# MANIA FOLLOWING LEFT HEMISPHERE INJURY

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*ABSTRACT*

***A case of mania following closed head injury to the left hemisphere is reported. The patient presented with a self-limiting manic episode that recovered without treatment. It is postulated that the head injury is the causative factor in this case. This was supported by laboratory results as well as psychological investigations. It is believed that non-dominant right hemisphere injury is related to the development of mania. However, a search of the literature revealed five other cases of mania following left hemispheric injury. Although the mechanisms implicated in the pathogenesis of secondary mania have not been established, this case adds to the growing evidence that head injury may be directly causative in affective psychoses. Therefore, it is premature to conclude that mania is a pathology of the non-dominant right hemisphere.***

***Keywords:*** *secondary mania, organic psychosis, head injury*

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## INTRODUCTION

The occurrence of mania following head injury was originally not included as one of the causes of secondary mania. Other than the two controlled studies involving 11 and 17 patients from John Hopkins University[1,2], and a recent paper which examined 6 cases of secondary mania[3], the medical literature on this subject comprises mainly of anecdotal case reports which fulfil the criteria proposed by Krauthammer & Klerman[4].

This lack of systemic study is probably due to the relatively uncommon occurrence of secondary mania. Most of these reports suggest that secondary manic patients are older than primary manic patients[4], have infrequent family history of psychiatric illness[4], and that there is a preponderance of lesions located in the right hemisphere[1,5]. More recently, Jorge et al[3] reported that secondary mania was not found to be associated with the severity of brain injury, degree of physical or cognitive impairment, level of social functioning, or previous family or personal history of psychiatric disorder. To date, only five cases of secondary mania were associated with brain injury to the left hemisphere (Table I). The author presents in this report, the sixth case of secondary mania following pathology of the left hemisphere.

## CASE REPORT

Patient A, a 18-year-old right-handed college student without previous history of mental illness, was admitted as a voluntary patient with three weeks history of over-activity, talkativeness, irritability and assaultative behaviour. He was grandiose and claimed to be possessed by 'Guan Yu'*. The patient was observed to talk to himself as he claimed that he was able to communicate with other spirits.

A month prior to his index admission, the patient was knocked down by a truck while riding on a motorcycle. He sustained facial laceration, periorbital haematoma and sub-conjunctival haemorrhage. There were also abrasions on his upper chest and right thigh. He was drowsy and restless but there was no history of loss of consciousness. Skull X-ray showed a close chipped fracture to the left inferior groove of the orbit. Chest and cervical X-rays were normal. He was treated with anneomycin and discharged from the surgical unit four days later.

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At home, the patient was initially noticed to be quiet and easily tired. However, over the next few days, he became restless, irritable and easily upset. Indeed, his assaultative behaviour towards his family members precipitated his admission to another psychiatric hospital but his family requested discharge the following day. Although he was prescribed haloperidol 1.5mg three times a day, the patient refused medication. His disturbed behaviour persisted and he was re-admitted two weeks later for inpatient treatment. Throughout this period, there was no report of confusion or disorientation from his care givers.

Mental state examination on admission revealed a very cheerful young man who was spontaneous in his reply. At times, he was restless, irritable, had short attention span and a reduced need for sleep. There was pressure of speech but no flight of ideas. He had grandiose delusion of being possessed by 'Guan Yu' and had special abilities to communicate with other spirits. There was no auditory or other forms of hallucinations. He was fully oriented in time, place and person. Serial-7, which is a test for concentration and mathematical ability, was quick and accurate. Remote memory, which was assessed from his ability to give an account of his past life, was intact. Although he recalled the date and time of the accident, he had amnesia of events at the point of impact. However, he remembered his admission and recovery in the surgical unit. There was also no difficulty with immediate recall and his intelligence was also not affected. Clinical examination was uneventful and revealed no neurological deficits.

Electro-encephalography (EEG) revealed decreased amplitude of the left hemisphere and increased slow wave activity over the left fronto-temporal/orbital region. Computerised tomography (CT) of the head revealed no abnormality. Psychological testing using Wechsler Adult Intelligence Scale (Revised) showed left hemisphere dysfunction, with a verbal IQ of 99 and performance IQ of 118. Wechsler Memory Scale and Benton Visual Retention Test were normal.

In view of the history of head injury, the patient was admitted for further management. In the ward, he did not exhibit any assaultative behaviour. His restlessness and irritability were effectively managed with occasional physical restrains. The patient gradually settled in the ward and his symptoms resolved without medication. Indeed, he was discharged one week after admission. A repeat EEG one month following discharge was normal. He remained well on follow-up one year later.

* Guan Yu was a famous General in the Chinese history. He was considered the personification of loyalty and was popularly known as 'Guan Gong' (Lord Guan) or 'Guan Di' (Emperor Guan). His exploits were recorded in the "Romance of the Three Kingdom", a historical novel by Luo Guanzhong. He was deified by later rulers and many temples were built in his memory.

**Table I – Secondary mania associated with left hemisphere pathology**

| Authors, year | Age | Sex | Handedness | Psychiatric history | Aetiology | Site | Interval | Depression | Treatment response |
|---|---|---|---|---|---|---|---|---|---|
| Bourgeois et al, 1967[6] | 25 | M | ? | - | Bone tumour | Sub-temporal | 15 years | + | + |
| Jampala et al, 1983[7] | 25 | M | R | - | Infarction | Temporo-parietal | 10 months | - | + |
| Herlihy et al, 1979[8] | 58 | F | ? | ? | Infarction | Fronto-temporal | ? | - | + |
| Starkstein et al, 1988[9] | 27 | M | R | + | Infarction | Frontal | 2 months | + | + |
| Drake et al, 1990[10] | 52 | M | ? | - | Infarction | Ventral-pontine | ? | - | + |
| Present report | 18 | M | R | - | Trauma | Fronto-orbital | 12 days | - | + |

## DISCUSSION

This patient satisfied the diagnostic criteria for secondary mania as proposed by Krauthammer & Klerman[4]. Their inclusion criteria were an illness duration of at least one week, an elated or irritable mood, and the presence of at least two of the following behaviours: hyperactivity, "push of speech", flight of ideas, grandiosity, decreased sleep, distractibility, and lack of judgement. Their exclusion criteria were a clear previous history of affective illness and the presence of a confusional state. It is generally accepted that the shorter the interval between the head injury and the development of a psychosis, the stronger is the likelihood of the two conditions being linked[11]. The temporal relationship between the patient's closed head injury and the development of mania, the negative past and family history of affective illness, and the absence of a confusional state support the diagnosis of secondary mania. Indeed, in a study of 66 consecutive patients with acute closed head injury, Jorge et al[3] reported that the frequency of secondary mania was 9% (6 out of 66 cases). This figure was significantly greater than the 1% of secondary mania observed in their group of 300 stroke patients[3]. Jorge et al[3] attributed this difference to the different pathophysiological mechanisms involved in stroke and traumatic head injury, and to the higher frequency of anterior temporal lesions observed in patients with closed head injury.

The duration of this patient's manic episode, from the time that he was observed to be irritable to the time of resolution, was approximately one month. This was similar to the finding of Jorge et al[3] where the duration of the mania was brief and lasted approximately two months. Although this patient was initially noticed to be quieter than usual, there was insufficient evidence to suggest that the patient was depressed prior to the onset of mania.

The presence of irritable mood and assaultative behaviours were the reasons for the patient's consultation. This presentation is in keeping with previous report that manic episode following closed head trauma were characterised by irritable mood and assaultative behaviours rather than by euphoria[12]. This observation seems to support the view that organic bipolar illness after head trauma may have distinct clinical pathological features[12].

This patient was fully right-handed and without any family history of left-handedness. His verbal IQ which was 19 points below the performance IQ, demonstrated that his left hemisphere was dominant for language as well as right-handedness. Although closed-head injuries may produce multifocal brain damage and there was no localising lesion on CT scan, the presence of a chipped fracture of the left inferior groove of the orbit, the abnormal neuro-psychological testing, and the spontaneous resolution of slow wave activities over the left fronto-temporal region which correspond with the clinical resolution of his manic symptoms, rendered the chance occurrence of his psychological condition very unlikely.

Lesions of either the frontal lobe or limbic system have frequently been invoked in the explanation of affective changes after brain lesions. Sackeim et al[13] reported euphoric mood following right hemispherectomy and pathological laughing associated with right hemisphere damage. These findings suggest a strong association between secondary mania and lesions to the right hemisphere. Cummings[14] suggested that lesions producing secondary mania are located close to ascending monoaminergic pathways which were thought to play a role in affective disorders. Starkstein et al[9] reported that the association between post-stroke mood disorder and lesion location might be a result of damage to the ascending biogenic pathways, with injury to one hemisphere producing a different biochemical and hence emotional behavioural syndrome from injury of the opposite hemisphere. More recently, Jorge et al[3] observed that the presence of temporal basal polar lesions was significantly associated with secondary mania, although lesions at the orbitofrontal region are seldom reported. Using logistic regression analysis, Jorge et al[3] showed a significant association between lesion location and dropping out of the study; the dropout group had significantly higher frequencies of both cortical and orbitofrontal lesions. The authors suggested that this could have resulted in an underestimation of both the prevalence of secondary mania, and the association with orbitofrontal lesion location[3]. Whatever the final pathophysiological pathways, it must be able to account for the observation that injury to the left hemisphere, albeit rare, can be associated with the development of a manic syndrome.

The treatment of secondary mania should be directed at the underlying condition and the adequate control of the manic episodes. The conventional management of acute mania includes treatment with neuroleptics[15], lithium[6,17], electro-convulsive therapy[18], and carbamazepine[7]. As far as the author is aware, this is the only report where the patient's manic symptoms resolved spontaneously without treatment. The patient's recovery was confirmed by the normal EEG one month after his discharge.

The infrequent occurrence of secondary mania is one of the characteristics of this disorder that has never been adequately explained[1]. Cummings and Mendez[5] attributed this rare occurrence to its association with relatively rare lesions of the right hemisphere. However, this and the other five cases of secondary mania following left hemisphere pathology suggest that other factors in addition to a right hemisphere lesion are involved in the pathogenesis of this condition. Further research is thus required before we can conclude that secondary mania is due to pathology of the non-dominant right hemisphere. Long term follow-up of this group of patients will also provide invaluable information on the course of this rare and intriguing condition.

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