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Sergio Ramos Pozón

Departamento de Enfermería Fundamental y Médico-Quirúrgica, Facultad Enfermería, Universidad de Barcelona, L'Hospitalet de Llobregat, Barcelona, Spain

E-mail address: sergioramos@ub.edu

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Precautions in prescribing serotonin reuptake inhibitors☆

Precauciones en la prescripción de inhibidores de la recaptación de serotonina

Dear Editor,

There are a considerable number of psychotropic drugs today that can cause endocrinological and metabolic changes which may affect both treatment adherence and the patient's quality of life and health status. Thus hyperprolactinaemia secondary to antipsychotics[1] — with its effects on gonadal function and bone metabolism —, or therapy with lithium and other antidepressants, with their effects on the thyroid function are just a few examples of this significant issue

Fluoxetine is a powerful selective serotonin reuptake inhibitor (SSRI) which is widely prescribed and indicated in the treatment of episodes of major depression, obsessive-compulsive disorder (OCD), and bulimia nerviosa.[2] The syndrome of inappropriate secretion of ADH (SIADH) consists of the secretion maintained by ADH failing to have its usual stimuli, in particular hypovolaemia and hyperosmolarity, and is characterised by the presence of hypoosmolar hyponatremia, urine sodium (NaU) above 40 mmol/l and urine osmolarity (OsmU) above 100 mOsm/kg, in absence of the effective reduction of volaemia (heart failure, ascities, hypovolemia,...), once thyroid and suprarrenal failure have been ruled out.[3]

Tumours, infections and very exceptionally drugs[4] are the most common causes. Antidepressants and in particular the SSRI have been frequently involved. Although not totally clarified, their physiopathological mechanism may depend on an increased secretion of ADH due to the stimulus of serotoninergic receptors and alpha-adrenergic receptors by the serotonin and noradrenaline.[5]

Pedrós et al.[6] reviewed 44 spontaneous reports of hyponatraemia and/or SIADH suspected of having been caused by SSRI between 1983 and 2003. Of these, 11 were attributed to fluoxetine. As a result, we present the case of a woman who developed hyponatraemia due to the use of this drug.

A woman aged 76 with a history of high blood pressure, Sjögren's syndrome and osteoporosis. She had been treated with 16 mg/day candesartan and 20 mg/day fluoxetine for the previous 4 months. She presented due to instability when walking, dizziness and mental torpidity of one month onset. Physical examination revealed the absence of oedema and signs of dehydration. Analysis highlighted a plasma NA of 125 mmol/l, plasma Osm 266 mOsm/l (vn: 280–300 mOsm/l), NaU 47 mmol/l and OsmU233 mOsm/l. Cortisol and thyrotophin (TSH) levels were normal.

On suspected SIADH secondary to fluoxetine, the drug was suspended and treatment was initiated with hypertonic saline solution and water deprivation. Clinical and analytical evolution was favourable, with normalisation of the natraemia.

According to its specification sheet[1] SIADH by fluoxetine is a serious and rare adverse effect (≥1/10,000 to <1/1000). Both Pedrós and Gandhi et al.[7] found there to be a higher risk of hyponatraemia due to SSRI in women over 65 years of age, in concomitant treatment with diuretics and a history of chronic kidney disease or heart failure. One cohort study of the British population[8] demonstrated a significant increase in the risk of hyponataemia with citalopram, fluoxetine and escitalopram compared with sertraline and paroxetine. The latter was found to be more frequently implicated in the Pedrós study.

To conclude, we believe that caution should be taken when prescribing SSRIs, especially in women over 65 years of age, where there is concomitant use of diuretics or a history of kidney or health failure. In these cases natraemia should be monitored and clinical follow-up should be performed at least in the first three months of treatment.

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Javier Miguel Martín Guerra*, Miguel Martín Asenjo, Luis Ángel Sánchez Muñoz, José María Prieto de Paula

Servicio de Medicina Interna, Hospital Clínico Universitario de Valladolid, Valladolid, Spain

*Corresponding author.
E-mail address: javi6vega@hotmail.com (J.M. Martín Guerra).

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Suicide: Contexts and persons☆

Suicidio: contextos y personas

Dear Editor,

Suicide is a public health problem of the highest magnitude. Of the 10 most common causes of death worldwide, suicide alone is the only one which has not decreased during the last decade, despite the enormous scientific accomplishments relating to it.[1] Despite the huge investment in research projects and increasing implementation of specific programmes, the prevention of suicide is a yet ''unresolved clinical requirement''.[2] In their editorial, Barrigón and Baca-García proposed that some of the bases upon which the field of investigation into the prevention of suicide has been constructed over the last few decades should be reformulated.[3] They suggested that the general framework of clinical research be based on individuals, and recommended evolving from the isolated study of risk factors to the definition of their algorithms, based on data processing with automatic learning techniques and personalised monitoring supported by new technologies.

We believe that the study and prevention of suicide should necessarily include a populational standpoint to understand and reduce their risk factors on an individual level. Since the publication of the Durheim sociological studies it has been accepted that suicide is a phenomenon where the rate at least partially responds to certain supraindividuals.[4] Some examples support this idea: several countries, such as Lithuania have standardised rates of suicide 5 times higher than other countries, like for example, Greece.[5] In U.S.A. the suicide rates in rural areas are double those of urban areas.[6] Indeed, within the same city, London, areas with higher ecological rates of social deprivation are associated with higher suicide rates.[7]

Modern epidemiology attempts to define what elements have a causal relationship with the presentation of diseases in populations. Development has led to paradigms which have determined the research questions and consequently the response obtained.[8] The initial idea was eminently ecological: it is believed that modern epidemiology emerged with John Snow and spatial distribution of epidemics of cholera in 19th century London. After this, the discovery of the tubercle bacillus and other infectious agents paved the way for the molecular era. Lastly, as a result of the second world war, epidemiology of chronic non-transmissible diseases appeared. These are centred upon the subject, their behaviour and individual risk factors such as a tobacco habit or high blood pressure. Characteristically, each era of epidemiology has focused on a single level of study (ecological, molecular, individual), negating all others to a greater or lesser degree.[8] Epidemiology of risk factors has therefore produced a sizable body of eminently individual causal knowledge. At present, however, we are witnessing the growth of an integral approach to epidemiology which some people have called multilevel epidemiology, and which proposes that disease phenomena respond to causal patterns with rationale that act at different molecular, individual and populational levels which interact on complex hierarchical networks.[9] From this outlook, information collected by ecological variables, far from being considered an exchangeable estimator with data collected on an individual level, enjoy their own relevance for proper understanding of the sociological motives of the disease.[10]

One initiative stands out as an optimisation opportunity when giving ecological causal factors the place they have earned: overall mental health collaborations. By definition, an epidemiological study is able to analyse potentially causal relationships when a control exists with which to generate comparison. Through the inclusion of different research contexts using ecological measurements to pinpoint the differences between contexts, we may calculate the role played by supraindividual variables on suicide rates.

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