of hyperthermia, including antihypertensives, antipsychotics or anticholinergic antimuscarinics. Three of the patients received treatment with cyproheptadine, which was administered at a dose of 4 mg/8 h. All patients improved on the muscular alterations, although the affectation by hyperthermia was serious in all of them and conditioned their poor prognosis. Two of the cases recovered, although with serious sequelae, and the other two died. We believe it is advisable to maintain a high suspicion of serotonin syndrome in elderly polymedicated patients who suffer neurological alterations and fever during periods of heat, even in the absence of recent changes in medication, to prevent or limit the possible neurological consequences of hyperthermia.

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Report of a case of leptospirosis: the importance of asking the patient about workplace factors

La importancia de interrogar el trabajo del paciente: a propósito de un caso de leptospirosis

To the editor:

Leptospirosis is a zoonosis usually produced by spirochetes of the genus *Leptospira interrogans*[1]. It is of global distribution, and considered as a rare disease closely related to environmental factors[1,2].

A 27-year-old man who visited in the emergency room for fever of less than 24 hours of evolution, lumbago and nausea, with normal physical examination except fever of 38.3ºC. Analytically he presented leukocytosis (19,000/µL) with neutrophilia (88.8%), PCR 2.06 mg/dl, creatinine 1.3 mg/dl and normal urinalysis. After an initial response to antipyretic treatment, she was discharged with the diagnosis of viriasis. Four days later he came back referring to persistent afternoon fever of up to 40ºC, nausea and arthromyalgia, mainly in the lower limbs. The physical examination was unremarkable. Analytically he presented 9,100 µL leukocytes (85% neutrophils), PCR 16.70 mg/dL, 80,000 platelets/µL, creatine kinase 1,155 U/L, GPT 87 U/L, GOT 130 U/L and total bilirubin 2.6 mg/dl . In the urine analysis there were 15-20 leukocytes/field and > 50 red blood cells/field. Urine culture was collected. After 24 hours of empirical treatment with intravenous ceftriaxone, he presented clinical deterioration again (persistence of fever of 40ºC and very intense arthralgias) and analytical (creatinine of 1.84 mg/dl and PCR of 22.8 mg/dl). The urine culture and serologies (HIV, hepatitis, CMV, EB virus, lues, dengue, toxoplasmosis, herpes, Lyme disease and Q fever) were negative. The patient was systematically re-interrogated, referring to working in sewer tunnels with the presence of rats in the work areas. With the suspicion of leptospirosis, which in turn could justify the alterations objectified in the urine, 3 samples were taken for diagnosis and treatment was started with intravenous penicillin 1 MU/4 h. At 12 hours after the beginning of the intravenous treatment, the fever disappeared and at 48 hours he presented an analytical improvement and was discharged. Afterwards, a positivity of leptospira IgM was obtained by ELISA. Microagglutination was not available in the reference centre, although the positivity of IgM and the therapeutic response to penicillin served as diagnostic confirmation in this case.

Leptospirosis is a rare disease in our environment, but with serious prognostic implications, with a reported in-hospital mortality of 6.3%2. Infection is acquired through contact with wounds on the skin, mucous membranes or conjunctiva with environments contaminated by urine or animal tissues[3]. The estimated incidence in the Community of Madrid is 0.36 cases/million inhabitants. Jaundice, renal failure, leukocytosis > 12,900/µL, rhabdomyolysis and thrombocytopenia are the main predictors of severity[3], all of them present in this patient. On the contrary, another characteristic feature, such as conjunctival suffusion, was absent[1,3]. Early antibiotic therapy has been shown to shorten the duration of the disease and prevent progression to more severe forms[3]. In the case presented, attention is drawn to the lack of response to ceftriaxone, described as an effective therapeutic alternative, of which we do not know the cause, although intravenous penicillin remains the treatment of choice. Diagnostic suspicion should be based on a history of environmental exposure, which requires a careful anamnesis in the emergency department[4].

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Double false result in a drug screen after accidental pediatric poisoning

Doble resultado falso en el cribado de drogas en una intoxicación accidental

To the editor:

The most frequent paediatric poisonings in children under 6 years of age in Spain are caused by accidental ingestions of psychotropic drugs[1,2]. In these cases, if a toxic urine screening test is requested, its limitations must be known.

A 17-month-old girl (12.1 Kg), who went to a paediatric emergency department for the presumed accidental intake 30 minutes before two tablets of quetiapine (50 mg) and a half of lormetazepam (1 mg). The initial clinical examination showed a triangle of normal paediatric evaluation (arterial O2 saturation: 99%, respiratory frequency (RF): 18 rpm, heart rate (HR): 154 bpm, blood pressure: 102/52 mmHg) and temperature (Tª) : 36ºC). The neurological examination revealed meiotic and reactive pupils, drowsiness and a score on the Glasgow scale of 15. The electrocardiogram (ECG) and gasometry were normal. A dose of activated charcoal (12.1 g) was administered. The analytical at 2 h posingesta showed neutropenia (1,910/µL) and plaquetopenia (87,700/µL). Toxic screening was negative for benzodiazepines and positive for tricyclic antidepressants (TAD) (Table 1). These results were reported as preliminary. The patient was admitted for clinical monitoring and cardiac monitoring. She remained stable, although without urinating from 2 h post-ingest. At 4 o'clock, the neurological examination was normal and the patient was discharged. At two weeks, the control blood count was normal. The confirmation of the toxicological analysis is shown in Table 1. The negative result of benzodiazepines was positive after pre-treatment of the sample based on the hydrolysis of the glucuronides of lormetazepam and reanalysis by the same immunoassay. In addition, lormetazepam was identified by gas chromatography coupled to mass spectrometry (GC-MS). The positive result of TAD was ruled out by screening these drugs and their metabolites in urine by GC-MS, only detecting quetiapine. Serum quetiapine was quantified by high performance liquid chromatography (HPLC) and the TADs were discarded (Table 1).

Lormetazepam in overdose can cause drowsiness, confusion and lethargy and, in more severe cases, hypotonia, hypotension, respiratory depression and coma[3]. Clinical symptoms in cases of overdose of quetiapine are predominantly neurological and anticholinergic with possible cardiovascular effects and changes in the ECG[3]. The patient presented decreased consciousness and urinary retention, signs that were resolved in

Table 1. Results of the toxicological analysis of screening and confirmation

Sample	Method	Magnitude	Result
Urine	Immunoassay DRI® (Abbott)[a]	Amphetamines	Negative
		Cannabis	Negative
		Cocaine	Negative
		Opiates	Negative
		Benzodiazepinas (pre-hydrolysis)	Negative (42)
		Benzodiazepines (pos-hydrolysis)	Positive (571)
	Syva® RapidTest d.a.u.® immunoassay (Siemens)[b]	TAD	Positive
	GC-MS (Agilent Technologies)	Lormetazepam	Positive
		Quetiapine	Positive
Serum	Quetiapine & TAD (HPLC, Chromsystems)	Quetiapine (ng/mL)	49
		TAD[c]	Undetectable

[a]Cutoff point (ng/mL) - Amphetamines: 1000; cannabis: 50; cocaine: 150; opiates: 300; Benzodiazepines: 200
[b]Cut-off point (ng/mL) - Amitriptyline: 800; clomipramine: 5,000; desipramine: 1,500; Imipramine: 1,000; norclomipramine: 850; nordoxepin: 5,000; nortriptyline: 1,000; trimipramine: 3,000.
cAmitriptyline, clomipramine, desipramine, imipramine, maprotiline, norclomipramine, nordoxepine, nortriptyline and trimipramine.

a few hours, probably due to digestive decontamination. The screening of benzodiazepines by immunoassay was falsely negative because lormetazepam is excreted in urine mainly in the form of glucuronides, which are not recognized by the test antibodies[4]. Hydrolysis with glucuronidases releases the drug, which is reactive in the immunoassay. This result was confirmed when lormetazepam was detected by GC-MS. On the other hand, the structural analogy of quetiapine with the main TAD explains the false positive result[5] that is confirmed by its detection in urine by GC-MS. In conclusion, the results of toxic urine screening tests should always be considered presumptive, since they can be false and confirmation by reference techniques is necessary for a correct diagnosis.

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Obligations of emergency responders and staff in cases of refractory asystole

Las obligaciones de los profesionales de urgencias en caso de asistolia no controlada

To the editor:

The article "Bioethics of family information on donation in out-of-hospital asystole", published last year in its Magazine[1], is well constructed from a methodological point of view and introduces accurate reflections. However, it deserves some nuances that contribute to understanding the problems that strict follow-up of its recommendations could pose. I believe, in fact, that this could lead to the commission of inaccurate healthcare practices that, although aimed at a laudable goal - guaranteeing organ procurement in patients with uncontrolled asystole - should be avoided. This result should, rather, be achieved through strict compliance with the regulations in force and the authorization of the necessary resources for this purpose.

Consequently, in light of the suggested procedure, I will express what, in my opinion, should be the protocol to follow when a healthcare professional assists a patient in out-of-hospital cardiac arrest. In the first place, what you have to do is follow the resuscitation protocol and, if necessary, transfer it to the nearest health centre prepared for the purpose. If this is impossible, I understand that the pertinent thing would be to declare his death and communicate it to his relatives as soon as the appropriate circumstances are met. What does not seem appropriate is that the professional refrain from issuing said statement and proceed directly to adopt measures aimed at preserving the organs (prior to obtaining family authorization), including the transfer of the deceased (in an ambulance) to a centre prepared for the donation. All this results in a certain contradictory point with the current regulations, which stipulate that "to initiate the preservation procedure it will be necessary that the medical team responsible for the patient has left a written record of the death, specifying the time of death." I am fully aware that this way of acting has a commendable purpose. However, it can cause serious consequences, such as the issuance of an inaccurate death certificate, which confuses the place and time of death, which could constitute a crime punishable by a fine of 3 to 12 months (Article 397 of the Criminal Code) and, beyond that, the transmission to families of an altered account of the facts.

What would happen if a family member asks uncomfortable questions afterwards? If the hospital prepared for the reception of organs to which he moved to the deceased was also the most suitable for the treatment of asystole, nothing. However, if this were not the case, that is, if there were a trained centre to deal with the emergency at a significantly shorter distance from the one to which the transfer was made, the health professional would be compelled to clarify the reasons for his / her action (with all what that would entail). This is the risk that our health care providers really assume when they lend themselves to a somewhat lax interpretation of the regulations caused by the impossibility of using ambulances for purposes related to organ donation (a question about what should be considered) and reluctance. to urgently inform families, for fear that this will reduce the rate of donations.

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Prehospital evaluation and treatment of pain due to trauma

Evaluación y tratamiento del dolor de origen traumático en medicina prehospitalaria

To the editor:

Pain assessment and the administration of analgesia are essential parts of the process of assistance to the traumatized patient. Acute pain has been known to have physiological and psychological adverse effects, which can influence the general assessment of the patient and the clinical course. The current guidelines for clinical practice in prehospital medicine emphasize the need to administer opioid analgesics to patients with multiple trauma with moderate-severe pain, regardless of the duration of the transfer; re-evaluate the pain every 3-5 minutes using a validated numerical scale and re-administer analgesia if necessary[1,2]. Specific quality indicators of pain management in the emergency department have been established: process (assessment and revaluation of pain using validated scales, administration of opioid analgesics and time of administration) and structure (protocols in organizations and training programs)[3]. However, it has been seen that the management of acute pain in emergencies and emergencies is not adequate and is scarcely documented[4].

We present a retrospective descriptive study. We reviewed the computerized care sheets of traumatic patients attended by the medical units of the Emergency System 061 of Galicia in 2015. We included patients over 18 years of age, with a score on the Glasgow Coma Scale ≥ 14 and with a systolic blood pressure ≤ 90 mm Hg. Demographic data were collected, the main diagnosis (polytraumatized, bone fractures and burns) the score obtained in the numerical scale of pain assessment, the analgesic used and the time of administration. We included 168 patients, 114 men and 54 women, with a mean age (SD) of 53 (SD: 20) years. In 23 (13.7%) cases, pain assessment was recorded by scale before and after analgesia. The initial mean score was 7.4 (2.1) and the final score was 4.2 (2.3). In 134 patients (79.7%) the drug used was registered; of these, opioids were administered in 131 (97.7%). There is a record of the administration time in 108 (80.6%). The average time was 12 (9) minutes.

The main finding of our work is that the numerical scale of pain assessment was only used in 13% of patients. Records are missing in relation to the administration of medication in 20% of cases. Both the analgesia time and the type of drug administered are in accordance with the recommendations of the guidelines; however, the absence of records constitutes an important limitation of this study. We do not know the causes of the lack of use of the numerical scale of pain assessment. It has been suggested that sex, age, race and saturation of emergency services are factors that influence the management of pain[5]. In the prehospital environment, where the patient professional ratio is more favourable than in a hospital, there could be other factors. It is necessary to perform additional studies aimed at developing strategies that allow us to improve the evaluation and treatment of pain in these patients.

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