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Review Article

Punch Drunk Syndrome – Chronic Traumatic Encephalopathy: Physiotherapy Approach

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Abstract

This Study presents a summary of current scientific data and concepts around Chronic traumatic encephalopathy (CTE). CHT is a progressive neurodegenerative condition caused by repetitive damage to the brain, including concussive and sub – concussive injuries. It was first described by Martland in 1928 as "The Punch – Drunk syndrome". CTE is closely associated with athletes participating in contact sports, such as boxing. The acceleration and the collisions of brain hemispheres in the walls of the dome of the skull can cause severe damage, as a result of punches in the head or head hits on the floor. Currently, prevention and education are the most effective way to combat CTE and should be emphasized by doctors and athletes. However, the existing scientific literature lacks suggestions of a treatment approach. In This study, the clinical presentation of the disease and the development of its symptoms is presented. The information gathered was used to design a proposed physiotherapy assessment and treatment plan. However, further research is needed to aid in the creation of clinical diagnostic criteria and finding objective biomarkers for the disease. Most importantly a need to identify evidence based approaches on treatment and support of individuals and their families living with CTE.

Keywords: Chronic Traumatic Encephalopathy; Boxing; Concussion; Traumatic Brain Injury; Knock Out

Introduction

The concept of the HTE was first presented by Martland in 1928, who introduced the term "drunken by fist" (Punch-drunk) or otherwise the "Boxer syndrome", as a set of symptoms that seemed to be the result of Repeated blows of high power to the head. It is a neurodegenerative disease consisting of exopyridal and cerebellum signs and symptoms, related to cognitive and behavioral abnormalities. They appear, initially, disturbances in attention, concentration and memory, as it is seen both disorientation and confusion, while occasionally accompanied by dizziness and headaches. With progressive deterioration there are obvious additional symptoms, such as lack of insight, impaired judgment and apparent dementia. In a large part of sufferers, it resembles [1,2]. Alzheimer's disease, showing parallel signs and symptoms of parkinsonism [3].

The purpose of this review is to present modern data and perceptions about the HTE, emphasizing the sport of boxing. In addition, a proposed plan of Conservative-physiotherapy evaluation and treatment, which aims at managing the symptoms of the disease and delaying its progress, is listed.

Epidemiology

The concussion often occurs in contact sports. 1.6-3.8 cm concussions related to sports are noted annually in the United States. Most related blows to the head are minor and although the majority of athletes who suffer from a concussion recover within a few days or weeks, a small number of people develop long-term or progressive Worsening symptoms. This is particularly true in cases of repeated concussion or mild traumatic brain damage in which at least 17% of people develop THE [2].

A repeated closed head injury occurs in a wide variety of contact sports, such as football, boxing, wrestling, rugby, hockey, lacrosse, and skiing. Other sports related to post-concussion syndrome include hockey, karate, horseback riding and parachute, although the list is almost certainly more comprehensive. In addition, additional large groups of people prone to repeated blows to the head, such as military veterans, may be at risk of displaying the HTE [2,3].

Abuse mechanism-RISK factors

Boxing is the only sport in which causing intentional acute traumatic damage to the opponent's brain through Knockout (KO) is accepted as a legitimate goal in a match. The speed of the fist on impact with the head can be 10m/s or higher. The force increases depending on the charge (according to the weight) up to more than 5000 Newton's, resulting in the transfer acceleration of the opponent's brain over 50G. The acceleration and collisions of cerebral hemispheres in Walls of the dome of the skull can cause serious damage as a result of fists on the head or blows of the head on the floor [4].

The severity of acute cranibrain injury (KEK) can range from mild to severe. Mild traumatic brain injury (Mild traumatic brain Injury) is the most common FORM of acute kek found and is often synonymous with the term "cerebral concussion". This lesion can lead to a variety of natural, psychosocial and cognitive symptoms, and when these deficits are not resolved, it can lead to post-concussion syndrome. In some cases, post-concussion syndrome may persist for months or years after the initial injury. However, it is believed that no more than 15% of people with mild KEK history still face some of the symptoms one year after injury [5].

One of the key features of the HTE is that the disease continues to evolve for decades after the cessation of activity in which traumatic injury has taken place. It is most likely that multiple pathological procedures continue to exert their effect throughout the life of the individual since the recurrent trauma was caused. The greater the survival after the initial episodes and the more serious the initial injuries, the greater the severity of degeneration [2].

The Stern and Riley in one investigation observed that the neuropathologically confirmed cases that had a history of brain exposure to trauma did not develop all HITE. Significant potential risk factors for HTE may be genetic predisposition, a family history of dementia, career duration and number of races, the age and cognitive redundancy of Athlete [3].

Pathophysiology

The neuropathological damage subject to boxers involves abnormalities of the cerebellum, cerebral atrophy and scarring, degeneration of specific groups (i.e., the melena substance, the Hypoblack area, and the basal nucleus of Meynert), neurovirals Complexes and destruction of the cavity of the transparent diaphragm [6].

Damage to the HTE may be due to mechanical shear forces in the large axon/dendrites. The loss of dendrides due to the shear may explain the reduction of the involvement of nerve cells in the plaques. Such a phenomenon could become even more intense with age since neurons, deprived of dendritic, would be impossible to present the dendritiki vegetation that occurs with age, something that is found in normal brains. Alternatively, the likelihood of damage to the cerebral blood vessels, with the leakage of serum constituents in the brain, should be considered [7].

Clinic presentation

The symptoms of the HTE are gradual and usually developed after the end of the career or even years after the retirement of a boxer from the ring. Noteworthy is the fact that in several cases chronic traumatic encephalopathy can mimic Alzheimer's disease both clinically and pathologically. Indeed in a study by Renata., et al. [8] the differential diagnosis from Alzheimer's disease proved to be very difficult, especially when based solely on clinical level, despite systemic neurological and neuropsychological assessments. However, the Neuropathologic findings were typical of the dementia of the boxers [1].

In the HTE, three stages of clinical deterioration are recognizable. Although the progression of symptoms is an unavoidable fact as we speak of degenerative disease type, the syndrome can remain clinically stable at any stage. They usually mediate several years between stages. The first (early) stage consists of emotional disturbances, psychotic symptoms, and a slight discount of cognitive functions that are often not perceived. During the second (late) stage an intensification of social instability and psychiatric symptoms occurs along with memory loss (early dementia) and parkinsonism signs/symptoms. The final stage consists of a significant further reduction in general cognitive functions that develops into full-scale dementia, along with simultaneous deterioration of motor problems, which gradually make the patient clinically [7].

Diagnosis of the HTE

Any boxer who suspects that he or she has experienced or developed the HTE should undergo a complete neurological examination including imaging of the brain, with appropriate laboratory tests if necessary, as well as microbiological examination Spinal fluid (CSF) and blood biomarkers [9].

Illustration

The imaging of the brain can be done using a CT scan or an MRI scan. The MRI is preferable between the two Tests. It can show cortical atrophy, hydrocephalus or both. Sometimes it shows a lesion that requires surgical treatment, such as subdural hematoma or hydrocephalus. Occasionally, more sophisticated imaging media, such as a simple photon-emission CT scan and positron tomography, can be useful [10].

EMS and Blood bioindicators

There are currently no biomarkers available to diagnose the HTE. The ENY is a logical source for bioindicators in neurodegenerative diseases, because it directly encompasses the brain and its biochemical composition, it can therefore reflect the pathologies of the brain. Because the bioindicators of the ENY have been successfully developed and used regularly in the clinical diagnosis of Alzheimer's disease, they also serve as a model for the hte. Studies have been conducted on concussions, but there are no published studies For HTE. The glial fibrous acid protein, a special ENY protein, was found to be strongly increased in plasma in patients with concussion showing abnormal imaging findings, suggesting that their injuries may, in Reality, they were more serious [11,12].

Prevention

The increased popularity of contact sports around the world exposes a large number of participants in both acute and chronic traumatic brain damage (HTE). Since its treatment options are relatively limited, its prevention is of paramount importance. Prevention will need to be specific to the sport, and undoubtedly to limit the exposure of athletes to high-risk injuries becomes necessary the neurological evaluation of the athlete, the imposition of strict adherence to the guiding Lines of "Return to play", training and supervision of athletes, increased medical supervision and the use of protective equipment [10].

Neurological assessment

It is important to note that, at present, there is no generally accepted definition or classification for the diagnosis of a concussion. A system often used for the classification of sports related to concussions is one proposed by Cantu (1998, 2001) and shown in table 1. This system identifies loss of consciousness, confusion and post-traumatic amnesia as important criteria for determining the presence and severity of a concussion. The purpose of these gradients is to standardize the classification system of the severity of a concussion, with the aim of influencing the clinical management (e.g. return to races or sparring) or to develop interventions that are specific to various Severity of an injury [13].

Evaluation next to the ring

The Doctor next to the ring is usually the first practitioner to evaluate the boxer after a KO or a technical KO, and performs an unspecified neurological examination, and possibly a cursory examination of the mental condition. These tests are important for detecting conditions potentially life threatening or even serious disability (e.g. cerebral hemorrhage or edema) but are not sensitive enough to detect and quantify the emerging of cognitive deficits that may develop after race [6].

The Abacus 1: Concussion scale with "return to sport" criteria (Return-to-play) (Cantu 1998).

Grade I - No loss of consciousness or post-traumatic amnesia for less than 30 minutes

- First concussion: Return to play if the patient is asymptomatic for 1 week
- Second concussion: Return to play if the patient is asymptomatic for 2 weeks
- Third concussion: Ending season

Grade II -Loss of consciousness for less than 5 minutes, or post-traumatic amnesia for 30 minutes to 24 hours

- First concussion: Return to play if the patient is asymptomatic for 1 week
- Second concussion: Back to play if, after at least 1 month, the patient is asymptomatic for 1 week
- Third concussion: Ending season

Grade III -Loss of consciousness for more than 5 minutes, or post-traumatic amnesia for more than 24 hours

- First concussion: Back to play if, after at least 1 month, the patient is asymptomatic for 1 week
- Second concussion: Ending season

Table 1

Back to sport

In conclusion, safe return to sport may require 4 to 6 weeks to facilitate full recovery and protect against a new injury, which is usually a second concussion that appears much more Often in the period immediately after the first. It is recommended that before returning to the ring, the boxer 1) must be free from all symptoms of post-concussion syndrome at rest, 2) must have a normal neurological examination, 3) must have results From neuropsychological tests, which are comparable to the initial scores (if initial scores had arisen) and 4) should not have neuroimaging findings showing structural damage [6].

Retirement

According to Echemendia and Cantu in 2003, there are two clinical variants that should raise concerns about when an athlete with multiple concussions should stop participating in contact sports, such as boxing. The first concerns the change in the duration of the post-concussion symptoms. For some athletes with a history of multiple concussions, their duration lasts longer than normal. The second variation of concern is the nature of the force needed to produce a concussion and prolonged symptoms. When the blows

to the body produce neurological dysfunction, it is strongly recommended that the boxer should retire [14].

Conventional approach

In the existing bibliography there is no proposed treatment plan so far. The HTE is a disease that recently began to study more extensively and still there are many gaps in knowledge on the subject, which need to be supplemented.

Taking into account the clinical presentation of the disease and the progression of symptoms, information was collected concerning both the treatment of parkinsonism and dementia, and were used to design a proposed plan Evaluation and treatment. It is worth noting that this is the first time such an attempt has been made.

Physical therapy evaluation

The evaluation is a continuous process, which is contained throughout the duration of the physiotherapy intervention and forms an essential part of the design of therapeutic targets in patients with HTE. The evaluation should not only be found in the patient's deficits. It should detect each patient's ability to produce posture and motion. The therapist will not use the deficits, but the possibilities available to the patient in order to increase them.

The symptoms of HTE are a component of both parkinsonism and dementia. As a result, the physico-therapeutic evaluation proposed by this review would involve two stages. One stage will relate to the evaluation of dementia and behavioural disorders and the other stage will concern the evaluation of motor disorders, namely Parkinsonic syndrome.

Evaluation of dementia

The evaluation identifies the subjective and objective data of the patient. It may prove to be the most important part of the evaluation. The subjective information includes the orientation of the patient, the mental level, the possibilities of communication and the relations with the family environment. On the first visit, the physiotherapist attempts to gather subjective information from comments, observations and information from his narrow environment. At the same time, the therapist must decide how much time he should allocate for the evaluation according to the patient's abilities.

After the collection of subjective data begins the process of collection of objective data based on the mental and physical condition of the patient. The evaluation of the objective data includes the assessment of the nervous, musculoskeletal, cardiovascular and respiratory system. Two tools proposed for this purpose are the MMSE (Mini Mental State Examination) and CDR (Clinical Dementia Rating).

Parkinsonism assessment

The evaluation of the Parkinsonic syndrome includes the evaluation of all the motor problems that the patient may be able to display. There are several evaluation tools, both questionnaires and tests, that help to collect information on both the subjective and the objective findings. Some of them, which may seem useful in clinical practice are: Parkinson's disease Questionnaire (PDQ-39) -Contains 39 questions, covering 8 aspects of quality of life [15], Parkinson's Activity Scale(PAS)-Assessment of problems related to functional mobility.Lasts about 10 to 15 minutes [16], Six-minute walk test(6mwt)-Locating and assessing physical capacity [17,18]; Ten-Meter Walk Test(10MWT)-Determination of the comfortable gait rate. Measuring stride length [19], Timed Up and Go test (TUG)-Gait and balance test [20].

Physical therapy

In order to determine a plan for conservative treatment for patients with HTE, clinical separation of the progression of the disease in three stages is necessary. These stages are: premature, late and final. The treatment proposed at each stage takes into account both mental and motor disorders of each stage.

Early stage

The early stage consists of emotional disorders, psychotic symptoms, and a slight discount of cognitive functions that are often not perceived. At this stage physiotherapy can not benefit the patient. The only therapeutic intervention that can be performed is the referral to a psychologist/psychiatrist, if this is deemed necessary.

Late stage

At the late stage an intensification of social instability and psychiatric symptoms occurs along with memory loss (early dementia) and parkinsonism signs/symptoms. The proposed physiotherapy treatment has the primary objective of dealing with motor problems, namely the treatment of parkinsonism symptoms. However, the cognitive dysfunctions that coexist in the HTE (memory loss, difficulty concentrating) can gradually force the therapist to adjust his therapeutic plan to the point where the treatment will be exclusively for the part of Exercise to maintain a general well-being and as much functional muscle and respiratory system as possible.

Treatment of Parkinson's symptoms

For patients with HTE at this stage, as far as Parkinsonic syndrome is concerned, the aim of the physiotherapist is to improve the quality of life by maintaining or increasing the patient's independence, safety and well-being. After receiving the medical history and the execution of the physical therapy evaluation, the physio-

therapist formulates, in consultation with the patient, a treatment plan. The treatment plan includes the goals of treatment and gives them priority.

The treatment targets in patients at this stage are:

- Improving transport performance: using slogans in conjunction with the implementation of cognitive movement strategies [21].
- Normalizing Posture [17]
- Stimulation of balance: walking out 3 times a day, Tai Chi, modifying any risk there is at home [22].
- Improving gait [23]
- Prevention of inactivity and maintenance or improvement of physical fitness: exercises with exercise equipment, treadmill
- Stimulation of approach and conception of objects: strategies of slogans and movement and avoidance of dual tasking [24]
- Prevention of falls: courses aimed at improving endurance, balance and coordination [25]
- The location, the time and the duration of the treatment, the proper participation of the caregiver and the rhythm of the exercises play an important role in the rehabilitation program [26-29].

Treatment of dementia

During progression of the disease occurs progressively worsening dementia. Due to the difficulty of concentration and the loss of memory it entails, the patient is struggling and tired increasingly to understand complex exercises. That is why we are gradually trying to integrate it into a programme of activities that is easier and less tiring for him. The activities and treatment program should be designed to maintain the independence of the patient, as well as improve the quality of life, if the course of the disease can not be traced.

The treatment plan at this stage includes physical activities such as castings, kicks, blows and dancing, verbal activities such as singing, touch activities such as scrubbing and brushing of various surfaces, as well as Housework.

Final stage

The final stage consists of a significant further reduction in general cognitive functions that develops into full-scale dementia, along with simultaneous deterioration of motor problems, which gradually make the patient bedridden. The condition of the patient is very difficult, the quality, the course as well as the rest of the time of his life is now dependent on the caregivers.

The aim is to avoid pressure sores due to immobility and the high degree of weakening of the respiratory system of the patient, reasons that will lead him to death. The treatment program will contain passive kinesitherapy of limbs, kneading for transient improvement of blood circulation, change of positions to avoid pressure sores and respiratory exercises.

Conclusions and Suggestions

Professional boxing is associated with many dangers since it is the only sport in which there is a subjective intent to cause physical damage to the opponent.

The Clausen., et al. In a review, shown in table 2, analyzed the data from published studies involving active boxers in Great Britain and Australia found that from 1930 to today. They noticed that there was a significant decrease in the average career duration and the number of matches of the professional boxer. So we conclude that the incidence of boxing-related HTE will be reduced to the current era of professional boxers despite the fact that participation in boxing in the population remains constant [30].

Boxing season	Sample number	MO career Duration (years)	Career length range (years)	MO Races	Number of races
1900 – 1955	254	19	3 – 22 of	336.5	< 50 – 700 +
1950 – 1995	77	9.6	1 – 22 of	39.6	0 – 122
2000-Today	295	5	1 – 32 in	13.3	1 – 138 in

Table 2: Summary of changes in career and periods in all boxing seasons

Although knowledge on the HTE has evolved quite a bit in recent years, it should not be surprising that there are still many crucial questions about this disease. That's where the investigation should turn. The questions include the following:

- What is the exact mechanism and what are the risk factors for the disease?
- What is the exact population of the HTE? What's the exact frequency of the HTE?
- How can the HTE be detected and diagnosed accurately during life?
- How can the HTE be prevented further? What interventions may lead to a successful modification of the disease?

Summary

This review presents a summary of modern scientific data and perceptions of chronic traumatic encephalopathy (HTE). The HTE is a progressive neurodegenerative disease caused by recurrent closed-ended head injuries, including concussivity and hypostatiki injuries. It was first described in 1928 by Martland as the "drunken Fist syndrome" (Punch drunk syndrome). The HTE is closely related to athletes participating in contact sports, such as boxing. At present, prevention and education are the only way to combat the HTE and should be emphasised to them by both doctors and athletes. However, in the existing scientific literature, there is no proposed treatment plan so far. In the context of this review, taking into account the clinical presentation of the disease and the progression of its symptoms, information was collected and used to design a proposed physical evaluation plan and Troubleshooting. However, further study is needed to help create clinical diagnostic criteria, to find objective biomarkers for disease, to define therapies and to support individuals and their families living with the Cte.

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