

# Case Study: Bipolar Disorder After Head Injury

KAPIL SAYAL, M.R.C.PSYCH., TAMSIN FORD, M.R.C.PSYCH., AND RODERIC PIPE, M.R.C.PSYCH.

## ABSTRACT

A case of bipolar disorder subsequent to a mild head injury in a 15-year-old girl is reported. Review of the literature indicates that this is an extremely rare outcome. Lack of adequate follow-up studies makes it difficult to accurately predict type and severity of psychiatric outcome. Assessment and management involves ongoing consideration of both organic and psychosocial factors even after initial negative investigations. *J. Am. Acad. Child Adolesc. Psychiatry*, 2000, 39(4):525–528. **Key Words:** head injury, adolescent, mania, bipolar disorder, psychosocial factors.

Head injuries and their sequelae are one of the most common reasons for emergency admission to the hospital. As intensive care facilities improve, many of those who in the past would have died of a head injury now survive (Gillberg, 1995). Their incidence among children is increasing, accounting for 25% of all head injuries. Those between the ages of 15 and 24 are at highest risk (Livingston and McCabe, 1990). Psychiatric disturbance after a childhood head injury is a well-recognized phenomenon, although much less predictable than cognitive outcome (Chadwick, 1985). However, mania or bipolar disorder after head injury is rare, even in adults (McAllister, 1992). We describe the management of a 15-year-old girl who presented with manic symptoms shortly after a head injury and discuss the issues raised with regard to the etiology, course, and prognosis of her illness.

## CASE REPORT

A.B. is a 15-year-old girl with a history of anorexia nervosa (13-kg weight loss, overvalued ideas about shape and weight, and amenorrhoea) 2 years previously. This occurred secondary to peer pressure about her appearance and exam stress. She was monitored by her general practitioner and improved after her exams. She had no developmental or physical health problems, and there was no family history of mental illness. She functioned

well academically and was described as being hardworking with high standards.

A.B. presented 4 weeks after a head injury which she sustained when she fell from her bicycle. The fall was not witnessed directly, and she may have lost consciousness for several minutes. She had posttraumatic amnesia for 5 hours and retrograde amnesia for 2 hours. Initially she was drowsy, slow to respond, and disoriented in time but oriented in place and person. A skull x-ray was normal. She was admitted for observation for 3 days, and during this time she described olfactory hallucinations on 2 occasions. At discharge she was fully oriented but had unequal pupils.

During the following 3 weeks A.B. was tired, lost interest in activities, had poor self-care, slept excessively, and worried about not remembering the accident. The family were then involved in a traffic accident with no reported injuries. That evening she became overtalkative, slept poorly, and was agitated and fearful. She was admitted to a pediatric ward where she was distractible, with pressured speech, elated and labile mood, and grandiose thoughts. A cranial computed tomography (CT) scan revealed no abnormalities. She began a regimen of oral chlorpromazine up to 300 mg/day but remained agitated, disinhibited, and thought-disordered. She acknowledged difficulties in thinking, and hearing voices, but was unable to elaborate.

The patient was transferred to an adolescent psychiatric unit, where she improved in the following 2 weeks; her psychotic features resolved completely. An EEG was normal. A magnetic resonance imaging (MRI) scan showed small lesions caudal to the right amygdala (Fig. 1), right putamen (Fig. 2), and to the right of the pituitary gland. It was unclear whether this represented hematomas secondary to the head injury or whether a spontaneous bleed had led to the fall.

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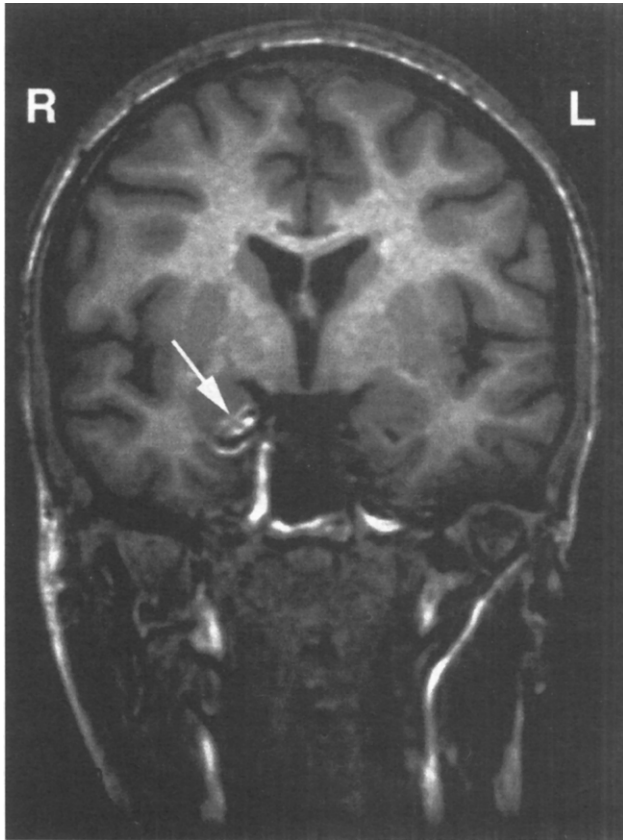
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From the Bethlem and Maudsley Hospitals, London.

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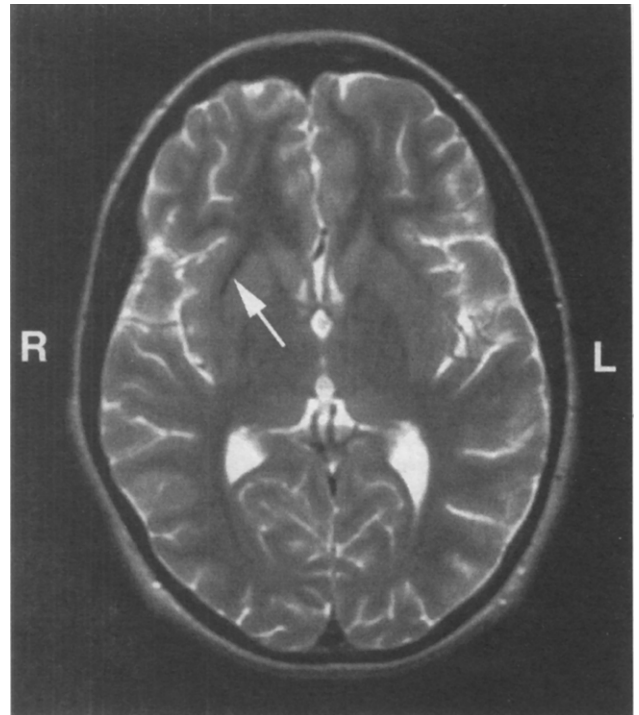
Correspondence to Dr. Sayal, Departments of Child Psychiatry, Institute of Psychiatry, Denmark Hill, London SE5 8AF, England; e-mail: k.sayal@iop.kcl.ac.uk.

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**Fig. 1** Magnetic resonance imaging scan showing hemorrhage immediately caudal to the right amygdala.

Soon after a reduction in the chlorpromazine dose, A.B. relapsed; she became distractible and perplexed, with irritable mood, pressure of speech, flight of ideas, ideas of reference, and thought disorder. During this episode there was no clinical evidence of a confusional state. Following expert neuropsychiatric advice that these manic symptoms might represent ictal phenomena, she began a regimen of carbamazepine (gradually increased to 800 mg/day). Her symptoms abated after 2 weeks but returned with similar intensity 2 weeks later. Over the following month she gradually improved but experienced residual irritability and lability of mood. A second EEG was normal, and a second MRI showed appearances consistent with partial resolution of the earlier hemorrhagic abnormalities. Carotid and invasive angiography revealed no abnormalities, clarifying that trauma had caused the hemorrhage. Metabolic disorders were excluded (investigations included routine hematology and biochemistry, lysosomal enzymes, and urinary vanillylmandelic and organic acids). Psychometry, including the Behavioural Assessment of the Dys-



**Fig. 2** Magnetic resonance imaging scan showing hemorrhage along the anterolateral aspect of the right putamen.

executive Syndrome (Wilson et al., 1996), showed subtle signs of a dysexecutive syndrome with difficulties in tasks involving planning and organization.

Before discharge A.B. experienced short-lived episodes of low mood reflecting increased insight into her illness and concern about weight increase. Discharge recommendations were made with respect to continuing carbamazepine and chlorpromazine under neuropsychiatric review, individual and family work, and minimizing initial academic pressures, in order to reduce the risk of relapse of psychosis, depression, and anorexia.

## DISCUSSION

Before we discuss the case, we will highlight some of the methodological difficulties inherent in this area. Many studies fail to control for preinjury factors, despite evidence that children with externalizing behavioral disturbance are more likely to sustain a head injury (Chadwick, 1985), and few studies involve non- or other-injured controls. According to the severity criteria used by Max et al. (1998), A.B. would be considered to have a mild traumatic brain injury (i.e., a Glasgow Coma Scale [Teasdale and Jennett, 1974] score of at least 13 and a normal brain

CT scan), and the posttraumatic amnesia was also relatively short. However, a severe mental illness followed.

There are difficulties in extrapolating to children findings from the adult literature, especially in relation to types of injury and forces involved. The etiology of head injuries and subsequent mortality varies with age, which has implications for the interpretation of pathological findings (Livingston and McCabe, 1990; Sollee and Kinson, 1987). It is also possible that there is a differential impact on emerging cognitive abilities and personality according to age at the time of injury (Satz et al., 1997).

Although the psychiatric outcome seems more variable than the cognitive outcome (Chadwick, 1985), there is little doubt that moderate or severe head injury is associated with an increased risk of psychological disturbance (Brown et al., 1981; Chadwick, 1985; Max et al., 1997b; Rutter et al., 1980). The sequelae of mild head injury in children are more controversial. A review of mild head injury in children reported that two thirds of studies failed to demonstrate any disability, and methodologically "strong" studies were associated with no subsequent morbidity (Satz et al., 1997, p. 123). In A.B.'s case, the psychological and physical features in the 3 weeks following the initial head injury suggest either a postconcussional disorder or a major depressive episode. Her presentation after the traffic accident involving the family had features suggestive of a delirium with perceptual abnormalities. Despite the lack of impact of this second accident, the acceleration/deceleration forces involved may have had a cumulative effect with the initial head injury. The subsequent psychotic illness with fear and perplexity was unusual in that it developed out of a confusional state, but then relapsed despite the confusional state improving. The characteristic features of a manic illness and the episodes being preceded and followed by depressive symptoms led to a diagnosis of bipolar disorder.

The range of disorders seen after head injury is similar to those in noninjured children, with a predominance of conduct and emotional disorders (Brown et al., 1981; Chadwick, 1985; Max et al., 1997a,b; Oddy, 1993; Rutter et al., 1980), although there is an excess of hyperkinetic disorder and socially disinhibited behavior (Chadwick, 1985). Very few cases of mania occurring secondary to a head injury in children or adolescents have been reported (Cohn et al., 1977; Joshi et al., 1985; Khanna and Srinath, 1985; Max et al., 1997c).

Evidence suggests that in adulthood, secondary mania is associated with the full range of head injury and has a similar course to primary mania, but that there may be a

higher relapse rate and irritability is commonly the predominant affect (Jorge et al., 1993; McAllister, 1992). One series of 20 patients presenting with mania after closed head injury reported that 50% had clinical seizures (Shukla et al., 1987). In A.B.'s case, investigations revealed no evidence of seizures, although her clinical presentation was highly suggestive of seizures. The olfactory hallucinations may have represented subclinical seizures, perhaps involving irritation of the mesial temporal cortex.

There are lower rates of family and past psychiatric disorder in secondary mania compared with primary mania or secondary depression (Jorge et al., 1993; Starkstein et al., 1987). The former is associated with injury to the right hemisphere, in particular to the limbic system or related structures (Jorge et al., 1993; Robinson et al., 1988; Starkstein et al., 1987, 1990). A.B.'s injury is in keeping with this. One study of secondary mania found a high incidence of frontal subcortical atrophy in those without a family history (Robinson et al., 1988). A greater depletion of biogenic amines has been demonstrated after insults to the right rather than the left hemisphere (Starkstein et al., 1990). Secondary mania may be rare because it requires the unlikely combination of genetic loading or anterior subcortical atrophy with greater amine depletion (Starkstein et al., 1990). For the case described, the close temporal proximity of the head injury as a risk factor and the absence of family history of affective disorder suggest a relatively good prognosis.

The case described emphasizes the need for clinical management to be guided by clinical features in preference to investigations with normal results. Early contusions following a head injury may not be seen on CT scanning, and a normal CT and EEG do not rule out an organic contribution to the etiology.

While the severity of the injury seems the most important predictor of psychiatric disorder (Max et al., 1997a), pre- and postinjury intellectual function, the child's and the family's psychiatric history, and current psychosocial circumstances are risk factors (Brown et al., 1981; Max et al., 1997a; Rutter et al., 1980). Follow-up studies suggest that difficulties fluctuate predictably over time (Oddy, 1993). Subtle cognitive deficits may combine with behavioral problems, leading to significant educational and social handicap, despite the preservation of global intellectual function (Chadwick, 1985; Klonoff et al., 1993). "In other words, it is not only the kind of injury that matters, but the kind of head" (Symonds [1937] ref. in McAllister [1992], p. 399).

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**Traumatic Brain Injury in High School Athletes.** John W. Powell, PhD, ATC, Kim D. Barber-Foss, MS, ATC

**Context:** The potential seriousness of mild traumatic brain injury (MTBI) is increasingly recognized; however, information on the frequency of MTBI among high school athletes is limited. **Objective:** To identify the type, frequency, and severity of MTBI in selected high school sports activities. **Design:** Observational cohort study. **Setting and Participants:** Two hundred forty-six certified athletic trainers recorded injury and exposure data for high school varsity athletes participating in boys' football, wrestling, baseball and field hockey, girls' volleyball and softball, boys' and girls' basketball, and boys' and girls' soccer at 235 US high schools during 1 or more of the 1995-1997 academic years. **Main Outcome Measures:** Rates of reported MTBI, defined as a head-injured player who was removed from participation and evaluated by an athletic trainer or physician prior to returning to participation. National incidence figures for MTBI also were estimated. **Results:** Of 23 566 reported injuries in the 10 sports during the 3-year study period, 1219 (5.5%) were MTBIs. Of the MTBIs, football accounted for 773 (63.4%) of cases; wrestling, 128 (10.5%); girls' soccer, 76 (6.2%); boys' soccer, 69 (5.7%); girls' basketball, 63 (5.2%); boys' basketball, 51 (4.2%); softball, 25 (2.1%); baseball, 15 (1.2%); field hockey, 13 (1.1%); and volleyball, 6 (0.5%). The injury rates per 100 player-seasons were 3.66 for football, 1.58 for wrestling, 1.14 for girls' soccer, 1.04 for girls' basketball, 0.92 for boys' soccer, 0.75 for boys' basketball, 0.46 for softball, 0.46 for field hockey, 0.23 for baseball, and 0.14 for volleyball. The median time lost from participation for all MTBIs was 3 days. There were 6 cases of subdural hematoma and intracranial injury reported in football. Based on these data, an estimated 62 816 cases of MTBI occur annually among high school varsity athletes participating in these sports, with football accounting for about 63% of cases. **Conclusions:** Rates of MTBI vary among sports and none of the 10 popular high school sports we studied is without the occurrence of an MTBI. Continued involvement of high school sports sponsors, researchers, medical professionals, coaches, and sports participants is essential to help minimize the risk of MTBI. *JAMA* 1999;282:958-963. Copyright 1999, American Medical Association.