CONVERSION DISORDER - NEUROIMAGING

DIANA GEORGIANA COTUNĂ[1]*, IULIA SANDU[1], ANA-MARIA CIUBOTARIU[1], FLORIN CODĂU[1], MĂDĂLINA PISTEA[1], ADELINA ȘERBAN[1], EMANUELA ANDREI[2]

[1]Resident doctor in Child and Adolescent Psychiatry "Prof. Dr. Alex. Obregia" Psychiatry Hospital, Bucharest, Romania

[2]Assistant Professor, Child and Adolescent Psychiatry Department, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Corresponding author email: cotuna.diana@yahoo.com

ABSTRACT

The diagnosis of conversion disorder can be a real challenge, the whole process may encounter many difficulties with a high risk of delay, largely relying on exclusion methods. There is a wide experience in adult patients that is not extrapolated to infants in the pediatric field. According to some recent studies, the prognosis and evolution are better in children, but this can often change when other variables are included such as comorbidities, late diagnosis and the stigma around the topic. This review discusses conversion disorder and the neuroimaging approach of the pathology which can lead us to new ways of diagnosis and a new understanding of the mechanisms behind the clinical manifestations.

Key words: conversion disorder, neuroimaging, fMRI, SPECT, PET.

INTRODUCTION

We found in the latest Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [1] that "conversion disorder (CD) is defined as (1) having at least one symptom of altered voluntary motor or sensory function, (2) the presence of clinical findings supporting incompatibility between symptom and neurological or medical conditions, (3) the symptom is not better explained by another medical or mental disorder, and (4) causes clinically significant distress or impairment."

Because of the issues regarding the way cases are defined, prevalence rates can be very varied [2].

Nevertheless, conversion disorder is not considered to be so uncommon (1% over to 3% of people in the general population). We found out recently, in a prospective cohort study that over 5% of all patients have conversion disorder [3].

"*Hysteria*"? What we know by now

The interest in what we known as *Hysteria* has appeared in the earliest days of antiquity, with the origins being located in the Egyptian theory of the *wandering uterus*, while some have argued that the term itself should be credited to Hippocrates [4]. In more modern times, Briquet talked about a so called *personnalité hysteriqué* and Charcot had even attempted to isolate and explore the disorder through its clinical signs. The famous neurologist from France used techniques like hypnosis that were, at the time, regarded as being a part of scientific methods. In the 20th century, Freud [6] suggested that the origins of the condition lie in an unconscious conflict that is symbolically converted into a somatic symptom.

Over the following decades, this condition came to be categorised as a form of dissociative disorder. The term "hysteria"

is currently considered to be offensive. However, in DSM-IV-TR (1994), under the name conversion disorder, the condition was first considered to be part of somatoform disorders in which you could have also found the Briquet's syndrome as well as other forms of somatizatin [7].

The symptoms in conversion disorder can look like any neurological manifestations. Diagnosis can be made based on discovered variabilities or inconsistencies during examination. Conversion disorder patients can have a paradoxical lack of interest or "lack of care" about their neurological problems - a peculiar *belle indifference*. Indeed, it is quite common for any neurological investigations to be without a pathological meaning [8].

Do we have someone to blame?

Despite the historical relevance of the disorder (being referred to as *hysteria)*, the current knowledge on etiology and neurological background of conversion disorder is unclear. Like other psychiatric disorders, an interaction of genetic patterns, neurobiological and psychosocial factors is highly possible. Epidemiological studies done on twins showed that approximately half of the cases could be explained by genetics [9]. There are some dissociative symptoms reported as a side effect of pharmacological treatment and associated with endocrine diseases [10], which can be a clue to neurobiological influences. Besides that, psychosocial influences are also known to be a possible cause of dissociative disorders and, as we know, conversion disorder is somehow related to dissociative disorders (according to ICD-10). We need to consider the importance of some emotional deficits as well. Alexithymia, which is defined as the inability of feeling emotions, is also found in many conversion disorder patients and dissociative patients as well. There are rising studies proving that trauma can lead to dissociative disorders, especially to depersonalization and derealisation. These two conditions are considered to appear as a response to a traumatic event or in patients with PTSD (post-traumatic stress disorder) [12]. For functional manifestations of conversion disorder, Perez and co [13] found some distinct patterns in brain activity: "(1) disrupted inhibitory abilities with dysfunction in primary somatosensory and motor cortex (2) modifications of the voluntary-intentional capacities with dysfunction in prefrontal areas [14] (3) impaired attention based on dysfunctional anterior cingulate cortex, parietal associative cortex, striatum, thalamus (4) misconceptions of action authorship as a result of dysfunction in the temporoparietal junction, somatosensory cortex, anterior cingulate cortex, parietal associative cortex, and gyrus temporalis superior [15] (5) as well as affective disorders due to dysfunction in the amygdala and anterior cingulate cortex [16]." Changes in cortical thickness and other structural alterations, have recently been discussed to be related to conversion disorder. These alterations have been found in the cerebellum and in some cortex areas like the premotor and primary motor cortex and other specific areas of the brain [17].

Why neuroimaging?

The main research that neuroimaging has made so far is like a new open gate in psychopathology. The research is concentrated into finding a model for conversion disorder, an accurate anatomical and functional pattern. In this review we will discuss, at first about the Functional magnetic resonance imaging (fMRI) and then about some studies made using positron emission tomography (PET) and in the end we will see some results made by single photon emission computed tomography (SPECT) as well. These investigations are making great progress in the research field. Anyway, the studies that we will discuss are quite limited. First of all you will see that it is difficult to compare the symptoms of conversion disorder and the process

behind it because of the huge variability in manifestations and comorbidities. Second of all there is a problem about the number of patients, the studies are quite restricted in this regard. By putting it in perspective, we can say that research is only in its beginning and is expecting to find a pattern for the presumed neurobiology malfunction behind conversion disorders symptoms [18].

Investigations targeting the function of the brain are the most promising and it seams that these investigations can help us in the diagnostic process. Often people with CD (conversion disorder) can not explain us exactly what they feel – in other words, they have a dissociate experience, their brain can not process informations in a relevant way, so we can not count on self-raport. In addition, symptoms seem to occur in an acute way, in response to psychological factors, like stress. The manifestations of conversion disorder tend to resolve spontaneously, other the symptoms seems to disappear with medical treatment. That shows that there is not a static or a structural change in the brain, in fact it can be a dynamic and functional problem [19]. In conclusion, we have the hypothesis for a pattern in which all the possible factors (biological, psychological and social) are involved. There are a lot of debates about a type of conversion disorder which is induced by stress. We know trauma has been associated with many dissociative disorders, even Freud described a connection between trauma and cases of hysteria, but, like other variable risk factors, childhood trauma or suggestibility did not have a consistent experimental support [20, 21]. First of all, the investigations that are needed are complex. Second of all, the whole process was slowed down because of the historical stigma, and also the possibility of someone feigning it [22]. As a result, there has been an encouraging drive in using more functional methods that investigate the brain activity and make the difference between a real disorder and feigning, much more efficient than reporting on a self-experience representation. That way we can find a correlation between the arias in the brain involved in processing emotions with these very well-known arias which are responsible in sensorimotor mechanisms and also cognitive processing. Hopefully, with this new perspective, we will find an elaborate model, for how a large range of different symptoms come from distressing emotions, in other words, how the "conversion" happens.

The conclusion, all this neuroimaging studies, especially which had used functional investigations, have the same mission, to indicate where the damage was done and exactly which neural circuits are affected [23]. There are recent studies which are focused on functional brain imaging techniques trying to locate specific damaged circuits in correlation with CD manifestations. We will present some of them in this review.

Methodology

We effectuated a literature research in scientific field using Google Scholar and PubMed. The search keywords were: *"conversion disorder"* AND *"neuroimaging"* OR *"f MRI" OR "PET" OR "SPECT"*. We limited our review only on studies written in English language. Therefore, we collected studies which used Functional Magnetic Resonance Imaging and Single-Photon Emission Computed Tomography (tree studies each) and other two studies which used Positron Emission Tomography. We made a detailed presentation of the studies in the following section.

Functional magnetic resonance imaging (fMRI)

Functional magnetic resonance imaging (fMRI) is not considered to be an invasive procedure and its availability is daily increasing. This procedure can shed light on the biological causes and reveal the mechanisms

behind the symptoms of conversion disorder [24]. We will discuss three studies which used fMRI (Table 1) in the following part of our review.

I. Stone et al published a study in 2007 with four patients with conversion disorder. The subjects presented with unilateral ankle weakness. The fMRI was performed on the patients and on the healthy controls simulating the disfunction [25]. The test requested for all the subjects to flex their affected ankle and then the other ankle as well. The study reported: "1) less intense and more diffuse activation of motor cortices contralateral to the weak limb in patients with conversion disorder and in controls simulating weakness than contralateral to the normally moving limb; 2) activation of regions of the basal ganglia, insula, lingual gyrus, and inferior frontal cortex in CD patients, but not in controls when moving weak right ankle compared with moving the normal left ankle. Increased activation of inferior frontal cortex was interpreted in the context of an executive role in conscious planning or preparation of the movement, and activation of the insula, lingual gyrus, superior parietal region, and precuneus as reflecting increased attention and effortful emotional processing in CD patients; 3) Relative hypoactivation of the right middle frontal gyrus and orbito-frontal cortex in CD patients on attempted movement of the weak ankle which the authors suggest as a possible a mechanism through which the movement is inhibited; 4) Activation of contralateral SMA in feigning controls when moving the weak ankle compared with moving the normal ankle, which has been suggested as an excess of movement planning activity in comparison to movement of the normal limb."

II. A study was performed in 2011 by van Beilen et al using Functional Magnetic Resonance Imaging and it had three sets of participants. First of all they used a group of control with persons which were considered to be healthy, then they investigated an other group of persons with unilateral functional paresis considered to be a symptom of CD. In the end there was another healthy subjects group investigated as well, but this time they were asked to feign the unilateral paresis [26]. At first all participants were put to imagine the motion and then to execute the actual act of motion. The feigners and the healthy group had almost the same model of neuronal activation. An increase activation was detected in the motor cortex (primary motor and secondary motor cortex as well), but a decrease activation in SMA (supplementary motor area) on the affected side (the side with the paresis) in contrast to the unaffected side. Besides, they found a decrease in activity in several prefrontal areas and a significantly increased activity in the contralateral primary motor cortex. Another unexpected observation was an activation on the cingulate cortex in patients with conversion disorder in contrast with normal participants. Moreover, the right TPJ (temporoparietal junction) -supramarginal gyrus- and DLPFC (dorsolateral prefrontal cortex) manifested a decrease in activation on patients comparing to feigners, but this was not significant compared to normal controls.

III. We found a very recent study (published in 2020), conducted by Longarzo et al. [27]. They studied a complex case of a woman presenting with motor rigidity in the lower limbs and also a sensory dysfunction, vertigo and postural instability. There was scans performed when the patient was moving and resting, after that the results were compared with eleven healthy participants. fMRI and other functional investigations have been performed to identify brain paths and new connections between areas of interest. The results found a significant a lack of connectivity between areas of the brain, the temporal gyrus and parahippocampal gyrus (PHC) in particular. There were no other structural changes.

Table 1 - studies using functional magnetic resonance imaging

Study	Participants	Manifestations	Conclusions
Stone et al.	4 CD 4 HC (Feigning)	Motor (ankle weakness)	- hyperactivity in the basal ganglia, the insular cortex, lingual gyrus and inferior frontal cortex in patients with conversion disorder; - activation of the right middle frontal gyrus and OFC in patients with conversion disorder.
van Beilen et al.	10 CD 21 HC 13 Feigners	Motor (Paresis)	- decreased activity in multiple PF areas and increased activity in the contralateral primary motor cortex in CD patients; - hyperactivity in the left cingulate cortex activation in patients with conversion disorder symptoms; - a lack of activity in right temporoparietal junction and dorsolateral prefrontal cortex in CD vs feigners.
Longarzo et al.	1 CD 11 HC	Motor (Rigidity)	- a lack of connectivity between the PHC cortex and STG.

CD = conversion disorder; HC = healty control; OFC = orbitofrontal cortex; PF = prefrontal; PHC = parahippocampal cortex; STH = superior temporal gyrus

Single photon emission computed tomography (SPECT)

SPECT is a nuclear imaging technique which is using computed tomography and a radioactive tracer to determine regional cerebral blood flow (rCBF). The procedure operate by measuring the distribution of a the radioactive tracer in the brain tissue [28]. We investigated three studies using single photon emission computed tomography (Table 2) on conversion disorder patients.

I. Tiihonen et al published one of the first study using SPECT. They performed a study on a single patient accusing conversion disorder symptoms (left unilateral sensory loss) [29]. They stimulate the affected side y using electrical impulses. The investigation showed a decrease in perfusion and also activity in the right somatosensory parietal cortex and an increase in perfusion (perhaps suggesting hyperfunction) on the right frontal areas.

II. Yezici et al [30] used SPECT in another early study (performed in 1998). They measured the regional cerebral blood flow in five conversion disorder patients. The study proved there was a drop in rCBF on the right temporal lobe in one of the five patients. Still, there was a drop in rCBF on the temporal lobe also (this time on the left side of the brain), found in all conversion disorder participating patients.

III. Vuilleumier and his team performed a study in 2001 [31] using SPECT. The study was following seven conversion disorder patients. All the patients were complaining about unilateral motor deprivation. Some of them had a unilateral sensory deprivation as well. They scanned a set of patients once their symptoms had disappeared. This approach allowed them to act as their own controls. This study used a special method to induced a stimulation in the affected part of the body. In order to obtain activation in the motor and also sensory cortical areas they used vibration. There was a hypoactivation of the thalamus found and an increase of activation after the symptoms were resoluted. Also they found the same effect in the basal ganglia contralateral to the affected limb. That proved them the connection between functional symptoms and a disruption in a somatosensory pathway, such as cortico-striato-thalamo-cortical loop.

Table 2 - studies using single-photon emission computed tomography

Study	Participants	Manifestations	Conclusions
Tiihonen et al.	1 CD	Sensory	- hypoperfusion of the right parietal cortex and simultaneous hyperperfusion of the right frontal lobe areas.
Yezici et al.	5 CD	Motor	- reduced regional cerebral blood flow in the dominant temporal lobe in all cases and right temporal lobe in one case.
Vuilleumier et al.	7 CD	Mixed (unilateral sensorimotor loss)	- hypoactivation of the thalamus and basal ganglia contralateral to the affected limb, which resolution after symptoms disappeared.

Positron emission tomography (PET)

Positron emission tomography (PET) is a type of investigation used in nuclear medicine. PET measure metabolic activity of the cells and target tissues of the human body [32]. We will detail two studies of conversion disorder that used Positron Emission Tomography (Table 3) in the following section.

I. Marshall et al conducted a study in 1997 on a single patient with left hemiparesis. The patient was diagnosed with conversion disorder two and a half years before the study was published [33]. They performed the measurements while the patient was asked to move the unaffected leg. Using PET, they determined the regional cerebral blood flow (rCBF) and found lower blood flow in some important arias of the brain. It was not a surprise to discover that even the plan of moving the unaffected right leg increased regional cerebral blood flow in the same brain arias. In contrast, the plan of moving the left leg and the actual performed act do not raise cerebral blood flow in the arias we discussed, but the areas in cerebellum was activated. Under the same conditions and in the same time, rCBF increased in two important areas of the brain. The authors pointed out that these two areas (right orbitofrontal cortex and right anterior cingulate cortex) are very important in suppression of functional manifestations, like inappropriate motor responses.

II. In 2000 Spence et al [14] conducted a study in order to find the distinction between feigned and real dissociative manifestations using Positron emission tomography (PET). They studied two cases of conversion disorder with patients accusing paresis on their left arm. One of them had had the symptoms for ten months and the other for twelve months. They also investigated an other conversion disorder patient with paresis on the right arm. This one had had the symptoms for six months. They measured the regional cerebral blood flow (rCBF) in all patients and also in six healthy subjects and four subjects simulating parasis. The subjects were asked to move their affected limb during the measurements. They found in the left dorsolateral prefrontal cortex (lDLPFC) a reduce rCBF in CD subjects in contrast with the other groups of subjects. Then, they investigate rCBF in the rAPFC (right anterior prefrontal cortex). In contrast, they found that feigners had a decreased rCBF comparing with the other two groups. We know that left dorsolateral prefrontal cortex (DLPFC) has been found to be involved in abstract reasoning and other cognitive processes, that been said a malfunction in this area can be interpreted as affected emotional reactivity.

Table 3 - studies using positron emission tomography

Study	Participants	Manifestations	Conclusions
Marshall	1 CD	Motor (weakness)	- increased activity in the rOFC and rACC;
Spence	3 CD 4 feigners 6 HC	Motor (weakness)	- lower activation in lDLPFC in participants when they are trying to make a movement on their affected side.

CD = conversion disorder; HC = healthy control; rOFC = right orbitofrontal cortex; rACC = right Anterior cingulate Cortex; lDLPFC = left dorsolateral prefrontal cortex

DISCUSSION

All that we found in these studies seems to be very important in the whole image of conversion disorder. It is a big difference of how we see conversion disorder now, first of all because of the fact that the emotion processing is taking a big step forward. Studies shown that isn't just one specific dysfunction in some component of the brain involved. There seems to be a disorder which implies a whole network of brain areas and lead to conversion symptoms. In contrast to healthy control subjects, those with conversion disorder suffer from malfunctions in arias of the brain involved in intention, action planning, decision making and in production of autonomic responses such as motor cortices, basal ganglia, amygdala, insular cortex, thalamus, parietal lobe and temporal lobe. There are, however, some possible causes involved in CD production in the studies that we have discussed. First of all, when it comes to feigning, all studies agreed that there is a clear difference in comparison to conversion disorder [34]. What we know about conversion disorder seems to be changing completely.

Secondly, it should be possible to distinguish a possible mechanism for at least some symptoms. Reduced performance of motor areas or in specific sensory networks and an increased neuronal activation in cingulate cortex and in other prefrontal areas seems to be involved in symptoms production [35]. This has led to suggestions of a decline in motor planning, with repeated implication of the SMA (supplementary motor area) [16]. There is a discussion about a potential dysfunction of several areas of the brain involved in emotion production due to the deficiency in self-monitoring. We are talking about TPJ (temporoparietal junction), DLPFC (dorsolateral prefrontal cortex) [26] and the deprived connections with limbic areas. All of this suggests that there is a connection between emotions and dissociation mechanisms of development [36].

Thirdly, we can talk about a predisposition or sensitivity in some subjects. Some studies made on asymptomatic patients are suggesting some flows in the way they deal with their emotions [16, 24]. Despite the fact that the subjects may have been symptomatic when the scans were done, this pattern was not directly separated from the symptoms. In this case, we need to consider hypersensitivity to physical, emotional, or social stimuli as a important causal factor and also we need to agree that there is a link between this sensibility and a motor output and therefore to a possible conversion disorders manifestation [18].

Fourthly, and last, trauma has been shown to be processed in a different way in relation to symptoms in conversion disorder (very similar to what Freud believed in): the recall of some traumatic life events seems to be relevant for etiology. We are assuming that the recalls are connected with some functional changes in supplementary motor area and in temporoparietal junction. both being known to be involved in memory suppression after traumatic events. These two

areas of the brain can be plausible sites of symptom production in conversion disorder [16, 26].

CONCLUSIONS

Conversion disorder is widespread and despite being both controversial and theoretically interesting, surprisingly few empirical studies have been made in this area. This new and innovative studies using functional investigations on brain are proposing possible functional explanations in psychiatry disorders including conversion disorder. Studies that include much larger numbers of patients and involving more complex investigations are necessary.

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