

## CASE REPORT

# Neurobiological Basis of Ganser Syndrome

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### ABSTRACT

Ganser syndrome continues to be a rare and widely misunderstood condition. While DSM-IV classifies Ganser syndrome as a dissociative disorder, its etiology continues to be debated. There are episodic reports in the literature of Ganser syndrome in patients with head trauma or strokes. However, the mechanisms by which these cerebral insults lead to Ganser syndrome or other dissociative states are largely unknown. A case of a patient with Ganser syndrome with a prior history of stroke and bifrontal infarcts is described. This case demonstrates how organic pathology may predispose a patient to dissociative states, such as Ganser syndrome. We review the relationship between hyperglutamatergic states, caused by stroke and stress, and dissociative symptoms.

**Key words:** Ganser syndrome, stroke, glutamate, dissociative symptoms

The Ganser syndrome (GS) is a very rare and unusual condition first described in 1897 by the German psychiatrist Sigbert Ganser in his studies of prisoners. The syndrome is defined by four essential clinical features 1) the approximate answer, 2) clouding of consciousness, 3) somatic conversion features, and 4) hallucinations (Enoch & Hall, 2001). The approximate answer to simple questions (the Ganser symptom) is the most striking symptom, but not alone diagnostic for GS (Scott, 1965). The etiology of the syndrome is still polemical. While psychological mechanisms have been fundamental to the development of GS, there are anecdotal reports associating this syndrome with organic brain conditions. However, most of these cases are of patients with head injury. We report a case of a patient who had a history of stroke and developed GS after he faced a psychologically stressful situation. The organic etiology of GS is further discussed and glutamatergic mechanisms in the development of dissociative symptoms are explored.

### CASE REPORT

Mr. B, a 54 year old single man, was

admitted with depressive symptoms and suicidal ideation. He had been doing well up to 3 months prior when he obtained a credit card and accumulated a large debt. He received a few threatening calls from creditors and began believing that the police would come and arrest him because of his debt. He then became depressed, afraid, and socially withdrawn. Mr. B's medical history was significant for a stroke 2 years ago, which left him with residual left sided weakness. His medical conditions included diabetes and migraine headaches. He had high school education and was currently unemployed.

During hospitalization, Mr. B displayed apathetic indifference and psychomotor retardations. His Mini Mental Status Examination (MMSE) was 16/27 (excluding items related to reading, writing, and design copying). His main deficits were in temporal orientation, serial 7's, and recall. On more detailed questioning, he gave approximate answers, interspersed with correct ones. When asked 2 + 2, he answered, after much deliberation 5. When asked the color of the sky, he said yellow. He answered that the month between Feb and April was June. The day after Sunday was Tuesday, and the day after Wednesday was Friday. He answered

that Christmas fell on November 27, that there were 6 days in a week and 10 months in a year. After slowly counting each finger, he correctly answered that he had ten fingers. He was able to say that he had ten toes, two arms, and two legs. To many simple questions of mundane knowledge, he would just answer 'don't know'. He became more tired and uncooperative as the questioning continued, with more and more 'don't know' answers. His answers were inconsistent and would vary day to day. He initially was able to say that a horse has four legs. A few to two days later, he changed his answer.

On initial examination, Mr. B endorsed difficulty seeing. He gave consistently incorrect answers to counting the number of fingers and claimed he could not see things during exam. However, Mr. B was able to easily maneuver himself throughout the hospital floor without problems and was even seen "reading" the newspaper at times. Upon questioning, Mr. B admitted to auditory hallucinations in the evenings, in the form of screaming voices and voices talking to him. He also gave a questionable history of visual hallucinations, which he noted might have just been dreams.

Mr. B's laboratory investigations were unremarkable. His EEG was normal while a head CT scan revealed a few scattered punctate foci of decreased attenuation in both frontal lobes, right greater than left, most consistent with old lacunar infarcts. On frontal assessment battery (Dubois et al, 2000). Mr. B displayed significant improvement in 5 out of 6 categories, including conceptualization, lexical fluency, programming, sensitivity to interference, and go-no-go testing. Overall, Mr. B scored 7/18 on this test, indicating severe frontal lobe dysfunction.

Mr. B was treated with risperidone (0.5 mg/d) and citalopram (30 mg/d). He often voiced his feelings that he needed housing with a higher level of care, after placement issues were settled, Mr. B was discharged one week after admission to a personal care home where he felt he would be safe. He was pleased with his new housing and appeared cheerful and energetic. However, before discharge, Mr. B continued to have cognitive deficits and to give approximate

answers. While he no longer had suicidal ideation, the persistence of approximate answers indicated that his Ganser syndrome had not yet resolved.

## DISCUSSION

This patient clearly met the four essential criteria for diagnosing GS. Interestingly, this patient had bifrontal of lacunar infarcts and impairment on neuropsychological testing, which indicated an organic etiology. Such an etiology is supported by anecdotal reports. In a review of 15 patients with GS, Sigal et al (1992) reported that 7 out of their 15 patients had evidence for organicity; with six patients having history of head trauma with loss of consciousness and one who had a right cerebrovascular event with left hemiparesis. Closed head injury with or without loss of consciousness has been associated with GS (Miller, 1997; Lee & Koenig, 2001). However, there are only rare reports of GS following a cerebrovascular event. In one such case, a Patient with micro-embolic shower to the brain, including the frontal region, developed GS after the event Latham et al. 1978. The increasing number of reports of brain insult-associated GS is not surprising considering the fact that two of Ganser's three original patients had history of head injury (Ganser, 1965).

The mechanism by which a brain insult may predispose one to GS and other dissociative states is unknown but recent literature supports the role of Glutamate in dissociative symptoms. The current consensus is that GS is a dissociative syndrome. Stress induces a cortico-limbic release of glutamate, leading to the hypothesis that hyperglutamatergic states may be involved

in the dissociative symptoms in PTSD (Chambers et al, 1999). As Ganser syndrome is seen in prisoners and those in stressful situations, there may be a significant role of stress, and possibly glutamate, in its etiology. Glutamate surge is also seen after a brain infarct and dissociative symptoms have been reported to occur following stroke (Chambers et al, 1999; Duggal, 2003). Further evidence supporting the role of glutamate in dissociative symptoms is provided by Moghaddam et al (1997) who showed that in rats, low doses of ketamine increased both glutamate and dephamine outflow in the prefrontal cortex. Ketamine, which activates glutamatergic neurotransmission by acting as a noncompetitive NMDA antagonist, has been shown in humans to produce symptoms similar to those seen in schizophrenia and dissociative states. Ketamine has also been shown to impair performance in frontal lobe sensitive tests (Moghaddam et al, 1997). Finally; frontal lobe involvement has been cited to result in dissociative symptoms has evidenced in the case of a man who developed recurrent dissociative flashbacks after suffering a traumatic brain injury to the right dorsolateral prefrontal cortex (Berthier et al, 2001). Thus drawing from these observations, it can be speculated that hyperglutamatergic transmission especially in the frontal lobes may predispose individuals to dissociative states such as GS. More research is headed so further address this hypothesis hyperglutamatergic transmission.

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