

Case report

Unusual visual symptoms and Ganser-like state due to cerebral injury: a case study using ^{18}F -deoxyglucose positron emission tomography

Stephen L. Snyder^{a,*}, Monte S. Buchsbaum^a
and R.C. Krishna^b

^a*Department of Psychiatry, Mount Sinai School of Medicine, New York, NY, USA*

^b*Formerly: Department of Neurology, Mount Sinai School of Medicine, New York, NY, USA*

Received 14 April 1997

Revised 19 February 1998

Bizarre visual symptoms and absurd verbal responses to questions, in a 32-year-old man recovering from a severe asthma episode, suggested a possible conversion disorder with Ganser-like symptoms. *Positron Emission Tomography (PET)* revealed bilateral lesions involving occipital association cortex and posterior temporal and parietal lobes, most likely infarcts from hypoxia. PET permitted correlation of the patient's specific cortical lesions with his unusual perceptual, cognitive, and speech symptoms, including *Ganser-like state*, to a degree not previously possible in such cases.

Keywords: Ganser syndrome, cerebral infarction, cerebral metabolism, polyopia

1. Introduction

Ganser in 1898 described four patients with acute mental disturbance marked by approximate or absurd answers to questions, clouding of consciousness, perceptual abnormalities, and conversion symptoms [9]. Since then, additional cases with more-or-less similar symptoms have occasionally been reported [6, 8, 10, 11, 12]. However, the precise noso-

logic significance of 'Ganser Syndrome' is uncertain. In practice, it may be unclear whether a given patient's 'Ganser Syndrome' or 'Ganser-like state' indicates an organic brain disorder, some other mental disorder, or a conversion or factitious disorder. We here report a patient with unusual perceptual and Ganser-like symptoms, in which conversion was suspected, but where Positron Emission Tomography (PET) eventually demonstrated an underlying neurological lesion which better explained the unusual behaviors.

2. Case description

A 32-year-old man presented to the emergency department complaining of headache and blindness. Seven days previously, he had been admitted to the medical intensive care unit with a severe asthmatic episode, in respiratory failure. He had been intubated for 24 hours, treated with intravenous corticosteroids, and discharged home on a prednisone taper after five days. He now returned to the emergency room complaining of headache and blindness, and was admitted to the neurology service.

On examination, pupils were 3 mm, reacting well to light. Acuity was light perception in both eyes. Ophthalmologic exam was otherwise normal. Head CT without contrast and cerebrospinal fluid examination were both normal.

The patient was noted to answer questions slowly and to have a blunted affect. At times he would giggle inappropriately. He was alert and stated his name correctly, but claimed not to know the time, the place, his age, or his date of birth.

*Corresponding author: Stephen L. Snyder, M.D., Mount Sinai Med. Ctr., Box 1228, One G.L. Levy Place, New York, NY 10029, USA. Tel.: +1 212 241 2155; Fax: +1 212 369 6817.

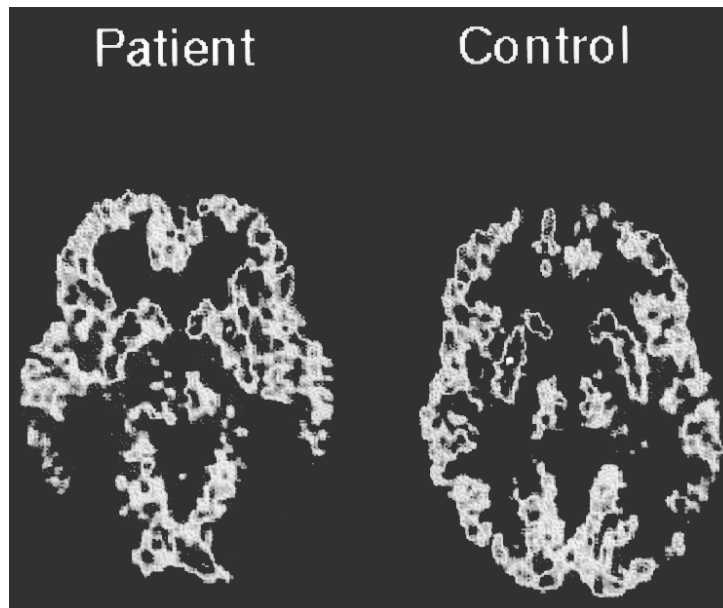


Fig. 1. Positron Emission Tomography (PET) scan with ^{18}F -deoxyglucose (FDG) in the patient and normal control. Note blue regions indicating severe metabolic decrease in occipital and parietal association areas.

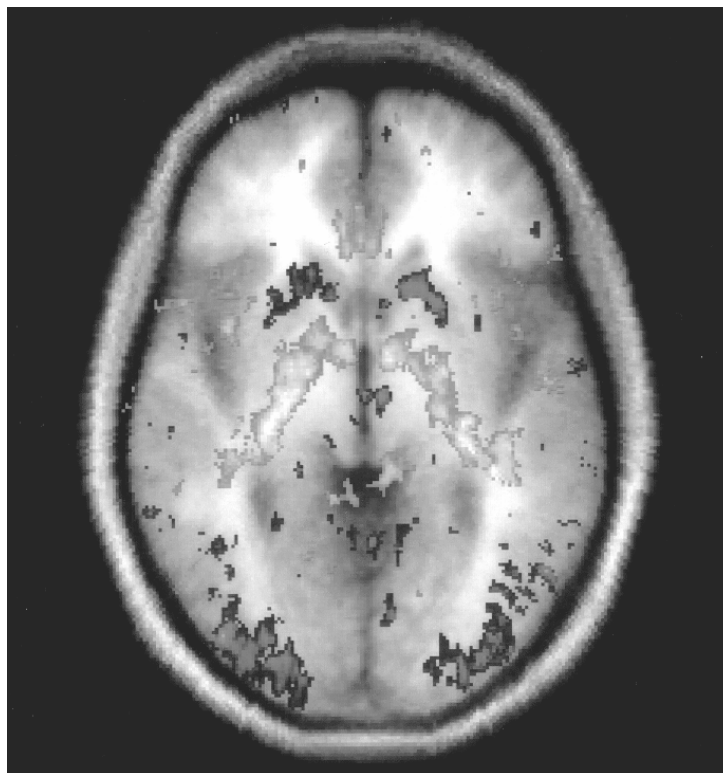


Fig. 2. Map of z -scores comparing the patient's relative metabolic values with 25 normal controls on a background of average control MRI. Patient's PET image was morphed using 500 edge points and 9 midline points to average of normal population. Blue areas are pixels with $z < -2.00$; mean value = -2.91 for the right 464 contiguous pixels, and -2.65 for the left 303 contiguous pixels. Clusters this size or bigger occur less than one time in 20 when samples of 25 individuals and a comparison subject are randomly drawn from a cohort population of 80 normal controls. This is tested by drawing 5,000 such samples from the population of 80 normals.

The day after admission to neurology, there was a single generalized tonic-clonic seizure lasting approximately 1.5 minutes with head and eye deviation to the right. EEG obtained the same day showed disorganization of the background with prominent rhythmic frontal theta and intermittent rhythmic frontal delta activity; there were no focal lateralized or epileptiform features. Visual evoked potentials were poorly formed, with significantly delayed P100 latency bilaterally. Phenytoin was started.

By hospital day 7, the patient could distinguish some distant objects. On hospital day 7, repeat EEG showed considerable disorganization and slowing of the background rhythm, and prominent frontal rhythmic delta activity. Two electroencephalographic seizures were recorded: the first involving a discharge centered on the left parietal derivations, and the second beginning in the right parieto-occipital area before becoming bilateral, with post-ictal persistence of epileptiform activity over the left parietal derivation for 10 seconds. There was no clinical correlate for either of these electroencephalographic events. Concomitant phenytoin level was 5 $\mu\text{g}/\text{ml}$, and the phenytoin maintenance dose was increased to 200 mg PO bid. The next day (hospital day 8), repeat visual evoked potentials showed improvement in the right P100 latency, which was now borderline normal, and no change in the left P100 latency, which was still delayed. On hospital day 9, the patient could recognize written numbers, and could name a pen and a knife. On hospital day 10, his visual acuity was 20/200.

The patient began to report a number of unusual visual symptoms. He described seeing not one hand held in front of him, but a series of hands moving in a circle. He reported seeing 'extra fingers' on hands. On visual field examination on hospital day 13, he described 'moving and flipping' images, especially in the central field of vision. On hospital day 14, a third EEG was significantly improved, still with some abnormal slowing but without epileptiform activity. Further diagnostic studies performed during the neurology admission included brain MRI without contrast, which revealed a signal abnormality in the right parietal cortex but was otherwise normal. Cerebral angiogram was normal.

The patient's unusual visual symptoms, absurd responses, and cheerful acceptance of his illness had initially raised questions of conversion blindness and Ganser-like state. On hospital day 13, he was interviewed in detail by the consulting psychiatrist. He was well-groomed and calm. He appeared cheerful and relatively unconcerned. He described strange distor-

tions of vision, which he said had begun as his vision was improving. People's noses, for example, looked to him 'flat – like bulldogs', while the rest of their faces looked normal. The fingers of an extended hand held straight up appeared to him to be bent 90 degrees lateral to the rest of the hand. At times he claimed to have double, triple, or quadruple vision.

He was completely oriented except for the date of the month, which he missed by four days. He registered three objects immediately, but recalled only one after five minutes. Simple calculations presented great difficulty. When asked to subtract serial 3's from 20, he answered, '20, 18, 16, 15, 14, 13', and when asked to try again, he repeated the same sequence. The sum '2 + 4' he gave as '5'. When asked the distance from New York to California, he replied, 'New York to Miami is a long distance'. He affirmed that he knew Miami was in Florida. He was unable to sign his name or copy geometric figures. He correctly named a pen and a shirt, but could not name the cap of the pen or the button of the shirt. He remembered his birthdate on some days but not others.

The patient was discharged on hospital day 15. Seventeen days later, at 39 days after the initial asthmatic episode, he returned as an outpatient for Positron Emission Tomography (PET). Using the methods described elsewhere [7], scans were obtained with an injection of 5 mCi of ^{18}F -deoxyglucose (FDG) following tracer uptake while the patient performed the California Verbal Learning Task. The patient reported difficulty reading the words from the screen. The words were read to the patient, who repeated them. Memory performance was poor on all indices. The scans showed severely reduced uptake in the lateral occipital lobes (Brodmann's areas 18 and 19, or association cortex) and the posterior regions of the temporal and parietal lobes (see Figs 1, 2).

The patient's bizarre visual symptoms had resolved by 46 days after the initial asthmatic episode. Vision continued to improve, although his useful vision remained impaired at seven months. Cognitive symptoms persisted. At 37 days after the initial asthmatic episode, he still found arithmetic baffling: 'It's like the math is lost in my head – I can't see it in my head.' At 83 days, he continued to have difficulty naming objects: 'I'm still digging for the words – I try to say something, and I jump and say something different – I try to find a better word, but it jumps around.' Interpretation of his speech difficulty was complicated by the fact that his original language had been Spanish, although he had been raised bilingually in English and Spanish since age 9.

3. Discussion

Bizarre visual symptoms, approximate or absurd answers, inconsistent deficits, and bland indifference led at first in this case to consideration of a diagnosis of conversion disorder with Ganser-like symptoms. Later, PET scanning revealed severe, bilateral, highly localized hypometabolic lesions in the posterior cortex, presumably areas of infarct due to an episode of asphyxia. With the aid of PET, it was possible to understand the patient's perceptual, cognitive, and speech abnormalities as symptoms of injury to specific cortical functions, and to correlate these with his specific lesions of association cortex to a degree not previously possible in such cases.

There appears to have been an initial stage of 'ictal blindness' [1], followed by a stage of 'cortical association blindness' [5]. The stage of cortical association blindness was marked by such bizarre symptoms as quadruple-vision and seeing objects bent 90 degrees or objects wandering in a circle. Critchley [4] emphasized that spatial disorders and 'metamorphopsia' could be symptoms of parietal lobe disease, and Bender and coworkers described patients with cerebral disease who showed polyopia and metamorphopsia similar to the current case [2, 3]. Cognitive symptoms included loss of mental representations of numbers and difficulty with calculations, consistent with dysphasia due to anoxic damage to posterior temporal and parietal areas. Speech abnormalities, including 'approximate' or absurd answers, were ultimately understood as the patient's attempt to compensate for a nominal dysphasia. The fact that nominal dysphasia might masquerade as 'approximate answers' was discussed by Whitlock [12] and supported by several of his published cases. Finally, the patient's attitude of bland indifference to his illness was understood as a cognitive deficit of a kind seen in parietal lobe injury [4].

The patient displayed nearly all of the symptoms of the so-called Ganser syndrome: approximate or absurd answers, clouding of consciousness, perceptual disturbances, and conversion-like symptoms [6, 9, 12]. The precise boundaries of Ganser syndrome and Ganser-like states are not well-defined. The current case seems to belong to a group of cases in the literature in which Ganser-like symptoms follow cerebral in-

jury [8, 10, 11, 12]. PET here demonstrates in such a case how damage to lateral occipital and posterior temporal and parietal lobes produced perceptual, cognitive, and speech abnormalities which presented as a Ganser-like state.

Acknowledgements

The authors thank Dr. C.T. Wei, J. Spiegel-Cohen, and C. Tang for technical assistance. This work was supported in part by MH-40071 to Dr. Buchsbaum.

References

- [1] E. Barry, N.M. Sussman, T.M. Bosley and R.N. Harner, Ictal blindness and status epilepticus amauroticus, *Epilepsia* **26** (1985), 577–584.
- [2] M.B. Bender and M.G. Kanzer, Metamorphopsia and other psychovisual disturbances in a patient with tumor of the brain, *Archives of Neurology and Psychiatry* **45** (1941), 481–485.
- [3] M.B. Bender and A.J. Sobin, Polyopia and palinopia in homonymous fields of vision, *Trans. Am. Neurol. Assoc.* **88** (1963), 56–59.
- [4] M. Critchley, Psychiatric symptoms and parietal disease: differential diagnosis, *Proceedings of the Royal Society of Medicine* **57** (1964), 422–428.
- [5] R. Efron, Post-epileptic paralysis: theoretical critique and report of a case, *Brain* **84** (1961), 381–394.
- [6] M.D. Enoch, W.H. Trethowan and J.C. Barker, The Ganser syndrome, in: *Some Uncommon Psychiatric Syndromes*, John Wright and Sons, Ltd., Bristol, 1967, pp. 41–55.
- [7] M.M. Haznedar, M.S. Buchsbaum, M. Metzger, A. Solimando, J. Spiegel-Cohen and E. Hollander, Anterior cingulate gyrus volume and glucose metabolism in autistic disorder, *Am. J. Psychiatry* **154** (1997), 1047–1050.
- [8] J. McEvoy and T. Campbell, Ganser-like signs in carbon monoxide encephalopathy (letter), *Am. J. Psychiatry* **134** (1977), 12–13.
- [9] C.E. Schorer (translator), The Ganser Syndrome, *British Journal of Criminology, Delinquency and Deviant Social Behavior* **5** (1965), 120–126. [Translation of S.J.M. Ganser, A peculiar hysterical state, *Archiv für Psychiatrie und Nervenkrankheiten* **30** (1898), 633.]
- [10] M. Sigal, D. Altmark, S. Alfici and M. Gelkopf, Ganser syndrome: a review of 15 cases, *Comprehensive Psychiatry* **33** (1992), 134–138.
- [11] H. Weiner and A. Braiman, The Ganser syndrome – a review and addition of some unusual cases, *Am. J. Psychiatry* **111** (1955), 767–773.
- [12] F.A. Whitlock, The Ganser syndrome, *Br. J. Psychiatry* **113** (1967), 19–29.



Hindawi
Submit your manuscripts at
<http://www.hindawi.com>

