# Conversion sensory symptoms associated with parietal lobe infarct: case report, diagnostic issues and brain mechanisms

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This case report suggests that diagnostic difficulties and brain mechanisms related to conversion disorder associated with cerebral lesions differ from those related to conversion disorder without cerebral lesions. A 35-year-old divorced woman was admitted to a psychiatric inpatient unit with multiple physical complaints. The symptoms first appeared 5 years previous and 2 months after a sexual assault. Three years later, she began to experience ill-defined sensory symptoms confined to the left half of her body (splitting the midline). Results of neurologic consultations were equivocal because of the subjective nature of the complaints, which were viewed as conversion symptoms. A magnetic resonance imaging scan demonstrated an old infarct in the right parietal lobe, suggesting a physical origin of the patient's symptoms. However, normal somatosensory-evoked responses from the affected area contributed little to establishing the diagnosis. The results of all further investigations to identify causes of the vascular pathology were negative. The multiple ill-defined somatic symptoms, the distribution of sensory symptoms and the resolution of symptoms with psychotherapy established the diagnosis of conversion disorder superimposed on a pre-existing right parietal lesion. This case highlights the importance of clinical features in establishing a diagnosis such as this. We suggest that reactivation of implicit sensory memories (represented at the thalamic level and resulting from decreased corticofugal inhibitions due to the lesion) may contribute to the formation of sensory conversion symptoms in individuals with parietal lobe lesions.

Ce rapport de cas indique que les difficultés diagnostiques et les mécanismes cérébraux reliés au trouble de conversion associé à des lésions cérébrales diffèrent de ceux reliés au trouble de conversion sans lésion cérébrale. Une femme divorcée de 35 ans admise dans un service interne de psychiatrie se plaignait de multiples problèmes physiques. Les symptômes ont fait leur apparition pour la première fois cinq ans auparavant, deux mois après une agression sexuelle. Trois ans plus tard, elle a commencé à ressentir des symptômes sensoriels mal définis confinés à la partie gauche du corps (franchissant la ligne médiane). Des consultations neurologiques ont donné des résultats équivoques à cause de la nature subjective des plaintes, considérées comme des symptômes de conversion. Une imagerie par résonance magnétique a démontré la présence d'un vieil infarctus du lobe pariétal droit, ce qui indique que les symptômes de la patiente étaient d'origine

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Medical subject headings: cerebral infarction; conversion disorder; parietal lobe; signs and symptoms.

*J Psychiatry Neurosci* 2002;27(2):118-22.

Submitted Feb. 8, 2001
Revised Jun. 15, 2001
Accepted Sept. 19, 2001

physique. Les réactions somatosensorielles normales provenant de la zone atteinte n'ont toutefois pas beaucoup aidé à établir le diagnostic. Les résultats de tous les autres examens visant à déterminer les causes de la pathologie vasculaire ont été négatifs. De multiples symptômes somatiques mal définis, la distribution des symptômes sensoriels et l'élimination des symptômes par la psychothérapie ont permis de diagnostiquer un trouble de conversion surimposé à une lésion pariétale droite préalable. Ce cas met en évidence l'importance des caractéristiques cliniques dans l'établissement d'un tel diagnostic. Nous sommes d'avis que la réactivation de souvenirs sensoriels implicites (représentés au niveau du thalamus et découlant d'une baisse des inhibitions corticofuges attribuables à la lésion) peut contribuer à l'apparition de symptômes de conversion sensorielle chez des personnes ayant subi une lésion du lobe pariétal.

## Introduction

The concept of hysteria has been questioned since antiquity because of the frequent association of physical disease and hysteria.[1–3] Slater[1] reported that 60% of his patients with hysterical symptoms had signs of physical illness, either initially or at follow-up, and Whitlock[3] found that 63.5% of patients with symptoms of hysterical disorder had accompanying cerebral disorder or a history of organic brain disease. Further, a significant proportion of patients with epileptic seizures may have concurrent nonepileptic or dissociative seizures.[4] Identifying the presence of a conversion symptom that resembles a physical symptom of coexisting organic pathology can be an extremely difficult clinical task[4] because current nosology fails to clearly distinguish conversion in association with a physical disorder from conversion without a physical disorder.[5] Investigations used to rule out physical causes when establishing conversion disorder not associated with physical disease may have limited value in the diagnosis of conversion symptoms that are associated with organic conditions. Further, brain mechanisms involved in the manifestation of conversion symptoms coexisting with an organic disorder may differ from those associated with symptoms and no physical disorder.

We describe the case of the rare association of conversion sensory symptoms with a parietal lobe infarct and discuss the diagnostic challenges in this case and the brain mechanisms that may be involved.

## Case report

A 35-year-old divorced woman who was a co-owner of a promotional clothing company was admitted to a psychiatric inpatient unit with multiple physical complaints. These symptoms first appeared 5 years previous, 2 months after she was allegedly raped by her former bisexual boyfriend who was described as an HIV-positive intravenous drug abuser. Since that time, she claimed to have developed abdominal rash, skin swelling, shrinking gums, facial pain, tunnel-like vision, speech motor incoordination and other symptoms involving chest, respiratory and musculoskeletal systems. The patient consulted several physicians who, after repeated physical examinations, could not establish any physical illness. Results of many relevant investigations, including a test for AIDS, were negative.

Three years after the onset of the physical symptoms, the patient began to experience sensory symptoms (i.e., numbness and tingling sensations), which started on both sides of her body but were later confined to the left side. These symptoms proceeded with an ill-defined uncharacteristic headache, which subsided, leaving sensory symptoms. The sensory symptoms, confined to the exact left half of the body (i.e., splitting the midline) involved the face, neck, trunk and limbs.

There was no indication of unipolar or bipolar illness, psychotic symptoms, cognitive decline or history of substance abuse. Although the patient expressed anxiety and depressed mood, these symptoms were specific to physical complaints, and there were no pervasive or severe depressive or anxiety symptoms that might cause one to consider coexisting clinical depressive or anxiety disorder. The patient's psychiatrist prescribed fluoxetine (20 mg/d) for the somatic symptoms and depressed mood, but with limited success.

The salient feature in the woman's psychosocial history was that she was brought up in a dysfunctional family. Her mother was described as being very manipulative, and her father was an authoritarian. Her father has seen several psychiatrists for an unspecified psychiatric condition, and her mother and younger brother were undergoing psychotherapy. A mental status examination on admission revealed preoccupation with somatic symptoms and depressed mood. There was no

evidence suggestive of somatic delusions, major depression or melancholic symptoms. The diagnostic impression on admission was somatization disorder with sensory conversion symptoms.

On the first neurologic consultation after admission, the sensory symptoms were considered to be conversion symptoms, on the basis of the distribution and nature of the symptoms (i.e., splitting the midline without any objective evidence of true sensory deficits). However, owing to the history of headache and preceding sensory symptoms, magnetic resonance imaging (MRI) was performed which demonstrated an anterior right parietal infarct. This new evidence forced us to consult the neurologist for the second time, but the neurologic findings of cortical sensory loss in the left side remained equivocal. When both sides were examined on symmetrical locations, there was a questionable impairment in light touch, pain for pinprick and temperature, joint sensation and vibration sense in the left upper limb up to the wrist. However, there was no information available regarding simultaneous examination on symmetrical locations.

Somotosensory evoked responses were assessed during this admission (i.e., 2 years after the onset of sensory symptoms); responses were obtained using both the median nerve stimulus at the wrist and posterior tibial nerve stimulus at the ankles. Potentials that were identified on median nerve stimulus were N13, N19 and P22, and on posterior tibial nerve stimulus were P37 and N45. The absolute and interpeak latencies and the amplitude of the responses were normal. There were no right-to-left differences noticed on stimulation of both the median nerves and posterior tibial nerves.

A neuropsychologic assessment indicated high average intelligence with no focal or global deficits. Further carotid Doppler ultrasonographic studies showed no flow abnormalities in the carotid vessels. Cardiac echo was reported to be normal, and no source of embolus was found. Results of investigations for collagen diseases were negative.

There was an incidental finding of a 13-mm cyst in the left lobe of the thyroid. However, thyroid stimulating hormone (TSH), triiodothyronine (T3) and free thyroxine (T4) levels were within normal limits. Free testosterone was 13.2 pmol/L above the normal range for her age (2.8–11.1 pmol/L) and dehydroepiandrosterone (DHEA) sulfate was 8.2 μmol/L within high normal range (1.2–10.3 μmol/L). This led to an ultrasonographic examination of her abdomen, which showed ovaries with small follicular cysts measuring up to 5 mm, suggesting polycystic ovarian cysts. There were no menstrual irregularities or clinical evidence of hyperandrogenism. There was no evidence of insulin resistance (fasting blood glucose was 5.8 mmol/L, and insulin was 90 pmol/L). A subsequent referral to the gynecology department did not reveal any new findings to suggest a link between her psychiatric condition and ovarian cysts.

The patient was discharged from the hospital with the final diagnosis of somatization disorder with left-side sensory conversion symptoms and a right parietal infarct. Psychotherapy performed in an outpatient setting induced a complete remission of sensory symptoms and partial recovery of other somatic symptoms.

## Diagnostic issues

At the initial evaluation, the distribution of sensory symptoms (splitting the midline) and the presence of other somatic symptoms caused us to consider conversion sensory symptoms. Splitting the midline indicates that sensory impairment abruptly stops at the middle of the face and body. This is traditionally considered to be an important sign in establishing the diagnosis of sensory conversion symptoms because sensory fibres of the skin normally spread across the midline.[6] However, the finding of a right parietal lobe infarct in this patient caused diagnostic uncertainty.

The acute onset of sensory symptoms preceded by a headache and the localization of sensory symptoms to the contralateral side of the infarct suggest that the association of a right parietal infarct and sensory symptoms may not be coincidental but may be etiologically related. The next most important clinical task was determining whether the presenting sensory symptoms were true physical symptoms, conversion symptoms or a mix of both. As recommended by Levy and Mushin,[7] we used somatosensory evoked response to differentiate physiologic from nonphysiologic disturbances of sensory perception. It has been generally agreed that a stimulus applied to an affected area will evoke a normal response recordable over the contralateral receiving area in conversion disorder, whereas no response or a diminished response will be recorded in organic disorders. Thus, the normal cortical somatosensory evoked potentials recorded in this patient could be interpreted as support of the diagnosis of conversion symptoms, despite the presence of a parietal lobe infarct. However,

some reports suggest those patients with a parietal lesion and definite sensory loss may have normal cortical somatosensory evoked responses.[8] The time interval between the onset of infarct and the sensory evoked response testing appears to be critical; some reports suggest the reversal of abnormal responses on retesting several months after the infarct.[9] Hence, given that the sensory testing was performed 2 years after the onset of acute lateralized sensory symptoms, the normal responses in this patient could be attributed to the prolonged time interval.

To complicate the matter further, it has been suggested that conversion sensory disorder may be associated with diminished sensory perception at the cortex. Low-intensity peripheral nerve stimulation on the affected side in patients with hysterical anesthesia may produce abnormal cortical somatosensory evoked responses with smaller amplitude.[7] As well, compared with nerve stimulation, skin stimulation has been reported to produce smaller responses, irrespective of intensity, implying a diminution in peripheral receptor sensitivity.[7] Thus, somatosensory evoked responses may not be helpful in establishing the diagnosis of conversion sensory symptoms coexisting with a parietal lobe infarct, but may provide valuable information about neurophysiologic mechanisms underlying conversion sensory symptoms.

We did not use forced-choice testing[10] to detect hysterical sensory symptoms in this patient. With forced-choice testing, patients with hysterical sensory symptoms give fewer correct responses than would be expected on a chance basis. However, it is possible that some patients with conversion symptoms will respond within the range associated with chance.[10]

Clinical responses to psychotherapy or suggestive techniques (e.g., hypnosis and amylobarbital interviews) can be used to differentiate conversion from a true physical symptom. Patients with conversion symptoms may demonstrate a marked or complete recovery with hypnosis[11] or amylobarbital interviews[12] or brief psychotherapy.[13] Psychotherapy effectively resolved the sensory conversion symptoms in this patient. Thus, aside from the clinical characteristic of splitting the midline, symptom resolution with psychotherapy clinched the diagnosis of conversion symptoms.

The parietal lobe infarct associated with conversion sensory symptoms is consistent with other reports of nonphysiologic sensory symptoms in patients with parietal lobe injury.[14] The frequent occurrence of conversion symptoms in association with physical disease is often considered to be evidence against the diagnosis of primary conversion disorder. However, it is possible that conversion symptoms (or disorder) may be secondary to physical disease. Although a causal association between conversion disorder and cerebral diseases could be explained by physiologic mechanisms, the present nosology fails to recognize the concept of secondary conversion disorder or conversion disorder due to physical disease. However, recognizing the existence of conversion disorder secondary to physical disease would allow us to consider the clinical possibilities that conversion disorder may be a manifestation of physical disease or conversion symptoms may coexist with or be superimposed on true physical symptoms.

The cause of the parietal lobe infarct in this young woman is another major clinical concern. An extensive search for thromboembolic or cerebrovascular abnormalities was unsuccessful. However, it could be argued that the parietal lobe infarct might be indirectly related to the "polycystic ovarian syndrome-like" clinical picture. Polycystic ovarian syndrome is frequently associated with insulin resistance syndrome (IRS), a major risk factor for cardiovascular disease.[15,16] However, given the normal fasting blood glucose and insulin levels, it is unlikely that insulin resistance increased the risk of cerebral infarct in this patient.

## Brain mechanisms

Conversion sensory symptoms related to a parietal lobe infarct may be associated with neurophysiologic mechanisms that differ from those that underlie conversion symptoms not associated with physical disease. According to the well accepted neurobiological theory of Ludwig[17] and Whitlock,[3] hysterical symptoms are due to a failure of integration of attention or conscious awareness with afferent stimulation and are mediated by abnormally increased levels of corticofugal (corticocortical and corticoreticular) inhibitions of these afferent stimuli. This model may not be relevant to this patient with a parietal infarct because cerebral lesions may be associated with decreased corticofugal inhibition. Hypochondriasis and psychogenic pain are presumed to be related to decreased corticofugal inhibition, which, in turn, permits increased intrusion and central processing of afferent stimulation.[17] For conversion symptoms associated with cortical lesions, decreased levels of corticofugal inhibition may be coupled with

decreased processing of afferent stimulation. However, this may not fully explain the formation of sensory conversion symptoms in patients with parietal lesions or the reason that the conversion symptoms closely resemble the previously experienced physical symptoms.

The model we propose suggests that decreased corticofugal inhibition due to a parietal lesion causes reactivation of somatosensory memories represented at the thalamus, and this may contribute to the formation of sensory conversion symptoms that resemble previous experiences of somatic sensation or sensory symptoms. This model is consistent with evidence that suggests somatosensory memories are represented at the thalamus and the parietal cortex and that cognitive and affective disturbances may reactivate somatosensory memories, as described in pain 'memories' in phantom limb sensations.[18] Further, this model does not preclude the contribution of social concepts such as the sick role[19,20] and illness behaviour[21] in the formation of conversion symptoms in this patient. Owing to her overconcern about emotional and somatic symptoms, the postulated nondominant parietal lobe dysfunction, anosognosia[22] (i.e., denial of illness) or hysteria-related "la belle indifference" (i.e., absence of distress) may not be relevant to this patient. The lateralization of conversion symptoms to the left side in this patient concurs with previous observations of left body predominance in the manifestation of unilateral motor or sensory conversion disorder.[23,24]

As in many areas in psychiatry, clinical indicators are vitally important in establishing the diagnosis of conversion symptoms associated with organic abnormalities. Given the frequent association of physical disease and conversion symptoms, the inclusion of conversion disorder secondary to physical disease in the current nosology should be considered. Further research seems warranted to determine the role of the thalamus in sensory conversion symptoms.

**Acknowledgements:** This work was partly supported by a fellowship grant from the Canadian Psychiatric Research Foundation. This report was presented in a poster format at the Psychosomatic Medicine Clinical Research Day — Toronto Hospital in Nov. 1994 and at the American Neuropsychiatric Association Conference — Pittsburg in Feb. 1995.

We thank Dr. Paul Sandor, Director, In-patient Psychiatry, Toronto Western Hospital for clinical supervision, and Ms. April Hummerston for typing the manuscript.

**Competing interests:** None declared.

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