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REVIEW ARTICLE

Eating disorders: Considerations on nosology, etiology and treatment in the XXI century☆

Luis Rojo Moreno[a,b,c,*], Javier Plumed Domingo[a,b], Llanos Conesa Burguet[a,d], Francisco Vaz Leal[e,f], Marina Diaz Marsá[g], Luis Rojo-Bofill[h], Lorenzo Livianos Aldana[a,b,c]

[a] *Departamento de Medicina, Universidad de Valencia, Valencia, Spain*
[b] *Servicio de Psiquiatría, Hospital Universitari i Politècnic La Fe, Valencia, Spain*
[c] *Research Group CIBER CB/06/02/0045 CIBER Actions—Epidemiology and Public Health, Universidad de Valencia, Valencia, Spain*
[d] *Unidad de Psiquiatría, Hospital de Sagunto, Valencia, Spain*
[e] *Área de Psiquiatría, Departamento de Terapéutica Médico-Quirúrgica, Facultad de Medicina de Badajoz, Universidad de Extremadura, Badajoz, Spain*
[f] *Unidad de Salud Mental y Trastornos Alimentarios, Complejo Hospitalario Universitario de Badajoz, Servicio Extremeño de Salud, Badajoz, Spain*
[g] *Departamento de Psiquiatría, Hospital Clínico, Universidad Complutense, Madrid, Spain*
[h] *Fundación Investigación, HU La Fe, Valencia, Spain*

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Abstract Amazing advances have been made in medical sciences since the first international conference on eating disorders (ED) was held in the 1970s, and there have been remarkable changes in the field of ED itself. Back then, virtually all that was talked about was anorexia nervosa (AN); clinicians and researchers were mainly concerned about the possible hypothalamic and endocrine factors that seemed to be involved and there had been no epidemiological studies or controlled trials with psychiatric drugs or psychotherapy. Although the picture today is quite different, there are still significant gaps which even affect the classification of these disorders, as well as their neurobiological bases and both the pharmacological and psychological treatments which should be used. This paper gives a brief summary of these gaps and discusses the need to find endophenotypes which may help in categorising and directing research into these disorders. Mention is made of possible contributions from other fields for the benefit of greater progress in understanding EDs. Specific reference is made to the addictive model, out of which neuropsychology and animal models may provide data transferable to our area of expertise. Lastly, the current state of ED treatment is discussed with pointers as to from what perspective it would be most useful to seek improvements.
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* Corresponding author.
E-mail address: luis.rojo@uv.es (L. Rojo Moreno).

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Los trastornos de la conducta alimentaria: consideraciones sobre nosología, etiopatogenia y tratamiento en el siglo XXI

Resumen Desde la primera conferencia internacional sobre los trastornos alimentarios, celebrada en los años 70, hasta la actualidad, el desarrollo de las ciencias médicas ha sido sorprendente. En el campo de los trastornos de la conducta alimentaria (TCA) también se han producido cambios notables. En las fechas iniciales prácticamente solo se hacía mención de la anorexia nerviosa y sus posibles factores hipotalámicos y endocrinos; no había estudios epidemiológicos ni ensayos controlados con psicofármacos o psicoterapia. El panorama actual es bien diferente aunque hay carencias importantes que afectan a la propia nosografía de estas patologías, al conocimiento de sus bases neurobiológicas y a sus tratamientos, tanto farmacológicos como psicológicos. Teniendo en cuenta estas circunstancias, hacemos un breve sumario de las carencias existentes y planteamos la necesidad de encontrar endofenotipos que ayuden en la categorización e investigación de los TCA. Se hace mención a las aportaciones que desde otros campos hacen posible un avance más profundo en el conocimiento de los TCA. Específicamente se recurre al modelo adictivo, desde el cual la neuropsicología y los modelos animales pueden ofrecer datos trasladables a nuestro ámbito de conocimiento. Por último, se hace mención al estado actual de los tratamientos de los TCA y se señala desde qué perspectiva sería útil plantear mejoras.
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Introduction

Eating disorders (EDs) are clinical entities that are drawing more and more interest among professionals and citizens, above all in the face of the perception that this is an emerging problem that is spreading. From the 70s until now, there have been many steps taken in gaining knowledge on EDs and they currently represent a consolidated category within the international classifications of mental illnesses, with anorexia nervosa (AN) and bulimia nervosa (BN) occupying a leading position. Other eating alterations, such as what is called *binge eating disorder* (BED), have been included in the DSM-IV, although only as potential diagnostic categories.

Over the last few years, many epidemiological and risk factor studies[1,2] have been published internationally and in our own country. These studies have provided solid evidence on the role that genetic factors play in AN and BN,[3,4] as well as on the influence of socio-cultural factors.[5,6] In addition, there are various meta-analyses on psychological as well as pharmacological treatments in AN, BN and BED. Obviously, this does not mean that all the problems have been solved, as the short- and medium-term challenges are numerous. In our opinion, the main questions to resolve involve the validity of the diagnostic categories we presently employ (with the advances and knowledge that research in other psychological, medical and psychiatric specialties are providing) and with analysing the efficacy of the treatments now in use. These factors will have to establish the priorities in the lines of work for the next few years. In light of the matters just brought up, this has to be organised around 3 great areas: nosology, aetiopathogenesis and treatment. To analyse the current situation and the short- and medium-term challenges, these 3 specific areas are reviewed in the following sections.

Nosology in eating disorders

One of the main problems to be solved in the matter of EDs is that of defining the case. As is true of many other diagnostic categories in psychiatry, the current diagnostic criteria do not have the requisite validity and reliability. This affects their usefulness when the attempt is made to apply them in clinical practice, to establish a prognosis on the course of the illness or to plan treatment.

The updates to the diagnostic criteria proposed for the DSM-V do not involve a substantial change with respect to those of its predecessor, the DSM-IV. However, they do eliminate some ambiguities and clarify the panorama insofar as identifying the clinical entities proposed. They also advance, to a certain degree, towards coinciding with the other basic diagnostic guide, CIE-10.

In the latest version of the DSM-5 draft (May 2011), accessible on the American Psychiatric Association website, we can see how the proposal for the chapter on eating and food disorders is still based, as regards EDs, on 2 main sets of symptoms—AN and BN—with a single additional clinical entity (BED). The key clinical elements for identifying AN and BN are decreased food ingestion and the consequent significant weight loss (in the case of AN) and the existence of binges and compensatory tactics (in the case of BN). However, together with these, other elements more or less common to both EDs appear, as modulating factors: fear of gaining back the weight lost, distorted perception of one's own body or extreme influence of the body in self-assessment. While the differentiation between restrictive and purging AN, present in DSM-IV, is conserved in DSM-V, only the purging form is considered a BN subtype. This makes the majority of the cases previously identified as non-purging BN switch to the category of binging disor-

der (which, as has been indicated, has its own identity in the new guide). In DSM-V, the diagnostic criteria become more flexible, similar to the situation in the CIE-10, fitting in atypical forms (with incomplete symptomatology) and subclinical (subthreshold) forms along with AN, BN and binging disorder. This is significant because, along the lines of what we will see below, a great number of patients move between the clinical/subclinical dimensions throughout ED development. It should be pointed out that medium- and long-term diagnostic stability of EDs is generally low,[7,8] with frequent patient migration from one diagnostic category to another.

To resolve the problem of diagnostic stability, Fairburn and Cooper[9] proposed adopting a *transdiagnostic ED model* some years ago. This model is structured around a single diagnostic category, defined by the presence of overvalued worries related to food, weight and figure control. Despite representing a notable advance in ED conceptualisation, the *transdiagnostic model* has a fundamental limitation, which stems from the elevated prevalence of these worries among the adolescent population.[10] That these concerns are so widespread among adolescents makes the border between cases and non-cases too imprecise and permeable. The psychopathological elements chosen by Fairburn and Cooper to define the key category have themselves limitations and can be subject to criticism in the sense that they can be influenced excessively by the ''occidental viewpoint'' applied to EDs. Based on Russell,[11] the only elements that have remained stable throughout the history of AN have been its prevalence in young women, food restricting and the patients' lack of recognition of illness. The rest of the symptoms could be understood as cultural elaborations, which could be demonstrated in the tangible differences in different societies in what we could call *added symptoms*.[5,6] These data support the need to include other elements that can provide greater reliability and validity to the diagnoses. In line with the *transdiagnostic model*, such elements would be choosing the existence of a primary eating dysfunction capable of affecting the subject's health and personal functioning as a central diagnostic category.

In a recent study focused on a critical analysis of the transdiagnostic model through applying *Hill's Criteria of Causation*, Birmingham et al.[12] reached the conclusion that AN and BN are better conceptualised as differentiated entities than as clinical forms of the same process: despite their strong association, analogy and possible common underlying biological mechanisms, both disorders do not fulfil the criteria normally applied to single clinical entities.

An alternative to the *transdiagnostic model* has been conceptualising the food EDs as subcategories of anxiety disorders.[13] From this perspective, EDs are characterised by the presence of anxiety and defensive behaviour focused on food, eating, weight and the body. Facts in favour of this viewpoint are the high comorbidity of ED and anxiety disorders, the association with personality disorders in which anxiety is also a significant factor (such as the obsessive-compulsive personality, borderline personality and avoidant personality disorders) and the significant grouping of anxiety disorders among family members of subjects with ED.

Another alternative way of conceptualising EDs stems from the consideration of personality and defining 3 basic patient subtypes: the high functioning-perfectionist, the constricted-overcontrolled and the emotionally dysregulated-impulsive.[14] This differentiation seems to have implications related to potential psychiatric comorbidity, to the level of psychosocial functioning and to an element that could have an aetiological dimension: antecedents of childhood sexual abuse. This is true because these clinical factors appear to be significantly associated with the 3 patient subtypes defined. In agreement with this point of view, the most appropriate way to divide the ED population would be differentiating between patients who have only had symptoms characterising AN throughout their lives, those that have only had symptoms characterising BN and those that have presented both clinical forms (patients with purging AN and patients with BN and AN antecedents). Along these lines of work, there are other notable studies that have attempted to identify ED phenotypes based on psychopathological criteria and personality-linked factors. In a recent study, Krug et al.[15] applied latent profile analysis techniques to the data provided by the revised Cloninger Temperament and Character Inventory and identified 6 patient groups, with specific personality profiles. They called these groups the ''self-focused'', ''inhibited'', ''average'', ''impulsive'', ''adaptive'' and ''maladaptive''. The last 2 groups presented the highest levels of eating pathology, but the concordance of the groups isolated with the clinical differentiation proposed by the DSM-IV was very low. In this line of work as well, Peñas-Lledó et al.[16] (based on Stice's[17] model) proposed that there are 4 patient subtypes, according to diet intensity and patient affective manifestations. However, when they analysed the concordance of their proposal with the DSM-IV diagnostic categories, they likewise found that the concordance was very low. This group of authors[18] also advanced a proposal based on the existence of social anxiety and novelty-seeking, a temperament-linked trait. Although they found a solution based on 5 factors, after analysing their concordance with the patient groupings based on DSM-IV diagnostic criteria, they found the same low correlation encountered in previous studies.

Finally, another matter (which we will explore in the following section) is considering EDs as a variation of addiction disorder. To avoid duplications, this aspect will be discussed only briefly now, approaching the questions related with neurobiology in the following section. The fact that there is a possibility that there is an addictive process involved with food in EDs must be mentioned. However, as with other proposals, there seem to be elements in favour and again this manner of understanding EDs, with part of the literature dissenting and another part supporting it. Wilson,[19] for example, has indicated being against the idea of considering that food (even some substances as specific as carbohydrates) can be addictive, despite the fact that the clinical pattern with which the patients with EDs present often brings to mind a potential association with addictions. At any rate, it would not be so much an addiction to the substance (the food) as a ''behavioural addiction'', which should be understood as a dependence disorder linked to various human activities (often pleasurable) not related

to the ingestion of substances; this group would include pathological gambling, sex addiction or pathological exercising. In the context of EDs, binging is perhaps the most suggestive clinical phenomenon. In fact, if we focus on the basic characteristics of binge eating, we see in it many elements in common with addictive behaviours: (1) discomfort when it cannot be performed; (2) ingestion of a greater quantity or during a longer period than intended; (3) ''incapability'' to reduce or control its performance; (4) dedication of a lot of time to the planning, performance or recovery phases; (5) frequent interference with carrying out other activities (putting off social relationships, for example); and (6) maintaining these behaviours in spite of the fact that the individual is aware of and fears their negative consequences. All these characteristics seem to be seen clearly in many BN and BED cases. However, what about AN? Can you be addicted to not doing something or not consuming something? In other words, does AN fall within the addiction model? The answer to this question would be affirmative if we bear in mind that (1) *tolerance* can be equivalent to the progressive increase in food restriction until complete fasting is reached, (2) *abstinence* can correspond to the anxiety that arises in the face of the obligation to eat, (3) the patients cannot control diet restriction, (4) the patients occupy a significant portion of their time thinking about how to avoid eating, (5) there are serious social, work, academic and psychological interferences and (6) there is a clear impossibility of limiting the restriction, even knowing the negative consequences that it has. Consequently, although it is not a question of throwing oneself entirely into this proposal, we believe that it is necessary to be bold in the current level of our knowledge and faced with the lack of developed therapeutic or aetiopathogenic models. The addiction model can offer, at least, a possible way of subtyping some of the cases, in which this could intervene more specifically. The addiction model also brings along interesting aetiopathogenic viewpoints that will be discussed below.

Eating orders from the viewpoint of addiction neurobiology

The literature in the areas of neuropsychology, neurobiology and the knowledge provided by animal models in the field of addictions is tremendously exciting. It can also shed some light over eating dysfunctions.

In the specific field of neuropsychology, Verdejo-García and Bechara[20] have transferred Damasio's theory of *somatic markers* to addictions. These authors start from the evidence of a disability in reaching long-term decisions in subjects with lesions in the ventromedial prefrontal cortex (VMPFC). In this region, which receives information from the amygdala, weighing the negative positive aspects of the decisions we reach occurs. The *somatic marker hypothesis* holds that the experiences we have (both positive and negative) remain associated with autonomic somatic phenomena in such a way that, when we are about to decide in the face of an experience, a somatic reaction (somatic marker) is produced. This marker indicates the positive or negative character of the reaction, facilitating or slanting the meaning of our decision in the VMPFC. This phenomenon can be assessed experimentally using what is known as the *Iowa Gambling Task*, a neuropsychological task develop to analyse the functional state of the VMPFC that is applied while electrodermal activity is registered simultaneously. If the VMPFC is intact, increased electrodermal activity (the somatic marker) is detected immediately before the decision. In contrast, in addictions there is a dysfunction detected in the VMPFC, associated with amygdala hyperactivity. This could explain the incapacity that addicts show in appropriately taking decisions, giving themselves over to immediate rewards and ignoring the long-term consequences of their behaviour.

Is the *somatic marker hypothesis* applicable to EDs? It must be said that the neuropsychological studies performed along this line are discrepant; only a single study, by Tchanturia et al.[21] backs the theory. A later study[22] by the same team did not reach this conclusion, nor did that of Herrera.[23] The latter study, in a very interesting design on 19 patients with BN, rules out the hypothesis upon demonstrating that the patients gave in (just as the controls did) when there is no reward.

Briefly, the neuropsychological data on the *somatic marker hypothesis* do not make it possible to approximate EDs to the addiction model. However, the fact that the samples used in the studies are not very large has to be considered, as well as that they could allow subtyping the EDs in the future, with a type characterised by a VMPFC deficiency. Cavedini et al.[24] have already found differences in advantageous decision-taking in patients with restrictive and purgative AN, which approximate the former to the neuropsychological model of obsessive-compulsive disorder.

Animal models are not so easy to apply in the case of psychiatry or clinical psychology as in other areas in medicine. There are no appropriate animal models for many of the mental disorders and the animals cannot tell us how they are feeling. At any rate, translational research can be an area of great future development. An example is the recent article by Attwood et al.[25] These investigators, using a complex method, showed that inhibition of a protease in the amygdala (neuropsin) through drugs or genetic manipulation blocks anxiety against stress in rats. What is interesting about the model is that the behaviour that makes it possible to infer that the rat is anxious is that it moves about the experimental area staying close to the walls, avoiding open and illuminated spaces, as generally happens in agoraphobia. The simplification is enormous, but we should not forget that we are mammals and that we share some behaviour patterns with all the others, in addition to many similarities in our most primitive brain.[26]

Speaking of animal models bring us to the mention of the work of Hoebel's team.[27] These authors worked with rats, comparing undernourished animals with others normally fed. They demonstrated that malnourishment facilitates the process of transforming normal behaviour into addiction behaviour, through the process of modifying neuronal neuroplasticity at the level of the dopaminergic neurons of the nucleus accumbens. Data of interest include that in undernourishment, at least in rats, there is increased

dopaminergic release in the accumbens when alcohol or stimulants are administered, and also that the processes of behaviour extinction are more difficult in this state, with relapse in the excessive consumption being easier in the face of environments similar to those in which the conditioning is produced. What is more, these authors demonstrated that sucrose, a desirable food, is capable of producing similar effects in undernourished rats; this is because the presentation of the desirable stimulus (in contrast to what occurs in well-fed animals) provokes intense dopaminergic release successively, in the same way that toxic substances do. We should add that sucrose reduces the desire for cocaine in over 90% of individuals[28] and that exposure to successive cycles of restriction and free access to sucrose makes it possible to produce an experimental model of binging. These data, interesting in and of themselves (suggesting that addiction behaviours use, or kidnap, the neurobiological structures the natural reinforcers utilise) also emphasise that malnourishment is a situation that facilitates establishing of neurophysiological variations (modifications in neuronal neuroplasticity) overlapping those demonstrated in addition behaviours.

The modification to the neurobiological systems associated with prolonged drug consumption has also been shown in behavioural addictions.[28] We know that physical exercise competes with self-consumption of drugs, which increases neurogenesis in the hippocampus, whose reduction is associated with depressive symptoms, In addition, curiously, in conditions of food restriction, rats given free access to running wheels stop eating, even to the point of dying because they cannot stop their activity.

Based on what has been indicated, we can state that substance addictions and behavioural addictions have, at least in part, shared substrates. Furthermore, malnutrition seems to favour significantly not only the generation of addiction, but also makes recovery more difficult and relapse easier. It is plausible, and the animal model based evidence insinuates, that at least some of the EDs can be considered from the pattern of behavioural addictions. In this sense, Gearhardt et al.[29,30] (using functional brain nuclear magnetic resonance imaging in humans) demonstrated that higher scores on a scale of food addiction are associated with a neural activation pattern similar to that of addiction-type behaviours.

How would behavioural addiction be explained from a neurobiological point of view? One model is that proposed by Robinson and Berridge,[31] later applied to food motivation by Volkow et al.,[32] which is known as the *incentive sensitisation theory*. According to this model, the dopaminergic waves provoked by the stimuli associated with addiction facilitate over-triggering these behaviours and the environmental conditions associated with them, making them excessively important. The nucleus accumbens and the mesolimbic/mesocortical dopaminergic system would be the main structures involved in the development of addiction in general.[29] However, there is a broader model, which does not reduce addiction to a heightened incentive to a substance or behaviour. We could call this the *Panksepp model*, Panksepp being the author of a developmental, neuroscientific model of emotions. In his opinion,[33,34] emotions have a specific meaning; they are not mere epiphenomena of the brain, they are associated with biologically significant situations and are a response to the adaptation needs of organisms. We mammals share many basic emotional systems, so (with due limitations) it is possible to consider similarities in the neurobiological systems sustaining them. The search-reward system and the system that sustains panic and grief are among the set of basic emotions in the matter which we are considering. The reward system drives the organism to seek relief from its needs and receive consequent gratification. It would thereby have an aspect linked to rewards, associated with the dopaminergic system as indicated earlier, as well as a consummatory aspect (of pleasure) associated with opioid (*mu* and *delta* receptors) release. Based on the *Panksepp model*, it can be sustained that the reward system has promoted social bonding in evolution. Evidence in animals shows that individuals are capable of ignoring their own safety and exposing themselves to situations of risk in favour of defending other subjects; they do so because, through this behaviour, they obtain advantages having to do with social status. This possesses an adaptive nature, both for maintaining the species and for the well-being provided the organism by feeling accompanied, cared for and, in short, bonded. The price to be paid is the discomfort generated by situations in which there is no bonding, with the unease or stress of separation and consequent grief behaviours. In this sense, from the perspective of the neuroscientific model of emotions, it supports the idea that addiction behaviour is just self-soothing that substitutes the bonding experience that is what the addict really desires. The psychological models of behavioural addictions back up this viewpoint; behavioural addictions would be lifestyles in which specific activities offer the individuals the opportunity to be absorbed by soothing situation and to eliminate the problems of daily life from their consciousness.[35] Looking at it from the ED perspective, we can see how patients respond to this profile on many occasions. Insecurity, fear of social exclusion, experiences of social exclusion or its expectation, difficulties in adolescent transition processes, emotional disorders and social anxiety[36] (as well as a good number of family and socio-cultural phenomena) convert these individuals into a population that is especially prone to engage in non-ideal behaviours, with which they hope to obtain satisfaction and security. One of these behaviours is corporal change through food restriction, which can become a trap from which it is sometimes difficult to break free—and undernourishment itself can be a crucial factor. It is not uncommon to hear female patients with EDs express statements that support that basic emotional element in their behaviour: ''It's that I won't be happy if I'm not focused on food'', ''when I didn't eat, I felt that I had achieved what I wanted'' or ''I felt much better when I could leave the house and get out of making snacks that way''. All these patients express an error that pushes them in a direction in which they will never be happy, from which they can escape by themselves only with great difficulty and in which (due to the neuroplasticity modifications that will undoubtedly be produced in their neuronal synapses, and which will be facilitated by malnutrition) they will tend to relapse many times without a clearly perceptible reason.

Treating eating disorders

Before concluding, we would like to share a brief reflection on the efficacy of current treatments. There are many revisions available on now abundant controlled therapeutic studies about AN, BN and BED.[37–42]

In AN, there do not seem to be any tests that support the use of any specific treatment, whether psychopharmacological or psychological.[43] A very recent, extensive study[44] reached the conclusion that there is no type of psychological treatment that seems to provide significant advantages other the others for patients with AN (although biases in sample selection and patient follow-up are very large). Along the same lines, an equally fresh study emphasises the usefulness of family therapy with patients with AN, although with no great differences over other forms of treatment.[45] Two relatively modern studies[46,47] show, surprisingly, that interpersonal therapy, cognitive behavioural therapy and clinical management with non-specific support therapy provide similar results in AN, in both the short- and long-term. Vanderlinden[48]—in an article that we recommend reading—emphasises the importance of the therapeutic alliance, of not overevaluating the importance of cognitions excessively, of paying more attention to social and family processes and, basically, of focusing on emotional experience and the emotional meaning of personal experiences: that is, on the emotional schemes that sustain patient attitudes, cognisance and behaviours.

In BN and BED, the most efficacious proposal is—and has been for a long time—the combination of psychotherapy and drug treatment,[49] which can provide good results.

Conclusion

After all of what has been indicated, we still have responded to the question: what is the direction we should take now? It is presumptuous to state that we know what will happen in future. Scientific development is exponential and it is logical to think that it will be possible to develop more reliable diagnostic systems that taken the nuclear, shared symptoms of EDs into consideration. Insofar as nosological problems, the imminent appearance of the DSM-V does not seem likely to solve many of the problems that we have before us presently and that we have pointed out in relation to this matter. This is especially true with respect to what are called the ''unspecified'' disorders,[50] to the limits between diagnostic categories[51] and specific populations,[52] among others. As Keel et al.[53] point out, we tend to study what we have previously defined, without noticing the fact that the diagnostic classifications are not necessarily the clinical reality. That is why we obviously have to continue searching for alternative categories and models.[54] It would not be surprising that in the future we could rely on neuropsychological endophenotypes to make it possible to subtype some of the EDs, nor that we could have neurobiological endophenotypes available. In this sense, translational research is crucial. We should consequently make efforts to collaborate with areas of knowledge currently foreign to clinical practice, which can help us to construct clinical models different from the present ones or to put work hypotheses that are now unknown to us to the test. Our interest in presenting here the advances now being made in a specific model that can contribute something—addiction—does not translate into a defence of or blind support for it in all its terms and conditions. Instead, it is a mere example of the open-mindedness with which we should consider our clinical and research work in the coming years.

Ethical disclosures

Human and animal protection. The authors declare that for this investigation, humans or animals were not used.

Data confidentiality. The authors declare that no patient data appear in this article.

Right to privacy and informed consent. The authors declare that no patient data appear in this article.

Conflict of interests

The authors have no conflict of interests to declare.

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