After head injury, obsessive compulsive disorder has been rarely reported. Only 22 cases could be found after an extensive literature search. The largest is a series of 14 out of a sample of 415 (Hillbom 1960). Two earlier reports described 4 more cases. (Lishman 1968 and Thompson 1965). A recent prospective study found history of head injury in 4 cases of obsessional disorder (McKeown et al 1984). Hillbom (1960) observed that unlike other psychosyndromes this one was directly related to the severity of the head injury. Several of the patients had depersonalisation and two thirds also had epilepsy. He thought this disorder occurred when the patients were able to struggle against their disability to preserve former standing.

Case Report

R, 18 year male, presented with a two year history of repeated actions and ideas. There was no family history of obsessive compulsive disorder or obsessional personality. There was no past history of psychosomatic complaints, peptic ulcer, constipation or dyspepsia.

After a bus accident 5 years earlier, the patient was unconscious and had a left occipital depressed fracture for which debridement and excision of bone fragments was done. Later he developed meningitis which was treated with the appropriate antibiotics. As the cerebellum continued to bulge, the part of it which was extruding was excised. Due to persistently raised intracranial pressure L2 laminectomy and thecoperitoneal shunt were done. About ten weeks after the head injury the patient had 3 generalised convulsions. Post traumatic amnesia lasted for 4 days. After that he had forgetfulness for recent events which subsided gradually. Three years after the accident the current problems started.

At this time neuropsychological evaluation had been done for legal purposes. It revealed gross dysfunction of cerebral lobe function indicating general and focal involvement of frontal, temporal and parietal lobes.

Since this time the patient was observed to be repeating certain daily activities, like opening and closing books, and picking and placing down a pen. While doing some other activity he would suddenly get a doubt that he had not done the act properly or that something was not in the proper place. These thoughts were repetitive and intrusive. He used to regard them and the resultant acts as senseless but attempts to resist them were associated with anxiety. He also started getting doubts that various things may enter his head through the body defect that he had. These thoughts used to occur in clear consciousness against his will. When presented with another task or problem, he would become irritated but would attend to it.
Mental status examination revealed a young male with obsessions and compulsions with impaired abstraction and preserved insight. On physical examination he had a body defect in the left occipital region, measuring 7 cms x 6 cms with irregular margins through which a pulsatile mass covered with skin protruded.

Psychometry was suggestive of organicity. Repeat neuropsychological assessment revealed that most of the frontal lobe functions were impaired. Apart from minimal impairment in learning and memory, all other temporal lobe functions were normal. Left parietal lobe functions showed minimal impairment. Compared to the previous deficit, the patient had showed remarkable recovery in his cognitive functions.

CT scan showed evidence of left occipital craniectomy. There was gliotic cavity in the left cerebellum and porencephaly in the left occipital horn.

Power spectral EEG analysis revealed that the powers of delta, theta, beta 1 and beta 2 ranges were approximately twice in the left parietal leads as compared to the right. In the temporal leads this increased power on the left side was apparent in the beta 1 and beta 2 ranges. There was reduced alpha power in the left parietal lead which could be due to the head injury. The frontal leads showed diminished power in all ranges, although there was no indication of asymmetry here.

In evoked potential studies, photic stimulation and checker board pattern stimulation showed inversion of P100, N140 and P180 components on the left side. Evoked potentials to tone bursts were normal on both sides.

This patient has been followed up for a period of two and a half years. Initially he had not shown adequate response to amitriptyline 300 mg for 3 months, imipramine 250 mg for 3 months and trifluoperazone 10 mg daily for 4 months. His subsequent response to amphetamine has been reported earlier (Khanna and Janakiramaiah 1984). The patient has continued to show sustained improvement and has not developed any other psychopathology during this period.

Discussion

This case presents with the interesting problem of having both post-traumatic sequelae and a psychiatric syndrome, namely obsessive compulsive disorder. It has been stated that reactive changes in the brain are completed within two years (Bay 1953). In this case the patient showed gradual improvement as evidenced by neuropsychological evaluation 3 and 5 years after the head injury. On the background of this diffuse dysfunction the appearance of a new psychiatric syndrome, which worsened while the organic state improved seems to be secondary to the injury. It is always possible that the occurrence of obsessional disorder in this case was coincidental.

The phenomenon observed here is intrusive, repetitive, resisted and regarded as senseless, and occurs in clear consciousness. By any criteria, it meets the definition for an obsession (American Psychiatric Association 1980). Its similarity to perseveration is however noteworthy. Perhaps frontal lobe dysfunction is common to both, but whereas in the former there is inability to change set, in obsessions it is not so.

The patient had had a head injury, meningeal infection and surgical intervention. However the surgical intervention predominantly involved a shunt, closure of the defect and excision of the protruding cerebellum; as such its etiological role is unlikely. Infection after open head injury is common, and a complication of the same. As such we feel that the syndrome can be regarded as being secondary to the head injury per se.
The power spectra EEG analysis showed a preponderance of left hemispheric activity, except in the frontal lobes where there was diminished power in all the ranges. Inversion noted in photic evoked potentials was probably due to the head injury as it was recorded across the P4-CZ-P3 leads.

There have been some reports earlier implicating the frontal lobe in obsessional illness (Minski 1933, Flor Henry et al 1979). We feel that to some extent our case provides some more support to this hypothesis. To conclude, an unusual case where an obsessional disorder developed after head injury has been presented and the diagnosis discussed.

References

AMERICAN PSYCHIATRIC ASSOCIATION, (1980), Diagnostic & statistical manual for mental disorders, 3rd edition, Washington, APA Press.

BAY, E. (1953), Die traumatische Hirnschädigung, In Handbuch der Inneren Medizin vol. 5, Berlin Springer, 373-342.

FLOR HENRY, P., YEUDALL, L. T., KOLES Z. J. & HOWARTH, B. G. (1979), Neuropsychological and power spectral EEG investigations in obsessive compulsive syndrome. Biological Psychiatry, 14, 119-130.

HILLBOM, E. (1960), After effects of brain injuries. Acta Psychiatrica et Neurologica Scandinavica (Suppl), 142, 1-95.

KHANNA, S. & JANAKIRAMAIAH, N. (1984), D-amphetamine in obsessive compulsive disorder, Pharmabulletin, 91, 204.

LISHMAN, A. (1968), Brain damage in relation to psychiatric disability after head injury. British Journal of Psychiatry, 114, 373-410.

McKEON, J., McGUFFIN, P. & ROBINSON, P. (1984), Obsessive compulsive neurosis after head injury: a report of 4 cases, British Journal of Psychiatry, 144, 190-192.

MINSKI, L. (1933), The Mental symptoms associated with 58 cases of cerebral tumor. Journal of Neurology and Neuropathology, 13, 330-343.

THOMPSON, G. N. (1965), Post traumatic neurosis: a statistical survey. American Journal of Psychiatry, 121, 1043-1048.