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MEDICAL-LEGAL FRAMING OF POST-CONCUSSIONAL SYNDROME (PCS) IN THE ITALIAN PENAL LAW AND CIVIL LIABILITY, SOCIAL AND PRIVATE INSURANCE FIELDS

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Abstract: The authors, both medical specialists in legal medicine, discuss about the medical framing of post-concussional traumatic head syndrome (PCS) in forensic medical evaluations, from both penal and civil liabilities as well as in Italian social insurance and private insurance. The authors describe a case that has come to their attention and discuss the difficulties of both clinical and medical-legal classification of PCS. They analyse possible diagnostic methods and instruments which can support the diagnosis and take into account possible differential diagnoses or clinical simulations. Finally, they evaluate the medical-legal framework of PCS in the fields of Italian civil liability and penal law as well as Italian social and private insurance.

Keywords: Post-concussional head syndrome, PCS, Italian legislation, social security, civil incapacity benefits, private insurance.

INTRODUCTION

Head injuries, even if minor, can produce a clinical syndrome of particular severity often characterised by a continuous headache and state-kinetic disorders [1]. Sometimes these symptoms become permanent and even reach a state which is considered stabilised, both neurologically and psychiatrically. It is thus necessary, for various medical-legal and jurisprudential purposes, to establish the severity of these symptoms and how the symptomatologic framework directly correlates to the cranial trauma which occurred.

The clinical symptoms of mild traumatic brain injuries (mTBI) is heterogeneous and, in many cases, can be difficult to ascertain according to the force of gravity causing the injury. From least force to greatest: concussion, with mild traumatic brain injury usually resulting from a blow to the head and clinically characterised only by a temporary loss of consciousness and amnesia [2, 3]. This set of neurological symptoms results from a traumatic kinetic incident affecting the encephalic areas and the reticular substance, thereby impairing the mental alertness of the subject. This is confirmed by anterograde and retrograde amnesia, typical in concussions. The anatomical nature of the concussion can often be attributed to bulb-pontine and cerebral micro-haemorrhages and neuronal alterations of vestibular nuclei. The mechanisms of secondary histopathological injuries relate to excitotoxicity, cytoskeletal proteolysis, calcium dysregulation, and mitochondrial dysfunction [4].

The level of severity higher than concussions is the post-concussion syndrome (PCS), also known as subjective cranial trauma syndrome or post-traumatic subjective syndrome, the most frequent complication of mTBI. This syndrome appears most frequently in the course of minor head trauma, occurring in 60 to 70 percent of cases, as proven by the Glasgow Coma Scale (GCS) with a score of 12 or lower [5, 6]. In these cases, the clinical picture is characterised by physical symptoms such as headache, dizziness, photophobia, difficulty concentrating, asthenia, reduced tolerance to noise and heat, with psychological and behavioural symptoms including a tendency to depression, memory loss, anxiety, insomnia, and irritability. This set of symptoms may continue for months or years after the brain trauma. Headache is the most common symptom: 30–90% of people treated for PCS report more frequent headaches, and 8–32% report them a year after the trauma [7, 8].

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History of the concept of post-concussion syndrome

From an historical point of view, it should be reported that the first description of cranial trauma syndrome dates back to 1866, when the Danish surgeon John Erik Erichsen (1818–1896) described the onset of subjective severity disorders which were not directly proportional to the actual extent of the head trauma resulting from a train accident [9, 10]. By 1879, however, the Prussian physician Johannes Rigler noticed an increase in the incidence of post-traumatic neurosis following the Prussian government's establishment in 1871 of mechanisms of economic compensation following rail accidents [11, 12]. He used the German term Renten-Neurose (Pension neurosis), translated in English as Compensation Neurosis [13, 14].

In 1899, Hermann Oppenheim (1858–1919), one of the leading neurologists in Germany, introduced the term traumatic neurosis, understood as "physical and psychic shaking that acts on the brain causing molecular alterations in those areas where psychic functions are carried out and where sensory centers are located" [15]. Oppenheim's theory was based on the fact that "in the genesis of this illness, physical trauma is only partially responsible (…) the mayor role is played by the psyche: terror, emotional shock" [16]. From then on, there was a blossoming of terms to identify this syndrome: De Morsier's traumatic encephalopathy [17], Friedman's concussion, Friedman traumatic encephalosis [18], Bini's physiological condition of neurasthenia, psychoreactive manifestations. In fact, Lucio Bini (1908–1964), an Italian psychiatrist and professor at the Sapienza University of Rome, categorised traumatic syndromes into three fundamental subgroups, depending on whether organic functional symptoms were involved: 1. post-traumatic neurastheniform physiogenal syndrome, now called post-traumatic subjective syndrome (PTSS), 2. post-traumatic neurasthenic psychogenic syndrome, and 3. mediated post-traumatic psychogenic neurasthenic syndrome or psycho neurosis from economic compensation.

The tenth revision of the International Classification of Diseases (ICD-10), approved in 1990 during the 43rd World Health Assembly and used since 1994, first set out neuro-psychiatric criteria for PCS; the Diagnostic and Statistical Manual of Mental Disorders, 4th revision (DSM-IV), published in 1994, listed similar clinical criteria for the diagnosis of postconcussional disorder (PCD) in people who had suffered head trauma with post-traumatic amnesia and loss of consciousness [19, 20]. The DSM-IV-TR criteria for PCD requires, first, a history of head trauma; second, evidence from neuropsychological testing or a quantified cognitive assessment of difficulty in attention or memory; and finally, three (or more) of the following occurring shortly after the trauma and lasting at least three months: easily becoming fatigued, headache, disordered sleep, dizziness, anxiety, irritability, depression, changes in personality, apathy, and lack of spontaneity [21].

Pathological bases of post-concussion syndrome

Head trauma can occur through two mechanisms: 1. the head is hit by a mass in motion, or 2. a moving head is bumped against a motionless and resistant structure, as is the case in the vast majority of road accidents. The pathogenic mechanism behind PCS is, undoubtedly, related to a sudden wave of acceleration and deceleration which affects the entire brain mass and the underlying nerve structures of the trunk, causing the nerve mass to stretch and subsequently bump against the bone structures of the skull. An alteration of the hydrodynamics of the cerebrospinal fluid could also harm the central nervous system (CNS).

In particular, the traumatic dynamics could generate lines of force which converge from the impact zone at the level of the brainstem and, more precisely, at the meso-diencephalic formations, also called the zone of vulnerability by De Morsier [22]. Circulatory reactions occur here, both vasospastic and vasoparalytic, which can prolong the phase of unconsciousness, initially due to the action of the trauma on mesodiencephalic structures. Sometimes, from a histopathological point of view, lesions are visible as small haemorrhagic outbreaks, spreading in the corpus callosum and the more rostral portion of the brain stem, which evolve into darker scars. This produces the typical pathological picture of axonal degeneration, with the extrusion of the axoplasm through torn neuritic structures. In the area surrounding the damaged fibres, the microglial reaction and degeneration of the distal stump of the fibres can be observed. The axonal degeneration at the brainstem and at the ascending reticular pathways are the histopathological basis of the cognitive deficits which characterise PCS.

It should be added, however, that these organic CNS signs are summed up, from time to time, to elements of psychological nature, able to greatly aggravate the symptoms' score, overlapping and adding to the damage of a histopathological nature produced directly by trauma.

Clinical symptoms set related to the PCS and its instrumental evaluation

Headache is, as we said, the most common symptom of PCS, which can occur in 30–90% of cases. It is more frequently atypical, irregular in topography, and almost always uncontrollable with the use of analgesics, anti-inflammatory drugs, or triptans. Dizziness, often triggered by abrupt head movements, rarely has a rotational character and often manifests itself with instability and balance disturbances. Memory impairments are extremely frequent, often accompanied by deficits of fixation. Finally, sleep disorders are largely conditioned by psychic reactions and conflicting situations.

PCS, due to the statistical frequency and the clinical importance it shows, was prematurely framed among the specific non-psychotic mental disorders (ICD-9 in 1978), since it can persist for several months, although with wide fluctuations and temporarily preventing the individual from working. The diagnostic approach to the syndrome can involve the use of numerous instrumental methods, such as a standard X-ray of the skull (usually low in information), a CT scan (which can visualise the most consistent brain lesions), an MRI (which shows a higher level of definition for brain tissue), and an EEG (an extremely important method for medical-legal purposes although often unable to provide valid judgment for the purpose of quantifying a possible inability to work or the severity of PCS). Sometimes, in order to delineate the psychic profile of the traumatised person, it can be useful to resort to psychodiagnostic tests of proven efficiency, including the Wechsler-Bellevue test, the Bentos test, the Minnesota Multiphasic Personality Inventory, and the Rorschach test, among others.

From the 1980s, in order to document in a scientific and unequivocal way, the subclinical alterations of PCS, brainstem auditory evoked potentials are used. Also called brainstem auditory evoked responses, these are small auditory evoked potentials in response to an auditory stimulus, which are recorded by electrodes placed on the scalp. They reflect neuronal activity in the auditory nerve, cochlear nucleus, and other parts of the brainstem. The subsequent use of ophthalmological explorations has made it possible to assess a narrowing of the field of vision, which is characteristic of many people suffering from traumatic head PCS. The introduction of neurotological evaluation techniques has recently been enriched by numerous detection methods aimed at objectivising and quantising, as far as possible, the symptoms in order to assess their true impact for clinical and forensic medicinal purposes.

The clinical symptom set of PCS should be evaluated at a distance from the accident event; this is of fundamental importance from a medical-legal point of view. Generally, the neuro-psychiatric examination should occur 17 days to 24 months from the traumatic event, although waiting 6 months is often a good prerequisite for a correct characterisation of the psychiatric symptoms. In many of the studies conducted, almost all subjects had symptoms of a state-kinetic nature at the time of the examination, while much rarer were the cephalalgic descriptions. The definition of psychiatric symptoms often requires longer time arcs in order to be appreciated.

The differential diagnosis of PCS from other post-traumatic stress disorders is often difficult, as it is hard to distinguish the psychogenic quantum from the anatomopathological one [23]. It is extremely easy to falsify headaches or excess dizziness or to simulate psychiatric symptoms. The element of medical-legal evaluation must, therefore, be based on the actual existence of the declared symptoms and on the appropriate and sufficient consequentiality between the onset of the neuro-psychiatric symptoms, the developed injury, and the declared trauma.

Recently, the neuro-psychiatric symptoms of the traumatised person have been objectivised through the use of electro-oculography: these techniques propose that the correct standardisation of post-concussion symptoms include the determination of spontaneous nystagmus (analysed in lightness and darkness), the positional nystagmus, the cervical nystagmus, the nystagmus of bounce, slow eye pursuit, rapid eye pursuit, caloric test, vision suppression test, and ocular motor testing based on the velocity step of the ocular muscles.

An emblematic case

A 52-year-old woman, carried on the right front seat of a car, was involved in a road accident: the driver of the vehicle, because of irregular asphalt, lost control of the car. The passenger was transported to the nearest emergency room. Here, she underwent X-rays of the skull and cervical column, both negative for bone lesions, and a CT examination of the skull which did not show parenchymal focal lesions. She was discharged with a diagnosis of "left temporo-parietal contusion with small excoriation and small excoriation to the right forearm." Because of the persistence of painful symptoms in the left temporo-parietal region, associated with the oedema of the homolateral ear lobe

as well as headache, difficulty in concentration, and episodes of amnesia, the subject repeatedly visited her family doctor, who prescribed additional rest periods and home care. The aforementioned symptoms did not withdraw and dizziness and hypoesthesia of the right side of the body appeared; she went to a specialist in neurology, who prescribed drug therapy. Three months after the accident, she underwent an MRI of the skull, which showed the presence of point areas of the white matter of the left temporal lobe from ischemic outcomes on a microvascular basis.

Between 10 and 15 months after the car accident, in fact, the women showed asthenia mainly on awakening, serotine severe headache, parenthesis of the left temporo-parietal region, attentive deficits, and difficulty in falling asleep – all elements which can be traced back to the PCS diagnosis according to ICD-10 and DSM-IV criteria.

Different medical legal framings of the PCS in Italian penal law and civil liability, social and private insurance

The possibility of a clinical and functional objectivisation of the framing of PCS through the use of electro-oculography acquires importance for forensic purposes. The use of these tests, associated with instrumental examinations of the skull (CT, MRI) and in-depth neurological and psychiatric examinations, are considered the gold standard for the diagnosis of PCS.

It is fundamental, from the Italian criminal law point of view, to recognise the specific causal linkage between the traumatic event, from the kinetic action with traumatic power to the anatomopathological CNS damage and the subsequent development of PCS. In particular, electro-oculographic determination can be useful precisely as a result of the slow temporal evolution of clinical symptoms, often defined only 18–24 months after the traumatic event. The duration of illness (more than 40 days – almost a constant proper to PCS) can, from a criminal point of view , frame the injury in the context of serious personal injury to a person (lesione personale grave in Italian), configured in the medical-legal permanent weakening of a body organ or apparatus. Much rarer are cases whereby the traumatic injury constitutes a permanent loss of higher neurological functions, outlining the case of the disease with certainty and likely indicating insanity; it must be included, from the Italian Penal Law, in the context of very serious malicious personal injuries (lesione personale gravissima) [24].

In the Italian civil liability field, PCS is recognised as a permanent damage only in those cases where the symptoms last so long that they do not foresee a cure (the medical-legal concept of stabilisation and permanence of damnum injuria datum). As Clemente Puccini, an Italian forensic medical doctor, stated [25], in this case, the damage will have to be economically compensated for its full amount since it can be a cause of injury aggravating the consequences of the damage itself. This is the concept of biological damage, that is, the "reduction of the physical and mental validity of the subject". From a medical-legal point of view, the first problem is that PCS is not framed in the main scales (barèmes in French language) of damage, the medical legal guidelines for evaluating the biological damage according to civil liability. Thus, the medical-legal percentage values of disability which could be attributed to PCS ranged from 3% to 5%, established using Luvoni–Bernardi guidelines [26], or 10% using guidelines from Bargagna-Canale [27]. In civil liability law, there is no problem with the demonstration of whether the obligation exists (an debeatur in Latin) but what this obligation amounts to (quantum debeatur): in response to this question, the Italian Supreme Court suggested the adoption of a fair criterion of damages compensation, as stipulated in article nos. 2056 and 1226 of the Italian Civil Liability [28]. Luvoni and Bernardi have argued that, in the civil liability field, PCS can rise from the context of the micropermanenti (the Italian term describing lesions which reduce the physical and mental validity of a person by 1–9 percentage points) to reach the macropermanenti area (lesions reducing a person's abilities by more than 10 percentage points). This increase in the medical legal evaluation, up to 15–20% of the disability rate, could happen when instrumental documentation (CT or MRI) can prove the existence of objective physical harm at the level of the CNS: contusive areas of the brain, either focal or widespread areas of ischemic suffering or reactive gliosis.

From a social insurance perspective, in Italy, there is a dual insurance protection system which distinguishes between the diseases caused by working activities or happening during work and common diseases not attributable to work, war, or service. The first group is protected by the National Institute for Insurance against Accidents at Work (INAIL), a public non-profit entity safeguarding workers against physical injuries and occupational diseases. Common diseases, instead, are under the protection of the Italian Institute of Social Security (INPS). When PCS develops during

work or is caused by working activities (for example, a head injury occurring in a worker), INAIL protection is activated: for table reference of INAIL pathologies according to Ministerial Decree July 12, 2000 [29], item 182 (Subjective syndrome of head traumatized) recognises up to 4% of biological damage.

In the common diseases' social protection, instead managed by INPS, there are no percentage reference table assessments; the status of INPS invalid is recognised when an insured person has his/her work capacity permanently reduced due to infirmity or physical or mental defect to less than a third in suitable activities [30-32]. In exceptional cases, PCS can cause a two-thirds reduction of working capacity in suitable activities.

In private car accident insurance, the right to compensation for the accident is configurable only in the case of objectively detectable injuries and, in these cases, the use of CT and MRI is of paramount importance: these methods demonstrate the real existence of damage and prove the quantum debeatur.

Finally, for protection with regard to illness or disability in persons who are not assisted by Italian social insurance (minors who do not work, indigents, persons who are inactive or who lack the administrative requirements for pensions), civil incapacity benefits have been established under Italian law no. 118 of March 1971 [33, 34]. In this field, there are still no references in which PCS can be framed: the item mild neurosis of an obsessive phobic nature (code 1202 of the percentage tables linked to the Ministerial Decree February 5, 1992 [35], used by law in the determination of impairments in civil incapacity benefits) establishes the loss of generic working capacity around 15%. It is below the minimum threshold of 34% for the recognition of Italian civil incapacity status.

In conclusion, the case examined by the authors is one of many various neuropsychiatric frameworks which can be enclosed in the vast and heterogeneous scope of PCS. In contrast to what is normally found in the aforementioned nosographic category, which by definition is based on the unique subjectivity of the picture, in this case, there is, instead, a rather suggestive instrumental importance of an organic interest of the CNS parenchyma. In fact, the MRI examination shows outcomes due to point ischemic suffering (previously called axonal degeneration) with reactive gliosis located in the left hemisphere, the site of the trauma. The existence of instrumental findings thus facilitates the demonstration of the causal link between the traumatic event and the neuro-psychiatric symptoms later produced. Moreover, the time taken by the event, more than six months, may be considered sufficient to stabilise the neuro-psychiatric symptoms set, the latter a prerequisite for the recognition of this nosographic entity for medical-legal evaluation purposes.

On the other hand, it is much more difficult to recognise and quantify the aftershocks resulting from those conditions of suspected PCS in which they do not concoct anatomical alterations which can be highlighted by both neuroimaging (CT and MRI) and functional methods (EEG, brainstem auditory evoked potentials, electro-oculo-graphics techniques). Thus, the forensic medical doctor becomes of paramount importance, to which the arduous task falls of bringing out that darker and perhaps evanescent aspect of biological damage – that is, the reduction of the only psychic and non-somatic validity.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. Fortuni G, Milazzo V. Contributo alla conoscenza della sindrome soggettiva nei traumatizzati cranici: aspetti clinici e medico-legali. Jura Medica. 1992.
2. Herbert DL. Medical-legal issues of mild traumatic brain injury. Curr Sports Med Rep. 2007; 6(1):20–24.
3. Zasler ND, Bigler E. Medicolegal issues in traumatic brain injury. Phys Med Rehabil Clin N Am. 2017; 28(2):379–391.
4. Winn HR. Youman's neurological surgery. In: Zacko JC, Hawryluck GWJ, Bullock R, editors. Neurochemical pathomechanisms in traumatic brain injuries. 6th ed, Philadelphia: Elsevier; 2011.
5. Iverson GL, Lange RT. Examination of "postconcussion-like" symptoms in a healthy sample. Applied Neuropsychology. 2003; 10(3):137–144.
6. Di Luca NM, Elia M, Larocca D. La sindrome soggettiva post-traumatica. Revisione dell'inquadramento clinico e dei profili medico-legali. Zacchia. 1990; 63:161–188.
7. Hall RC, Chapman MJ. Definition, diagnosis, and forensic implications of postconcussional syndrome. Psychosomatics. 2005; 46(3):195–202.
8. Seifert TD, Evans RW. Posttraumatic headache: a review. Curr Pain Headache Rep. 2010; 14:292–298.
9. Erichsen JE. On railway and other injuries of the nervous system. Philadelphia: Henry C Lea, printed in 1867.
10. Della Beffa A. I traumi cranici. Esiti ed aspetti medico-legali. Milano: Vallardi; 1963.
11. Randolph EW. Secondary headache – an issue of neurologic clinics. Philadelphia: Ed Elsevier; 2014.
12. Valent P. From Survival to Fulfilment. A framework for the life-trauma dialectic. Brunner – Mazel Ed, Ann Arbor, MI; 1998.
13. Rigler J. Über die Folgen del Verletzungen auf Eisenbahn, insbesondere der Verletzungen des Ruckenmarks [On the impact of injuries on the railway, particularly spinal cord injuries], G Reimer Ed, Berlin; 1879.

14. Slyomovics S. How to accept German reparations. Philadelphia, Pennsylvania Press; 2014.
15. Oppenheim H. Die traumatischen Neurosen [The traumatic neurosis]. Springer Verlag Berlin, Heidelberg GmbH; 1915 http://libarch.nmu.org.ua/bitstream/handle/GenofondUA/11449/68620c00415b643ba5c2800e8977f10a.pdf?sequence=1 (last checked May 20, 2020).
16. Oppenheim H. Lehrbuch der Nervenkrankheiten für Ärzte und Studierende, Berlin Karger; 1894.
17. De Morsier G. Disorders of the autonomic nervous system in traumatic encephalopathy; adynamia, bulimia, sialorrhea. Acta Neuroveg (Wien). 1950; 1(1–2):114–122.
18. Friedman AP, Brenner C. Psychological mechanisms in chronic headache. Ass Res Nerv Dis Proc. 1950; 29:605–609.
19. McCauley SR, Boake C, Pedroza C, Brown SA, Levin SH, Goodman SH, Merritt SG. Postconcussional disorder: are the DSM-IV criteria an improvement over the ICD-10? J Nerv Ment Dis. 2005; 193(8):540–550.
20. Yeates KO, Taylor HG. Neurobehavioral outcomes of mild head injury in children and adolescents. Pediatric Rehabilitation. 2005; 8(1):5–16.
21. Friedland D. Postconcussion syndrome/disorder or mild traumatic brain injury: diagnostic issues and treatment. Available from: https://www.acnr.co.uk/2015/04/postconcussion-syndromedisorder-or-mild-traumatic-brain-injury-diagnostic-issues-and-treatment/ (last checked May 20, 2020).
22. Schweizerische Gesellschaft für Psychiatrie. Zeitschrift für Kinderpsychiatrie: Revue de psychiatrie infantile. Acta paedopsychiatrica, Schwabe; 1937.
23. Fornari U. Psychopathology and Forensic Psychiatry. Turin, Ed UTET; 1989.
24. Art. 583 and 590 Italian Penal Code. Available from: https://www.brocardi.it/codice-penale/libro-secondo/titolo-xii/capo-i/art590.html (last checked May 15, 2020).
25. Puccini C. Institutions of Legal Medicine. 6th ed, Milan: Ambrosiana Ed; 2005.
26. Luvoni R, Mangili F, Bernardi L. Guide to medical-legal assessment of biological damage and permanent disability. 5th ed, Milan: Giuffrè Editore; 2002.
27. Bargagna M, Canale M, Consigliere F, Palmieri L, Umani Ronchi G. Guide for the evaluation of biological damage. 3rd ed, Milan: Giuffrè Editore; 2001
28. Italian Tort Law. http://italiantortlaw.altervista.org/civilcode.html (last checked 15 May, 2020).
29. INAIL. Tables of impairments resulting from injuries of psycho-physical integrity, including dynamic-relational aspects, March 2000 In: Macchiarelli L, Arbarello A, Di Luca NM, Feola T. Legal Medicine, 2nd Ed, Turin, Minerva Medica Editions. 2005: 858-879.
30. Italian Law no. 222 of 1984 June 12 ("Review of the discipline of pension disability"), published in the Official Gazette of the Italian Republic; no. 165: 1984 June 16. Available from: http://www.handylex.org/stato/l120684.shtml (last checked May 2, 2020).
31. Sammicheli M, Scaglione M. Employment suitable for capabilities: the medical-legal principle in Italian invalidity social security benefits in the current historical work context in Europe. European Journal of Social Security. 2018; 20(3):242–252.
32. Catalan C, De Luca F, Vitiello G. Elements of Social Legal Medicine. Milan: Giuffrè Ed.; 2006.
33. Sammicheli M, Scaglione M. Information technology as a tool for the Italian Institute of Social Security (INPS) in the management of social security and civil disability: pro and cons. Technology and Health Care (Journal of the European Society for Engineering and Medicine). 2018; 26(2):343–347.
34. Italian Law no. 118 of 1971 March 30 ("Conversion into law of legislative decree January 30, 1971, n. 5 and new rules in favour of the civil disable persons"). Available from: http://www.handylex.org/stato/l300371.shtml (last checked April 6, 2020).
35. Ministerial Decree, Italian Ministry of Health, 1992 February 5 ("Approval of the new indicative table of invalidity percentages for disabled people and invalidating diseases"). Available from: http://www.handylex.org/stato/d050292.shtml (last checked May 8, 2020).