REPEATED HEAD TRAUMA – DECISIVE FACTOR OF DELAYED ONSET EPILEPSY IN BOXERS

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Summary

The repeated application of blows to the boxer's head result in chronic brain lesion known as "punch drunkenness", "chronic progressive traumatic encephalopathy in boxers", "dementia pugilistica" and "psychopathic deterioration of pugilists". Morphological findings of late traumatic damage in boxers include abnormalities (cavitation or rupture) of the septum pellucidum, focal scarring of the cerebral and cerebelar hemispheres, degeneration of the substancia nigra, of the temporal lobe and the hypothalamic structures. One of the sequalae of repeated blows to the boxers' head is the increased convulsive predisposition and the occuring of epileptic seizures.
Clinical analysis of epilepsy in boxers, in whom history does not reveal any other etiological factors than chronic craniocerebral trauma, was performed.
A group of 9 boxers suffering from trauma-induced epileptic seizures (ES) was examined. The age of boxers ranged from 21 years to 29 years.
Neurological examination does not reveal any abnormality. Psychiatric changes include decreased memory and emotional instability. CT scan was normal in 4 patients and shows the initial cerebral atrophy in 5 patients. The interictal EEG in all patients revealed diffuse abnormality with irregular spike and wave complexes. Generalized seizures of Grand Mal type were identified in 8 boxers (88%), partial seizures with complex symptomatology of temporal lobe - in one boxer. Regarding the onset of boxing, 6 boxers (66,7%) had their first epileptic seizures within two years, two patients within 3 years and one patients develop his first ES within one year after the onset of boxing.
The younger the boxer, the higher the probability of epilepsy. Most frequently generalized tonic-clonic epileptic seizures occurred - usually two years after the onset of boxing. The occurrence of epileptic seizures in boxers following a brain injury depends upon individual predisposition and upon the frequency and severity of repeated head trauma. The possibility of the repeated blows to the head to create potential epileptics arises many serious questions of great importance for the heath of thousands of young people.

Key words: repeated head trauma, boxers, epilepsy

Introduction

Boxing is the sport where repeated trauma to the brain is inevitable. The cumulative effect of multiple blows to the boxer’s head results in chronic brain lesion known as ‘’punch drunkness’’, ‘’dementia pudgilistica’’ (‘’punch drunk syndrome’’), ‘’psychopathic deterioration of pugilists’’ and ‘’chronic progressive traumatic encephalopathy of boxer’’ (6). The brain trauma in sports and particulary the blows to the head can provoke epileptic seizures (2,4,5,7,8).

Early sequelae of trauma in boxers include depressed fractures of the skull, intracranial hemorrhage, lesions of the dura matter, long periods of posttraumatic amnesia and early-onset epileptic seizures (3). Late sequelae of boxing trauma mostly include the occurrence of delayed onset epilepsy due to focal cerebral scarrings, parkinsonismus (cells loss in substancia nigra) and dementia, with predominantly impairment of short-term memory, due to the lesions of the medial temporal lobe structures (mainly of the hippocampus and their hypothalamic connections) (3,6).

In the previous report we showed in experiments in rats thet repeated head blows of the lesser intensity, when applied in close succession cause increased convulsive predisposition and increased incidence of seizures (6).

In present study the role of the repeated head trauma in boxers resulting in late-onset epilepsy is discussed.

This study concerns young boxers' epilepsy of delayed onset in which history does not reveal any other etiological factors than repeated craniocerebral trauma as a result of multiple blows to the boxer's head!

Patients and Methods

Nine boxers with epilepsy aged from 21 to 29 years were examined as out-patients in Military Medical Academy in Sofia during the period 2003 – 2006. Somatic, neurological and psychiatric statuses were examined. EEG and CT scan were performed. There was no family of epilepsy in all patients.

Results

Somatic and neurological examination of all patients does not reveal any abnormality. Psychiatric changes include increased emotional instability. CT scan was normal in 4 patients and shows the initial cerebral atrophy in 5 patients. The interictal EEG of all patients revealed diffuse abnormalities with irregular spike and wave complexes, spike and sharp wave complexes or a mixture of these.

The onset of epileptic seizures (ES) after the onset of boxing shows that six patients (66, 7%) had their first ES within 2 years, two patients (22, 2%) within 3 years and one patient (11, 1%) develop his first ES within 1 year after the onset of boxing. The type of epileptic seizures shows that eight boxers (88, 9%) had generalized epileptic seizures of Grand mal type and one boxer (11, 1%) had partial seizures with complex symptomatology (psychomotor ES with oro-alimentary automatisms). The epileptic seizures occurred at intervals of 3-4 months in 2 patients with Grand mal, at intervals of 5-6 months in 2 patients with Grand mal and yearly in 4 patients with Grand mal and 1 with psychomotor epileptic seizures.

Discussion

The distribution of posttraumatic cerebral lesions shows that most injuries occur in the fronto-temporal regions. The abrupt forward and backward movements of the head by direct blows result in sudden displacement of the brain against the cranial cavity with subsequent injury most often of the poles and bases of frontal and temporal lobes. It should be stressed that the ‘’contacoup effect’’ is also responsible for contusions in these structures (6, 7). The brain injuries are very often of multifocal character (7). The site and the size of the concussion focus may vary both on the surface and in the deep brain structures. The effect of head trauma causes disturbances in the brain stem as in the hemispheres (7, 9).

In the head of boxer widespread micro-haemorrhages and brain edema start on the concussion. At cellular level the damage of blood vessels and the glia cells can be seen. But the most important degenerative process and functional damage were found in the dendrites of neurons and in the synapses (10). Electron microscopy studies of the cortex show that many presynaptic terminals and synaptic vesicles had disappeared and dendrites with a watery appearance and lack of neurotubules can be seen (10). The deficiency of vesicles in the synapses might be accounted for by profuse consumption of neurotransmitters (10). These alternations in synapses and in dendrites deprive the cell of connections and lead to neuronal hyperexitability. It should be stressed that the alternation in synapses and dendrites preceded the appearance of degenerative changes in neuronal cell bodies and axons (10). These findings can explain the fact that ES in some cases can precede the appearance of neurological symptomatology.

Generally post-traumatic epilepsy (PTE) is divided in two categories: PTE of early onset and PTE of delayed onset. PTE of early onset results in epileptic seizures occurring within the first week of the trauma (3, 4). Risk factors are post-traumatic amnesia of more than 24 hours duration, intracranial haematoma, a depressed fracture, and neurological signs. PTE of delayed onset results in epileptic seizures occurring more than one week after the trauma (3, 9). The boxing epilepsy is assigned to this category. The late morphological findings in the boxer's brain show degeneration of the substantia nigra, focal scarring of cerebral and cerebellar hemispheres (loss of cells with fibers demielinisation), degeneration of the medial temporal lobe structures (most commonly the hippocampus), abnormalities of the septum pellucidum (cavitation or rupture) and degeneration of the hypothalamic structures (mainly the nucleus supraopticus and other nuclei) (6). Impaired metabolism of the cerebral tissue is considered conductive to posttraumatic cerebral atrophy and dementia. Cerebro-meningeal scars due to brain damage accounts for most cases of posttraumatic epilepsy in boxers. It has been found that it is the cortex in the vincinity of the scar, rather than the scar itself, which is the source of epileptic discharges. It should be remembered that it is quite possible the development of a secondary brain ("mirror") foci after a period of time (7, 8).

New studies showed that repeated blows to the head cause an increase levels of three chemicals in the cerebrospinal fluid of boxers: neurofilament light protein, total tau protein and glial fibrillary protein (1). Compared with nonboxers it was found that the boxers had higher levels of neurofilament light protein and glial fibrillary protein immediately after a bout (1). Three months later the boxers still had higher levels of neurofilament light protein than the control group (1). These three chemicals can be used as markers of brain injury and damage to nerve cells of the brain (1).

Conclusions

Repeated head blows to the head lead to increased convulsive predisposition. Most frequently ES occurring in boxers are the generalized tonic-clonic ES. The occurrence of ES in boxers depends upon the age, the frequency and severity of blows to the head. The early onset of boxing, the early occurrence of epilepsy. The younger the boxer, the higher the probability of occurrence of seizures. The purpose of every sport is to prolong the years of life in either man and to create healthy man. The possibility of the repeated head trauma to create potential epileptics arise many serious questions of great importance for the health of thousands of young people.

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