

Usefulness of diagnostic models in the process of qualifying thought contents as delusional: case analysis

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Summary

Aim: The purpose of this study was to attempt to apply a two-factor diagnostic model for delusional content qualification.

Method: This is a case study of a patient with a history of ischaemic stroke who developed delusional beliefs.

Results: The two-factor diagnostic model has shown promising applicability in the case analysed, however, guidance is provided on the need to expand and change some of the criteria with the possibility of complementing the model with elements from neurobiological models.

Conclusions: The case analysis allowed us to point out the strengths and weaknesses of the diagnostic model discussed. We propose that strictly neurobiological diagnostic categories should be replaced by those that allow the diagnosis of cognitive deficits. We also hypothesise that problems with inference, as one of the main factors contributing to delusions, should not be analysed only in terms of the damage to the right hemisphere.

delusions / delusional models / diagnostic clues / case study

This article analyses the usefulness of several models of delusions described in the literature in the diagnostic process of a patient with a history of ischaemic stroke affecting the right hemisphere. Previous studies hypothesised on the process of delusional content formation and verified the delusional contents using available diagnostic procedures. Creating models to explain how certain disorders develop has two main advantages: it shows how a specific disorder forms and indicates the main types of factors involved

in this process. Thus, it enables a better understanding of the essence of the disorder and helps formulate diagnostic guidelines.

Models of this type can have various forms: from a simple presentation of a causal relationship between a factor and a disorder to complex, multifactorial network models, in which each element can potentially interact with the other. The task of theoretical models of delusions will be to indicate the main factors necessary for the emergence of delusional beliefs and to describe the dynamics of the process leading from these factors to delusion formation. The need to create such models primarily stems from a strong internal differentiation within the category of beliefs determined as delusions. Due to this heterogeneity, it is diagnostically interesting to identify and differentiate such factors depending on the contents of delusions.

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This paper includes a critical evaluation of theoretical models for the diagnosis of delusions and indicates practical challenges to which such models are exposed.

METHOD

Model

The two-factor model created by M. Coltheart and R. Langdon [1] is the most popular and widely discussed in the literature theoretical model for delusions. As a result of our case study analysis, a need has been identified to extend the model by adding elements that allow the diagnosis of delusions of reference and delusions of a persecutory type. In addition, we show how certain dysfunctions of the inference process can be complementary to the model.

Case study

We conducted a clinical examination of a patient with a history of right hemisphere ischaemic stroke who had been treated on a rehabilitation ward a month post-stroke. Patient K was aged 70, was of higher education and married. He was neat and quiet. He was mobile, but his speech was sometimes incomprehensible. He showed characteristics of left-central facial paresis and left upper limb paresis. His neurological reflexes were described as symmetrical, with a negative Babinski reflex.

The patient was treated due to ischaemic heart disease – stable angina pectoris – and right hip osteoarthritis (the patient had had metallic fusion implanted as a result of an acetabular fracture 20 years earlier). In addition, lumbosacral spine deformation was found. The patient was not receiving psychiatric treatment.

Brain computed tomography was performed twice – immediately after the admission and two days later. The first scan was performed without an intravenous contrast medium, layers: 1.25, 2.5 and 5.0 mm. It showed no signs of intracranial haemorrhage or focal changes in the brain. However, slightly increased radiodensity of the right middle cerebral artery was observed,

which could suggest an early stage of occlusion. No dislocation within the central structures was observed. The ventricular system was defined as symmetrical but slightly widened throughout. Widened convexity furrows and fissures related to atrophy and calcification of internal carotid and vertebral arteries were observed.

The second examination, with an intravenous contrast medium, provided details of changes observed. In the deep structures of the right hemisphere of the brain (putamen, corona radiata, head of the caudate nucleus) a streaky hypodense area, 24x9 mm in size and with attenuation coefficient at approximately 22 jH, was observed and defined as an ischaemic change. An ultrasound of carotid and vertebral arteries (Doppler) revealed that the common, internal, external and vertebral arteries have slightly thickened, sclerotic walls. The upward flow was devoid of haemodynamically significant disturbances.

The patient remained calm and polite when in contact with medical staff. He responded adequately to the contents and emotional loading of messages. He interacted with other patients, remaining popular due to his amiable personality (nice, cheerful, with a good sense of humour).

During the semi-structured interview the presence of productive symptoms was initially excluded, but further discussions with the patient revealed some contents that could be considered to be of delusional nature.

In particular, the patient believed that people sometimes insinuated things about him and sent him ambiguous messages. He determined that he did not often think about that, expressing confidence that there was no truth to what the others had imagined. He stated that this was not a source of anxiety or distress for him. Going further, he indicated that he felt that some people were not as they had seemed to be. He was convinced this premonition was absolutely true. These thoughts did not dominate his thinking, nor did he consider them worrisome. The patient also admitted to thinking that others were plotting behind his back. He believed that his sister-in-law, with whom he said his family had fallen out over money, is responsible for his illness. This belief accompanied him all the time. He considered it to be absolutely true, but also very disturbing. Recently, he became convinced that

people could communicate telepathically. This was not disturbing for him, nor did it dominate his thoughts. Patient K believed he had been fed those ideas by his son. He told of how his son had wanted to call on an exorcist to “heal” his aunt, but she did not consent. The patient did not consider himself to be particularly close to God, but during his stay in hospital he began to read the Bible, for “general knowledge”.

The patient’s beliefs, when determined as delusional, can be termed delusions of reference and delusions of control. It should be noted that they are systematised, forming a compact mental construct, possible to occur, and therefore can be described as paranoid. Of course, at this stage, we cannot determine the extent to which these beliefs are due to stroke and to what extent they are part of a well-established, pre-stroke belief system of patient K. It is also difficult to unambiguously determine whether these beliefs should be considered delusional, seeing that the patient does not perceive most of them as disturbing and they are not persistent mental rumination. On the other hand, they seem to be stable and, at least partially, affect the patient’s daily activities.

During a neuropsychological examination the patient achieved generally good results in the area of cognitive functioning, except for discrete characteristics of attentional functions disorders – concentration, attention-switching and selectivity of attention. It is worth noting that pre-stroke the patient was taking part in a cognitive training programme for people over 60 years of age. During his stay on the ward he participated in any workshop offered to patients, including a research programme taking place at the time. He arrived ahead of the scheduled appointment time to all his sessions with a speech therapist and a psychologist. Furthermore, staff observed that the patient read, did complex crosswords and played Scrabble. As he was often unable to find a partner to play with, he played alone, standing in for two players. Later he found a patient who played with him a few rounds, and when that patient left the hospital, patient K began asking the lead psychologist to with him, turned out to be an extremely skilled player.

Case analysis using the two-factor delusions model

Describing delusional disorder, it is necessary to answer two questions: where the delusion comes from, i.e. what factors determine the contents of delusion (the question about the aetiological factors) and why the patient does not reject the delusional contents, even though they are often bizarre beliefs that are universally accepted as false (the question about the supporting factors).

Specific neuropsychological deficits, such as “mirror agnosia” (confusing the image of the object with the object itself when looking in the mirror), which is a type of visual agnosia combined with damage to the right parietal region of the brain, can be a factor which determines the occurrence of delusion, for example in Alzheimer’s dementia [3]. Neuropsychological deficit cannot be a sufficient factor for the occurrence of delusion. If it were, all patients with “mirror agnosia” would have to present delusional beliefs regarding erroneous self-identification in the mirror (i.e. believe that the person they see in the mirror is not themselves). However, this does not happen; for example, in a study conducted by Binkofski et al. [4] none of the five examined patients with mirror agnosia show related delusional thoughts. In this context it seems reasonable to consider a factor that would explain this transition – from a neuropsychological deficit to a fixed belief. Therefore, we would be looking for additional circumstances that cause a patient to be convinced that the reflection in the mirror is not himself. It could be similar to another neuropsychological deficit called prosopagnosia – face blindness. This disorder itself cannot be a sufficient cause of delusional beliefs in which the patient dissociates from his mirror image, but it is necessary for a delusion to occur. Somatoparaphrenia, in which the patient denies that a part of his body belongs to him, is another interesting disorder. Also in this case the deficit itself does not necessitate the occurrence of delusional beliefs. In patients with this disorder the structure of the left hemisphere remains mostly intact, in contrast to patients with left-sided hemiparesis, who usually have damage in the right side of the brain. Is the imposition of images characteristic of the two disorders, but corresponding to different deficits, resulting in a delusional belief that the patient’s hand is not in fact his? The studies appear to give an affirma-

tive answer [5]. Thus, the concept adopted in the interpretation of delusional disorders in somatoparaphrenia seems to be as follows: delusional contents seem to be associated with the impossibility of any free movement of the limb – and, in fact, we cannot move the limbs we believe are not our limbs. The patient does not reject this belief, because brain damage in the right hemisphere makes it impossible. This causes the patient to adopt and maintain delusional beliefs. The damage is located in a region that, in a properly functioning brain, is responsible for shaping beliefs in accordance with the laws of probability and evidence “for” and “against”. Thus, the concept assumes that there is a region located in the right hemisphere of the brain whose damage results in “disinhibition” of the rejection of beliefs that in the absence of the damage would not be accepted as true. An overview of specific deficits that do not lead perforce to the occurrence of delusional beliefs is included in work by McKay [6].

In conclusion, we put forward a hypothesis that finding the answers to the two questions about the etiological and supporting factors of delusions fill up the description of the delusional disorder in the patient. The answers to these questions should identify two deficits:

- deficit A: a neuropsychological deficit, which may be associated in a reliable way with the delusional contents presented by the patient
- deficit B: a structural damage located in the right hemisphere of the brain.

Creating a model based on these principles can proceed in four steps, which may be used as a diagnostic heuristics [7]:

- step 1: identify a neuropsychological deficit, which may explain the existence of anomalous sensations in a patient
- step 2: show that the delusional belief is the result of inference to the best explanation on the basis of anomalous data
- step 3: show that the observed anomalous data do not cause the formation of specific beliefs themselves; the easiest way is to compare the delusional patient with patients with a similar neuropsychological deficit who do not show delusional beliefs
- step 4: identify the deficit associated with the functioning of the right hemisphere.

These steps are further hypotheses that were subjected to verification within the frames of the current diagnostic situation.

At first glance, the case of patient K appears to be a good illustration of the assumptions of the model. Therefore, there is a neuropsychological deficit for which we can consider attentional functions disorders, isolated from the general good or very good cognitive functioning of the patient (step 1). The damage in the right hemisphere is also identifiable (step 4), but the difficulty concerns indicating the location, which will transform to type B deficit. Empirical research is lacking in this area, relating to both the premier and the subcortical location in the aetiology of delusions. Attentional functions disorders may lead to erroneous assignment of interest in the patient by other people (step 2).

In patient K both the widening of convexity furrows, especially on the right side (e.g. McKay et al. [7]; Staff et al. [8]), and damage to subcortical structures (e.g. Box et al. [9]) may be significant in the formation and maintaining of delusional contents. In a simple way, we are also able to identify patients who do not develop delusional beliefs having type A deficits similar to patient K (step 3). Thus, it can be concluded that in the context of patient K the two-factor model is only partially diagnostically useful. We will attempt to indicate some of the disadvantages of accepting the model's assumptions as true.

One disadvantage of the model is the possibility of not being able to extract any of the above deficits, because both may have a strong neuropsychological basis. The authors of the model cope with this kind of argument by proposing to extend it from two deficits to two factors, indicating that the contents of delusions or their maintaining factors do not always need to occur due to neuropsychological and structural deficits. They allow for a situation in which other mechanisms can take over the functions of the deficits, at the same time indicating the necessity of simultaneous occurrence of two elements. For example, in delusional disorders comorbid with alcohol disorders, such as pathological jealousy, which may indicate the Othello syndrome, the neuropsychological mechanism seems to be still unconfirmed. Of course, the traditional recognition of delusional contents as functional does not negate the usefulness of the neuropsychological

factor in explaining their components. Similarly, delusions characterised as typically organic do not exclude the importance of motivational factors in explaining their substantial contents [10]. It should also be noted that the presented models show the way of delusion forming within the general model of belief formation. Within the models trying only to explain the origin of the delusion, or those that attempt to explain the development of multi-thematic delusions, a two-factor structure may be unnecessary [10]. It seems that this is a wide-ranging problem and that a thorough analysis of multi-thematic delusional beliefs should be carried out at the neurobiological level [11, 12].

Another issue is the usefulness of identifying various deficits in explaining the delusional contents presented by patient K. It seems more useful in the context of the so-called monothematic delusions rather than for the usually persecutory delusions comorbid with disorders which are the effect of real harm experienced by the patient. In this case, we find no neuropsychological factors as such, and unless we are able to prove that the expectation of social risk may be associated with deregulation of the neural network connecting the amygdala with the prefrontal circuits, we should ask, what is the primary cause of delusions: communication disorders between these regions or experience of harm? [13].

The case of patient K raises fundamental doubts about the duration of identified defects, but above all, about pre-stroke functioning of the patient. An unambiguous classification of delusional disorders which occurred in the patient also causes difficulties. On the one hand, the spoken contents are focused on one topic, which can be considered as a wrong interpretation of the behaviour of one and the same person. On the other hand, the theme of patient K's delusions clearly fits into the category of persecutory delusions. Additionally, there are problems noted in the literature with applying the two-factor model to persecutory delusions and delusions of control. First of all, precisely determining which cognitive systems are mainly involved in the development of anomalous data (memory, improper reference to I, improper validation of objects and events, attentional disorder, etc.) is problematic [12, 13]. On the other hand, finding a basis for the recognition of different data

within the persecutory delusions (encompassed as multi-thematic) is also difficult [13]. As a partial remedy, the authors of the two-factor model proposed introducing psychological elements to the image of type A deficit, for example, by extending it, within the persecutory delusions, to include wrong motivational processes which determine the occurrence of, at least, social anxiety [13]. Such a broad approach, which is also used in this work, seems to be particularly useful in the context of explaining delusions associated with other people's actions or the patient's interactions with the environment.

In conclusion, it should be noted that the ability to identify specific structural deficits, which the two-factor model indicates, is its unquestionable advantage. A particular issue is the possibility of combining two separate, seemingly independent, deficits in explaining the development of contents that (in principle) remain monothematic persecutory contents. The process of explaining the transition from a structural deficit through a neuropsychological deficit to the delusions of this type does not seem, in the light of the two-factor model, sufficiently clear. This results in a situation where having a lot of data, we still face the problem of a "black box" in which we cannot point to the mechanisms of how delusional contents are formed and sustained by the patient. Hypotheses on how to fill this gap will be presented in the next section of the paper. Alternative models of delusions and their usefulness in the current case study.

MODEL OF THE LEFT HEMISPHERE RELEASE

The idea underlying the development of the second diagnostic model of delusions was to improve Coltheart's model. The authors, C. Braun and S. Suffren [14] gathered factors which determined the need to enrich the two-factor model. Firstly, the majority of diagnosed delusions are not of monothematic nature. This is justified by the statistical data. Secondly, based on literature analysis they note that in delusional disorders it is difficult to find a deficit that could perform an initiating function in the sense adopted by Coltheart. Thirdly, the general deficit regarding monitoring our own beliefs associated with the damage to the frontal lobe does not oc-

cur in delusional disorders. Finally, the formation of additional cognitive contents, especially of the higher order, cannot be easily correlated with brain damage. The last argument is mainly supported by case analysis of delusions induced by psychoactive substances or the occurrence of delusions associated with hypermetabolism or hyperperfusion of certain parts of the brain [14, 15].

In addition to the problems associated with the two-factor model, Braun and Suffren decided to focus on several issues that should be modelled, regarding them as key issues directly related to the mechanism of delusion formation. First of all, any adequate concept explaining the formation of delusions should focus on the problems with inference (i.e. how the delusion, which is a wrong belief, is formed), and secondly, excessive tendency to draw conclusions, especially inference frenzy. Thirdly, the model should include the explanation of unjustified reference to "I", as one of the elements of the formation of persecutory delusions (or generally, delusions of reference). Relying on the aspect related to inference is, in Braun and Suffren's opinion, the first element of the new model. Disorders of inference are explained by the second element, which is the release of the left hemisphere, i.e. the imbalance of hemispheric specialisation as a result of damage to the right hemisphere. This approach is consistent with the assumption that brain damage cannot be a direct cause of delusional contents, as evidenced by the possibility of inducing delusions by using psychoactive substances and by the effectiveness of antipsychotics in the treatment of delusional syndromes [16]. Laterality disorders mentioned by the authors of the model fit, to some extent, in the concept of left-hemisphere interpreter [17, 18]. Regarding the concept of hemispheric rivalry, Braun and Suffren characterise the left hemisphere as an activator of mental activities, and the right as their inhibitor. This is supported by the fact that the majority of psychiatric symptoms related to the damage to the right hemisphere, are positive (productive). Once again, the authors use statistical data. What is more, if you add to this that the left hemisphere is responsible for generating inferences and referring to "I", which seems to be confirmed by the examination of patients who underwent commis-

surotomy, the result is an image of laterality disorders consistent with the first of the factors in the described model. This concept can serve as a general model of delusion formation, as it is not "internally" limited to one type of delusions. However, the fact that the review and analysis of the literature allowed the authors to form only a moderate confirmation of their model seems to be problematic.

NEUROBIOLOGICAL MODEL

Finally, a strictly neurobiological model directly related to inference on the basis of beliefs that the patient already possesses and inference errors, is proposed in the literature [11]. In this model the authors point to the biological basis of predicting inference errors and hypothesise that deficits in the operations of those systems lead to forming contents that perfectly explain delusional beliefs that one already possesses. Additionally, this process can be modelled using Bayesian inference. The strength of this model is also its disadvantage. At the research level of standard battery tests concerning delusions, its authentication or invalidation is impossible. On the other hand, this model is a refinement of the models discussed earlier, with a greater emphasis on the inference process, simultaneously highlighting problems in the computational aspect of inference, more than the in physical changes (lesions) in normal brain tissue. This model, due to the incorporation of Bayesian methods of inferential process modelling (which consists of, among other things, inference from sensory data to beliefs), has a very high ability to model existing delusional contents, at the same time being a weak predictive tool regarding such contents. This model can be seen as a refinement of models described previously at a neurobiological level. In a situation where other models refer to a deficit, the authors of the neurobiological model refer to specific changes in neurotransmission which result in disturbance of the correct inference process. Further implications of the model and its relationship with psychometry (and the texts used where there are delusional contents) open a very interesting field of research.

Within the framework of conducted deliberations, the neurobiological model, as the most de-

tailed, will not be useful en bloc. This is because it would require, above all, a thorough examination of the patient in the field of neurotransmission and, at the same time, validation of the entire model. However, it is worth noting that if we accept the general assumptions of the model, without reference to neurobiological mechanisms, it will complement the two-factor models.

CONCLUSIONS

Do the diagnostic models discussed here have clinical importance? The final answer is complex. At the point of diagnosis of a deficit, we can use a diagnostic model in the framework of an outlined diagnostic procedure, which will indicate when the occurrence of delusional beliefs can be expected or when the diagnosis of delusions will be the most accurate (damage to the right hemisphere etc.). The situation becomes complicated when, as in the present case study, the nature of the deficits will force on diagnosticians the inclusion of non-neurological anomalous data (e.g. impaired motivational processes) into the diagnostic process. For this reason, it seems that the proposed model should be enriched. The authors do not advocate a new approach, however, they indicate some diagnostic difficulties related to a fluent withdrawal from monothematic delusions, treated paradigmatically by Coltheart & Langdon [2] (e.g. the Fregoli delusion), toward multi-thematic disorders, or at least those in which input data are not homogenous (as in the commented case). Another issue worth underlining is the lack of clear interpretation of the inference process in the specific model. The authors of the paper assume that the inference process will be perceived as impaired in the event of right hemisphere lesions. More detailed models of inference within the frames of delusion formation can be found in the literature [6, 19]. It seems that the existence of any circumstances which may affect the evaluation of the data in the course of belief formation should be a diagnostic clue. In the case of patient K it can be inferred on the basis of identified disorders of attentional functions, which may result in including random disconnected information into the patient's cognitive system [20] and which may lead to the prolongation of

the process of assigning meaning to certain pieces of information while recollecting and thus – to inference errors. Additionally, in this case, including so-called delusions of reference to I, which relates to some concepts of persecutory delusions and delusions of control, can be important [10]. It seems that this deficit is most fully explained by the self-monitoring concept proposed by Frith [21], which allows to distinguish one's own actions or thoughts from those produced by other factors in such a way that one central process monitors the differences between predicted and observed consequences of actions or mental processes. In a situation when there is no difference, action or thought is classified as produced by the self. In such patients, this process may be impaired, the consequence of which is inappropriate assignment of the origin of actions or thoughts [22].

These deliberations prompted us to a moderately positive response to the question about the clinical importance of diagnostic models. The case analysis presented here shows that the diagnostician usually does not have to deal with the idealised situation described in the models. At the same time, these models impose a certain structure to the process of collecting patient information, which otherwise could be chaotic, and allow directing it when the symptoms are not identifiable during a general interview.

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