CASE REPORT

A punch drunk jockey?

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The case is reported of a retired professional jockey with progressive memory loss. The concern is that he may be suffering from chronic traumatic encephalopathy or the ''punch drunk syndrome''.

Although the bulk of patients reported to have chronic traumatic encephalopathy are boxers, it has been anecdotally suggested that jockeys, particularly jump jockeys, may develop this condition because of the high rates of concussive brain trauma seen in this sport.[1–3]

One of the practical difficulties in neurological practice is where a patient presenting with cognitive decline gives a history of recurrent sport related head injury such as seen in a retired boxers, jockeys, and soccer players. In such situations, it is often assumed that the ''punch drunk syndrome'' is the basis for the symptoms rather than other far more likely causes of dementia such as Alzheimer's disease. This case illustrates the diagnostic difficulties in assessing such patients.

CASE HISTORY

The patient is a 48 year old retired professional jockey suffering from a three year history of progressive short term memory loss. There are no reported speech problems, balance disturbance, behavioural change, tremor, or other neurological symptoms.

His race riding career began at the age of 15 in point to point events. Before that, he was a show jumper. In 1970, he rode his first winner on the flat and continued to have a successful amateur career with a total of 23 winners. He turned professional at the age of 18 and continued to ride until the age of 35 averaging 200–250 rides a year. In his professional career, he won a total of 250 races including two at Cheltenham and was placed 5th in the Grand National.

During his racing career, he had a number of orthopaedic injuries, including seven fractured clavicles, an occupational hazard for jockeys. With regard to head injuries, he had a major concussion at the age of 16 when he was thrown from a horse, with the horse landing on top of him. He states that he was unconscious for 24 hours and he remained in hospital for one week because of a coexistent pneumothorax. He was unable to return to race riding for one month thereafter, although continued to school horses on the gallops (''riding work'') during this time. He has had 10–12 admissions to hospital for concussions. These injuries would generally be classified as a Cantu grade 1 or 2 injury. In this sport, jockeys with mild concussion are monitored in the ''jockeys' medical room'' located on the racecourse, unlike the situation in other sports where such patients would not be admitted or monitored. His last race fall was in 1988 when he was unconscious for two to three hours, and a computed tomographic brain scan at the time was reported as showing a brain contusion.

At the present time, he works as a farmer and trains about 20 horses for point to point racing. There is no family history of neurological disease or dementia.

EXAMINATION

Neurological examination was normal.

COGNITIVE ASSESSMENT

A variety of neuropsychological tests were performed including both ''pen and paper'' tests and the CogSport computerised neuropsychological test battery. Table 1 summarises the results.

Overall he has slow processing speeds and reaction times, with impaired short term memory but relatively intact working memory. No evidence of gross word finding difficulties, visuospatial processing problems, or perseveration was present.

A CogSport computerised assessment showed an impaired performance that was greater than two standard deviations below the normal range on tests of psychomotor speed, decision making speed, problem solving speed, memory speed, psychomotor accuracy, memory accuracy, psychomotor consistency, decision making consistency, and problem solving consistency.

IMAGING STUDIES

Magnetic resonance (MR) brain scan (figs 1 and 2) and MR angiogram revealed mild ventricular enlargement of the lateral ventricles and the supratentorial subarachnoid spaces. A cavum septi pellucidi was present. No forniceal or hippocampal atrophy was found, and no haemosiderin deposition was present to suggest previous haemorrhage.

GENETIC TESTING

ApoE testing revealed a 3/3 phenotype. This indicates both a low risk of poor outcome from head injury and a reduced risk of Alzheimer's disease compared with patients with the ApoE4 phenotype (see below).

DISCUSSION

This jockey has a history of 10–12 concussive injuries in his 20 years of amateur and professional riding. During his career, he reported no continuing neurological symptoms, but he developed progressive short term memory loss about 10 years after retirement. At no stage has he exhibited any neurological signs such as dysarthria, tremor, or gait difficulties. His ApoE phenotype would place him at low risk of chronic neurological injury, and his neuropsychological profile is consistent with an isolated short term memory impairment associated with slowed reaction times and processing speeds. The presence of memory loss would be consistent with early Alzheimer's disease. However, this is not typically accompanied by slowed reaction times or information processing deficits. His MR brain scan shows cerebral atrophy with no other diagnostic features. The clinical question central to his case is whether he is developing a chronic traumatic encephalopathy (punch

Table 1 Neuropsychological test results

Test	Score	Centile
Mini mental state examination	24/30	–
Reaction time (ms)	0.747	–
Silly sentences	3	1
Digit symbol	5	5
Colour trails	73	4
Stroop	73	2
Wechsler memory scale		
Auditory immediate	65	1
Visual immediate	81	10
Auditory delayed	61	0.5
Visual delayed	65	1
Working memory	111	77

drunk syndrome) or whether the history of trauma is incidental and that he is in the early stages of a dementing illness such as Alzheimer's disease or frontotemporal dementia. In some situations, alternative functional neuroimaging techniques such as positron emission tomography (PET) scanning may be of use to differentiate such conditions, although ultimately a pathological diagnosis is the only way of confirming such diagnoses. It will probably be a number of years before the cause is formally established in this case.

Concern is often raised in such situations that malingering may in part explain the cognitive abnormalities seen in professional sportsmen after head injury. This may be a particular concern when insurance or medicolegal considerations apply. This has been specifically examined with computerised test batteries such as Cogsport, and the findings in this case do not indicate that this is an important concern.[4 5]

The punch drunk syndrome

In 1928, Harrison Martland[6] first described a syndrome in prizefighters that was known in lay boxing circles as the "punch drunk" or "slug nutty" state. In the mildest cases, the most common presenting symptom is slurring dysarthria, which is seen in 90% of cases and is often accompanied by a gait ataxia or disequilibrium. In the later stages, cognitive impairment becomes the major neurological feature. Throughout the course of the condition, various neuropsychiatric and behavioural symptoms may occur. Typically there is a lack of insight by the affected boxer with regard to the development of specific problems.[7–9] About one third of cases are progressive in nature.

Neuropsychological studies of boxers suffering from chronic boxing related neurological injury have typically found difficulties in memory, information processing and speed, finger tapping speed, attention and concentration, sequencing abilities, and frontal executive functions such as judgment, abstraction, reasoning, planning, and organisation.[10–12]

Modern neuroradiological imaging techniques have been unsuccessful to date in producing any systematic evidence of brain injury in boxers. Early anecdotal reports using pneumoencephalography and computed tomography have not been validated using newer MR technologies.[13] The presence of a cavum septi pellucidi alone is insufficient to entertain the diagnosis of the punch drunk syndrome.[14]

Factors other than simple trauma may have a causative role in dementia pugilistica. The ApoE4 phenotype has been associated with an increased risk of chronic boxing related neurological injury in boxers.[15–18] In a non-boxing population, the ApoE4 polymorphism has also been shown to be significantly associated with death and poor outcomes following all levels of acute traumatic brain injury.[19–21]

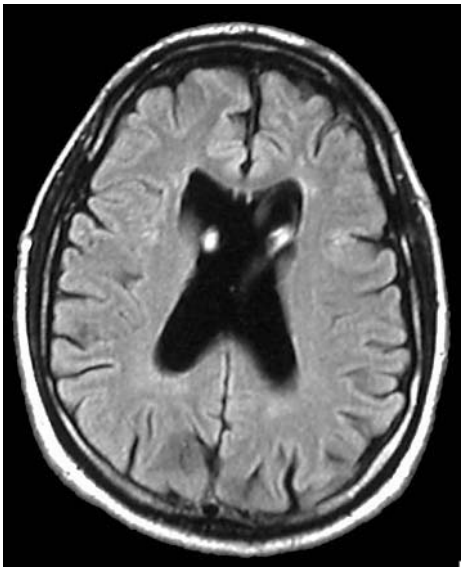

Figure 1 Axial T1 magnetic resonance brain scan showing mild cerebral atrophy and ventriculomegaly.

Do jockeys get punch drunk?

Corsellis *et al*[2] reported the results of an informal survey of neurologists and neurosurgeons in the United Kingdom in which several jockeys were reported to be suffering from the punch drunk syndrome. No further details were provided on these cases. Foster *et al*[1] anecdotally reported several cases of brain injury in national hunt (jump) jockeys. These findings were not supported by the more recent prospective study of injuries in professional horse racing in which no jockeys were found to be suffering from, or retired because of, the punch drunk syndrome.[3]

Given the current screening system in place in the United Kingdom by the Jockey Club whereby all jockeys are annually

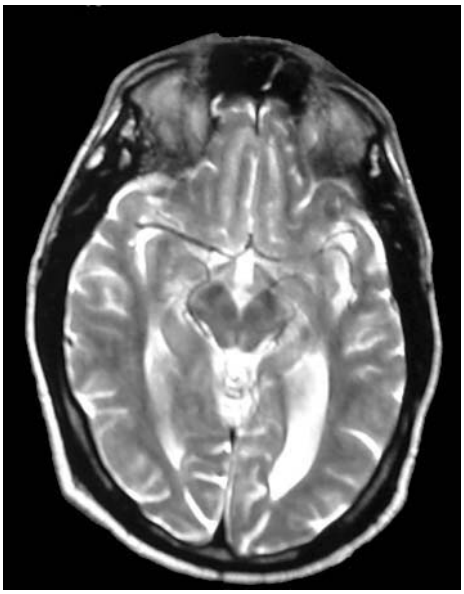

Figure 2 Axial T2 magnetic resonance brain scan showing normal midbrain size and normal substantia nigra appearance.

screened for the development of neurological problems with neuropsychological test batteries, it is likely that any jockey at risk of developing neurological problems will be detected before any permanent damage ensues. Whether subsequent deterioration after retirement may be prevented requires prospective validation.

CONCLUSION

In this case, there is no convincing evidence that this retired jockey is suffering from the punch drunk syndrome. He does not exhibit any of the typical neurological or neurobehavioral symptoms of this condition and his genetic phenotype would put him at low risk of the syndrome. He clearly has a progressive short term memory impairment associated with radiological evidence of cerebral atrophy. At this stage, his diagnosis is unclear and it may be a number of years before the cause can be formally established.

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REFERENCES

1 **Foster JB**, Leiguarda R, Tilley PJ. Brain damage in national hunt jockeys. *Lancet* 1976;**i**:981–3.
2 **Corsellis JA**, Bruton CJ, Freeman-Browne D. The aftermath of boxing. *Psychol Med* 1973;**3**:270–303.
3 **Turner M**, McCrory P, Halley W. Injuries in professional horse racing in Great Britain and the Republic of Ireland during 1992–2000. *Br J Sports Med* 2002;**36**:403–9.
4 **Collie A**, Maruff P. Computerised neuropsychological testing. *Br J Sports Med* 2003;**37**:2–4.
5 **Collie A**, Maruff P, Makdissi M, *et al*. Reliability and correlation with conventional cognitive tests used in post concussion medical examinations. *Clin J Sport Med* 2003;**13**:28–33.
6 **Martland HS**. Punch drunk. *JAMA* 1928;**19**:1103–7.
7 **Roberts AH**. *Brain damage in boxers: a study of the prevalence of traumatic encephalopathy among ex-professional boxers*. London: Pitman, 1969.
8 **Jordan B**, ed. *Medical aspects of boxing*. Boca Raton: CRC Press, 1993.
9 **Jordan BD**. Boxing. In: Jordan B, Tsaris P, Warren R, eds. *Sports neurology*. Philadelphia: Lippincott-Raven, 1998:351–67.
10 **Casson I**, Siegel O, Sham R, *et al*. Brain damage in modern boxers. *JAMA* 1984;**251**:2663–7.
11 **Levin HS**, Jordan BD. Neuropsychological assessment of brain injury in boxing. In: Jordan BD, ed. *Medical aspects of boxing*. Boca Raton, FL: CRC Press, 1993:197–206.
12 **Roberts GW**, Allsop D, Bruton C. The occult aftermath of boxing. *J Neurol Neurosurg Psychiatry* 1990;**53**:373–8.
13 **Moseley IF**. The neuroimaging evidence for chronic brain damage due to boxing. *Neuroradiology* 2000;**42**:1–8.
14 **McCrory P**. Cavum septi pellucidi: a reason to ban boxing? *Br J Sports Med* 2002;**36**:157–62.
15 **Nicholl J**, Roberts G, Graham D. Apolipoprotein E4 allele is associated with deposition of amyloid beta protein following head injury. *Nat Med* 1995;**1**:135–7.
16 **Jordan BD**, Relkin NR, Ravdin LD, *et al*. Apolipoprotein E epsilon4 associated with chronic traumatic brain injury in boxing. *JAMA* 1997;**278**:136–40.
17 **Jordan B**. Genetic susceptibility to brain injury in sports: a role for genetic testing in athletes? *Phys Sportsmed* 1998;**26**:25–6.
18 **Jordan BD**. Apolipoprotein Ee4 and fatal cerebral amyloid angiopathy associated with dementia pugilistica. *Ann Neurol* 1995;**38**:698–9.
19 **Friedman G**, Froom P, Sazbon L, *et al*. Apolipoprotein E-epsilon 4 genotype predicts a poor outcome in survivors of traumatic brain injury. *Neurology* 1999;**52**:244–9.
20 **Teasdale G**, Nicol J, Murray G. Association of Apolipoprotein E polymorphism with outcome after head injury. *Lancet* 1997;**350**:1069–71.
21 **Liberman J**, Stewart W, Wesnes K, *et al*. Apolipoprotein E e4 and short term recovery from predominantly mild brain injury. *Neurology* 2002;**58**:1038–44.