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AFTER A MILD TRAUMATIC BRAIN INJURY, CAN A PERSISTENT POST-CONCUSSION SYNDROME BE PREDICTED? A PROSPECTIVE CLINICAL STUDY ON 55 CASES

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SUMMARY

Background Every year, in Europe, 1 million people suffer a traumatic brain injury (TBI). 80% of these are mild, but 10 to 15% are left, 3 months after the accident, with somatic, cognitive and behavioral disorders, often thought of as psychogenic, and therefore disregarded. Our study was aimed at early prediction of a negative course after MTBI.

Material/ Methods: 55 MTBI patients were recruited in the emergency units of 4 French university hospitals. The patients were evaluated between 8 and 21 days (T1), then between 3 and 4 months (T2) after the accident. The evaluations comprised a complaints questionnaire, simple neuropsychological tests, the Hospital Anxiety and Depression scale, and visual analogue scales of pain and quality of life.

Results: At T2, 29 MTBI did not report, but 26 MTBI were reevaluated: 15 had a persistent post-concussional syndrome (PPCS) and 11 had a positive course (non-PPCS). Neuropsychological scores and general complaints – especially pain – as well as quality of life were significantly lower, with a higher anxiety score, in the PPCS group as compared to non-PPCS, not only at T2, but also at T1.

Conclusions: We tried to predict – and proposed to prevent – the negative course of MTBI, thanks to a complaints questionnaire and a few quick neuropsychological tests. This preliminary study comes prior to a current study of an MTBI population of 200, in neuropsychology and diffusion tensor imaging.

Key words: somatic complaints, neuropsychology, anxiety, depression, pain, quality of life

INTRODUCTION

Traumatic Brain Injury (TBI) is a major cause of death and of disability for persons under the age of 40. The prevalence of disability due to TBI is about 6.2 million persons in Europe, and 5.3 million in the US (Langlois et al., 2006; Tagliaferri et al., 2006).

Each year in France, 100,000 people are admitted to an emergency unit for a traumatic brain injury (TBI). 80% of these are Mild Traumatic Brain Injury (MTBI). The prognosis and recovery are usually satisfactory, and intracranial lesions are unusual (Holm et al., 2005). A few patients show a post-concussion syndrome, which usually resolves in a few days or a few weeks (Levin et al., 1987b; Bohnen & Jolles, 1995; Ponsford et al., 2002; Caroll et al., 2004; Anderson et al., 2006; Sheedy et al., 2006).

However, about 15% of MTBI patients, “the miserable minority” (Wood, 2004), will suffer a negative course, characterized by a persistent post-concussion syndrome (PPCS). The same prevalence has been observed in international reports (Caroll et al., 2004).

The post-concussion syndrome (PCS), defined by DSM-IV-R (APA, 1994) or ICD 10 criteria, is characterized by somatic, cognitive, emotional and behavioral complaints. After a three-month period, clinical practitioners refer to it as a persistent post-concussion syndrome (PPCS) (Rutherford et al. 1977; Dikmen et al. 1986; Levin et al. 1987a; Mittenberg and Strauman 2000; Ponsford & al. 2000; Carroll & al. 2004; Wood, 2004; Iverson 2005).

Persistent complaints are relatively stereotyped, often numerous, but with varying acuteness from one complaint to another. Somatic complaints are as follows:

- headaches/pains;
- fatigue;
- dizziness;
- sleep disorders (hypersomnia, insomnia);
- feeling of an empty head or a full head;
- balance disorders;
- sensory disorders (phosphene, tinnitus);
- intolerance of noise.

Cognitive complaints include memory capacity, data processing speed, and executive functions, such as cognitive plasticity and attention capacity (Gronwall, 1976, 1977; Stuss et al., 1989; Stablum et al., 1994; Ponsford et al., 2000; Chan et al. 2003; Vanderploeg et al., 2005). Mood and behavior disorders mainly present as a depressive syndrome and an alteration of personality (irritability, apathy, lack of interest in the environment) (Levin et al., 1987b; Parasuraman et al., 1991; McAllister, 1992 ; Bohnen & Jolles, 1992, Bohnen et al.,1995; Brooks et al., 1999; Belanger & Vanderploeg, 2005; Lundin et al., 2006). The persistent PCS (PPCS) is often recognized as a potential risk factor for long-term disabilities (Yang et al., 2009). These

persons not only lack productive capacities, resulting in consequences for private and professional areas of life, but also represent a burden for public health at the national level.

Since these complaints are rarely visible as abnormalities in the scanner or the standard MRI, the lack of knowledge regarding the pathophysiology of PCS has led such complaints to be classified for many years under the ambiguous term “subjective syndrome.” Differing descriptions proposed by the DSM IV-TR and the ICD 10 also illustrate how difficult it is to define PCS (Boake et al., 2004). For several years now, however, the knowledge of both PCS and PPCS has improved (Lankford et al., 1994; Bohnen et al., 1995; Spikman et al., 1996; Bogduk, 2000; de Kruijk et al., 2002; Kurça et al., 2006; Covassin et al., 2008), especially due to the introduction of more accurate technologies in the field of brain imaging, such as diffusion tensor imaging. Abnormalities resulting from diffuse axonal injury have been objectivized through these new techniques (Arfanakis et al., 2002; Inglese et al., 2005; Rutgers et al., 2008). The resultant observations suggest an organic component in persistent disorders after an MTBI. However, looking for factors of organic origin does not belittle the role played by psychological factors in PPCS (Suhr & Gunstad, 2002 ; Bryant et al., 2003; Wood, 2004; Meares et al., 2006; Whittaker et al., 2007; Bigler, 2008).

Nowadays, most authorities believe that there are multiple causes for the occurrence and persistence of the complaints (Kay, 1992; Chan, 2001a, 2001b; King, 1996), both organic and psychological. Rutherford (1989), for example, makes a distinction between early and late-developing disorders. Early-developing disorders would tend to be more organic-related, implying physiological mechanisms triggering headaches, dizziness, nausea and visual disorders, whereas late-developing disorders would rather originate from psychological factors, and would express themselves via attention and memory disorders, increased fatigue and sleep disorders, or irritability. Nevertheless, the relative proportion of organic and psychological patho-mechanisms are difficult to determine (Wood, 2004).

Much research has attempts to identify the factors presently immediately after an MTBI that would predict PPCS (Ponsford et al., 2000; Carroll et al., 2004; Wood, 2004; Bigler, 2008; Kashluba et al., 2008; Dischinger et al., 2009; Yang et al., 2009). These risk factors can be broken down into three large categories:

- Risk factors linked to the injured person: coagulation disorders, age group (under 2 and over 60), intoxication (alcohol, drugs), isolation or environmental and social problems, stress or depression at the time of the trauma;
- Risk factors linked to the violence of the trauma: speed of the vehicle over 50km/h, biker riding over 30 km/h, biker ejected off his/her bike, degree of damage to the vehicle, freeing from the vehicle, pedestrian or biker injured by a car, aggression, falls over 6 meters, undetermined circumstances;

- Risk factors linked to initial severity: focal neurological deficit, convulsions, vomiting, headache, Glasgow coma score under 15, loss of consciousness, persistent post-traumatic amnesia, over 30 minutes of retrograde amnesia, head or neck trauma, including fracture (Vos et al., 2006).

The main objective of our study lies in predicting, as early as possible, from the injured person's complaints and a quick neuropsychological assessment, which MTBI persons risk a negative course leading to PPCS. Accordingly, we separated, more than 3 months after the MTBI, a group with a positive course (non-PPCS) and a group with a negative course (PPCS), using the complaints questionnaire derived from the ICD 10 criteria for PPCS, together with simple psychological and neuropsychological tests.

MATERIAL AND METHODS

Design

This prospective, multicentered, open longitudinal study dealt with persons who had suffered an MTBI and were evaluated twice: firstly at an early stage, between 8 and 21 days after the accident (T1), and then again between the 3rd and 4th months after injury (T2).

Population

We initially recruited 58 patients upon admission to the emergency units of four French hospitals in Paris (University Hospital Kremlin-Bicêtre and Tenon) and in the provinces (University Hospital of Lille and Nantes). Of these patients, 3 were excluded and 26 were reevaluated at T2.

Procedure

A medical doctor collected clinical data and selected patients for the study. The injured persons had to meet the MTBI diagnostic criteria described by the American Congress of Rehabilitation Medicine (1993). The study was approved by the local ethics committee. Informed consent was obtained from all subjects. The examination included a medical check-up, with MRI, and then psychological and neuropsychological tests, at T1 and T2.

At T2, we divided the patients into two groups: one with "good prognosis," called the "non-PPCS group," and the other with a poor prognosis, called the "PPCS group." This was based on a complaints questionnaire derived from ICD 10 diagnostic criteria (Caplain et al., 2008).

The inclusion criteria were as follows: traumatically-induced physiological disruption of brain function manifested by at least one of the following:

- loss of consciousness (<30 min; GCS 13-15);
- loss of memory (<24 hours);
- any alteration in mental state;
- focal neurological deficits.

The exclusion criteria were as follows:

- intubated or ventilated patient, or patient having received sedatives upon arriving at hospital;
- medullary trauma combined with neurological signs or an incapacitating polytrauma (with at least an associated life-threatening lesion);
- cranial trauma that occurred in a suicide attempt;
- psychologically incapacitating trauma that could interfere with the follow-up or evaluations;
- patient under psychotropic treatment at the time of trauma, with hospital antecedents in a specialized psychiatric environment or one/several sick-leave period(s) for psychological reasons;
- progressive neurological disease or drug addiction.

Measurements

To identify post-concussion syndrome, complaints and pains, we referred to the ICD 10 post-concussion criteria. Some recent studies (Boake et al., 2004; McCrea, 2007) have shown that the PCS criteria in the ICD 10 might present a more comprehensive description and a higher sensitivity to make a PPCS diagnosis, as compared to DSM-IV. The diagnosis of PCS requires three of the following symptoms, grouped here into three categories:

- somatic disorders (headaches, fatigue, dizziness, fainting, intolerance of noise);
- psychological disorders (irritability, anxiety, depression, emotional lability);
- subjective cognitive disorders (concentration, memory, intellectual disorders), without significant deterioration at a neuropsychological level.

A complaints questionnaire composed of the three categories of symptoms defined by ICD 10 was administered. The typical complaints mentioned by patients are as follows:

- Somatic complaints:
  - tiring quickly;
  - increased sleep time;
  - trouble with tolerance to noise;
  - need for quiet environment;
  - dizziness;
  - headaches.
- Cognitive complaints:
  - slowness;
  - memory;
  - concentration;
  - trouble in doing two things at the same time;
  - discomfort in performing tasks in a chronological sequence.
- Mood and behaviour complaints:
  - irritability;
  - lack of initiative and motivation;

– anxiety;
– trouble doing things spontaneously;
– tendency to remain idle for long periods of time;
– difficulty controlling reactions;
– feeling sad.

In addition to this questionnaire, two analogue visual scales (VAS) were used to measure, on the one hand, the pain linked to headaches and, on the other hand, any other acute pain described by the patient.

Neuropsychological tests

A one-hour neuropsychological check-up was administered at T1 (between 8 and 21 days), then at T2 (between 3 and 4 months). This check-up aimed at evaluating the cognitive functions which are the most frequently involved in a TBI: working memory, reactive flexibility, inhibitory control and attention processing (focused and divided attention). It included the following tests:

- forward and backward digit spans of the Wechsler Memory Scale (WMS III);
- the Trail Making Test B (TMT B) (Reitan, 1958);
- the number/letter sequence of the WSM III;
- the STROOP test (Stroop, 1935);
- verbal fluency in one minute (semantic category “animals” and phonemic category letter „m”) (Thuillard & Assal, 1983);
- the dual task of Baddeley (1986).

Mood, anxiety and quality of life evaluations

We evaluated mood disorders by using the DSM-IV-R depression criteria, together with the HAD (Hospital Anxiety Depression Scale), which determines how acute a depressive and anxious syndrome is.

A visual analogue scale (VAS) evaluated the subjective quality of life.

Statistical analysis

Epidemiological data were based on descriptive statistics of average and standard deviation according to age, socio-educational level, sex and the type of accident.

The comparison of neuropsychological, psychological, pain and quality of life variables between the two evaluation times (T1 and T2) for the two sub-groups was based on the t-Student test and the Wilcoxon test.

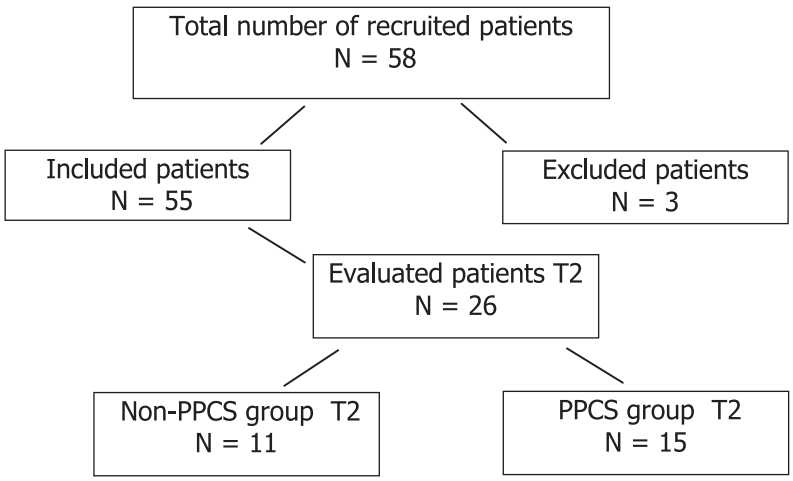
Statistical data were processed with „R” and STATISTICA 7© software.

RESULTS

Characteristics of the non-PPCS group and the PPCS group

Three months after a TBI (T2), 26 MTBI subjects were reevaluated. We then divided them into two groups (a non-PPCS group and a PPCS group)

Table 1. Characteristics of the mild TBI group



	T1	*T2*
Number of patients	55	26
Age (average and standard deviation)	32.98 (11.72)	31.27 (9.91)
Distribution male/female	19 F (34%) ; 36 M (65%)	9 F (34%) ; 17 M (65%)
Socio-Cultural level (average and standard deviation)	3.18 (1.35)	3.63 (1.25)
Types of accidents RA = Road Accident	Work Accidents : 3 RA car : 7 RA motorbike : 5 RA bike : 1 RA pedestrian : 8 Falls : 17 Agressions : 9 Sports accident : 2 Other : 2	Work Accidents : 0 RA car : 2 RA motorbike : 3 RA bike : 1 RA pedestrian : 4 Falls : 10 Agressions : 3 Sports accident: 2 Other : 1

based on the number of complaints collected from the complaint questionnaire derived from the ICD 10 post-concussion syndrome criteria.

Neuropsychological and psychological results for non-PPCS and PPCS groups at T1

The Student t test for independent samples at T1 showed a significant difference between the two groups for the average scores of:

- total digit spans from WMS III ($p < .05$);
- the Stroop test ($p < .05$);
- category fluency "Animals" ($p < .05$);
- the HAD anxiety reference ($p < .05$);
- the global quality of life ($p < .05$);
- the number of complaints ($p < .01$).

The neuropsychological results of the PPCS group were not as high as those of the non-PPCS group, except for the Baddeley's double-task score. Comparing results on the different scales, we observed a higher score in complaints with the PPCS group regarding anxiety, pain, and general complaints, as well as a lower quality of life score; these differences are statistically significant.

The Wilcoxson test showed statistically significant differences between the two groups with respect to mood and cognition from the complaint questionnaire. The PPCS group mentioned more complaints on the three dimensions of the questionnaire than the non-PPCS group. Besides, their complaints correlated in a statistically significant way with the two visual analogue scale scores for pain (headaches: 0.56 and other pain: 0.62), plus the anxiety dimension of HAD (0.72).

Table 2. Characteristics of the two groups: Non-PPCS and PPCS

	Non-PPCS group	*PPCS group*
Number of patients	11	15
Age (average and standard deviation	30 (12.7)	31 (7.71)
Distribution male/female	2 F ; 9 M	6 F ; 9 M
Socio-Cultural level (average and standard deviation)	3.09 (1.14)	3.93 (1.16)
Types of accidents RA = road accident	Work Accidents: 0 RA car: 0 RA motorbike: 2 RA bike: 0 RA pedestrian: 1 Falls: 6 Aggressions: 1 Sport accident: 0 Other: 1	Work Accidents: 0 RA car: 2 RA motorbike: 1 RA bike: 1 RA pedestrian: 3 Falls: 4 Aggressions: 2 Sport accident: 2 Other: 0

Table 3. Significant differences between the two groups (non-PPCS vs PPCS) at T1

Tests and scales	N	GROUPS	average	SD	p
Backward digit spans	11 15	Non PPCS PPCS	7.63 5.8	1.91 1.01	p<.01
Total digit spans MEM III (standard score)	11 15	Non PPCS PPCS	10.54 7.64	2.50 3	p<.01
Trail Making Test B (Raw time scores)	11 15	Non PPCS PPCS	62.90 104.53	18.12 59.56	p<.05
Stroop test – " words "	11 15	Non PPCS PPCS	52.63 44.20	8.77 9.51	p<.05
Stroop test – " colours "	11 15	Non PPCS PPCS	54 47.26	5.79 7.28	p<.01
Stroop test – words / colours	11 15	Non PPCS PPCS	59.09 50.40	10.63 10.25	p<.05
Category fluency " animals "	11 15	Non PPCS PPCS	24.45 19.13	5.61 5.97	p<.05
HAD A	11 15	Non PPCS PPCS	4.18 7.93	1.53 3.47	p<.01
Global quality of life	11 15	Non PPCS PPCS	7.45 5.7	.65 2.31	p<.01
Complaints questionnaire	11 15	Non PPCS PPCS	3.09 8.73	3.14 3.32	p<.001

Neuropsychological and psychological results for non-PPCS and PPCS groups at T2

The Student t test for independent samples showed statistically significant differences between the two groups on the neuropsychological tests evaluating attention, working memory, inhibition and mental flexibility. Also, anxiety, depression, pain and complaints scores were significantly higher, with a poorer quality of life, for PPCS subjects, compared to non-PPCS MTBI patients (Table 4).

Course of neuropsychological and psychological changes between T1 and T2

In the non-PPCS group, from a descriptive point of view, between T1 and T2, the average scores in neuropsychological tests were within the norms, and were better at T2. On the two visual analogue scales of pain, there was a statistically significant improvement with headaches ($p < .05$) and any other pain ($p < .05$). This result was confirmed with the complaints scale, in which the item “headaches” decreased to a statistically significant extent ($p < .05$).

In the PPCS group, between T1 and T2, reactive flexibility scores as well as data processing speed improved to a statistically significant extent ($p < .05$).

Conversely, although the results were not statistically significant, the acuteness of headaches tended to increase (1.40 at T1 and 2.03 at T2), as well

Table 4. Significant differences between the two groups (non-PPCS vs PPCS) at T2

Tests and scales	N	GROUPS	average	SD	p
Total spans MEM III (standard scores)	11 15	Non PPCS PPCS	11.90 8.64	3.53 1.44	P< .01
Trail Making Test B (Raw time scores)	11 15	Non PPCS PPCS	54.09 79.80	16.41 30.90	p< .05
Sequences letters / figures MEM III (standard scores)	11 15	Non PPCS PPCS	12.09 8.42	3.04 2.31	p< .01
Stroop test - words / colours	11 15	Non PPCS PPCS	64.81 50.93	9.62 9.07	p< .001
HAD A	11 15	Non PPCS PPCS	4.90 8.86	1.51 2.94	p< .001
HAD D	11 15	Non PPCS PPCS	1.45 4.6	1.50 2.09	p< .01
Headaches	11 15	Non PPCS PPCS	0 2.03	0 2.53	p< .05
Other pains	11 15	Non PPCS PPCS	.22 3.53	.60 3.3	p< .01
Global quality of life	11 15	Non PPCS PPCS	7.81 6.3	.68 1.77	p< .05
Complaints questionnaire	11 15	Non PPCS PPCS	1.6 10.86	2.01 3.04	p < .001

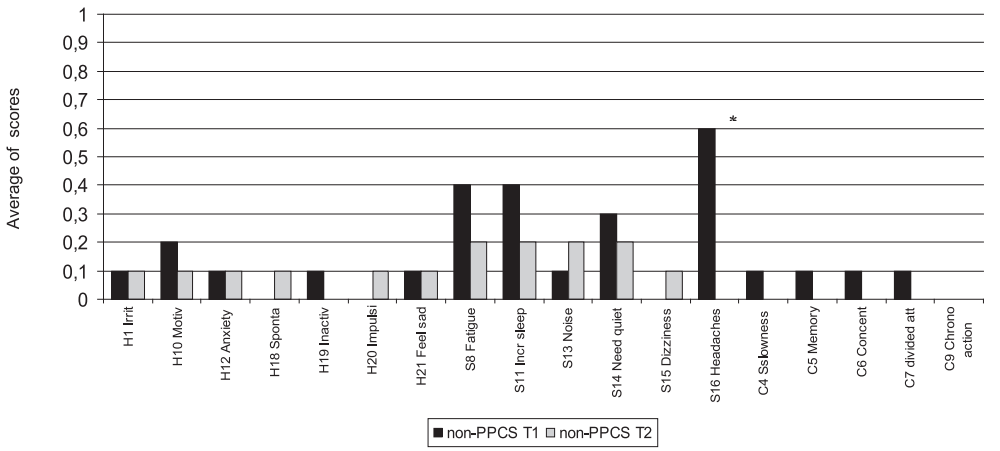
as the acuteness of other pain (2.6 at T1 and 3.53 at T2) and anxiety (7.93 at T1 and 8.86 at T2).

At T2, the number of complaints in the PPCS group was higher than in the non-PPCS group. Statistically significant differences ($p < .05$) were manifested in the following three dimensions:

1) Somatic dimension: fatigue and increase in sleep time, need for peace and quiet, headaches;
2) Cognitive dimension: slowness, memory, concentration, divided attention, trouble with doing things in chronological sequence;
3) Mood and behaviour dimension: irritability, lack of initiative and anxiety.

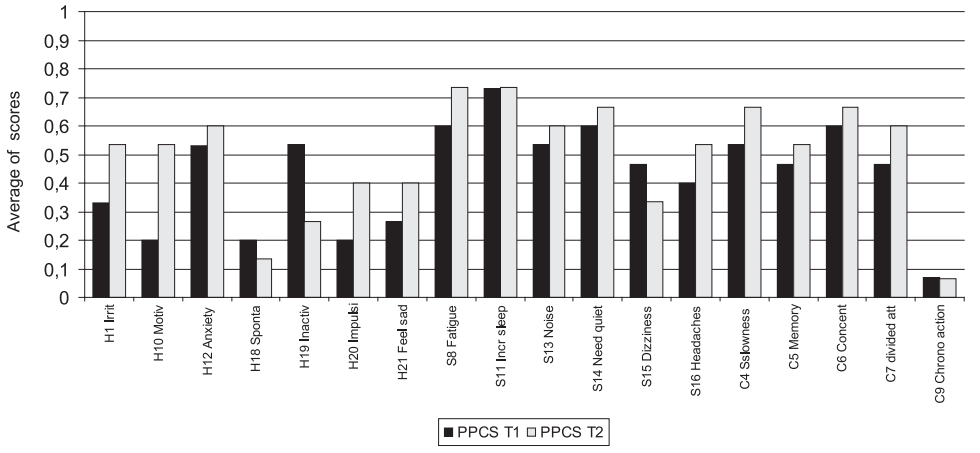
In addition, at T2, analysis of correlations between the complaint scale (somatic, cognitive, mood and behavior dimensions) and the other psychological and neuropsychological tests (see Figs. 1 and 2) shows that, with PPCS subjects:

- the acuteness of headaches is positively correlated to all somatic complaints ($p < .05$);
- the level of anxiety and depression – indicated on the HAD – is correlated significantly to all of the complaints that are linked to mood ($p < .05$);
- the performance indicators in neuropsychological tests (short-term memory, work memory, reactive flexibility and inhibition capacities) are correlated to all cognitive complaints ($p < .05$);



* significance at $p < 0.01$

Fig. 1. Non-PPCS group complaints per dimension between T1 and T2



Graphs key

Mood and behaviour dimension: H1, irritability ; H10, lack of initiative and motivation ; H12, anxiety ; H18, difficulty doing things spontaneously ; H19, tendency to remain idle for long ; H20, difficulty controlling reactions ; H21, feeling sad.
Somatic dimension: S8, quick fatigue ; S 11, increased sleep time ; S13, trouble with tolerance to noise ; S14, need for quiet environment ; S15, dizziness ; S16, headaches.
Cognitive dimension: C4, slowness ; C5, memory ; C6, concentration ; C7, difficulty in doing two things at the same time ; C8, uncomfortable when achieving a sequence of chronological tasks.

Fig. 2. PPCS group complaints per dimension between T1 and T2

- the complaints in each of the 3 dimensions of the questionnaire show no correlation.

DISCUSSION

The main objective of our study was to identify, as soon as possible after a mild traumatic brain injury, those patients at risk for a negative course,

leading to PPCS. In order to do that, we relied on subjective complaints and a quick neuropsychological assessment. This preliminary study was intended to identify those complaints and neuropsychological results which are predictors of PPCS.

To this end, more than 3 months after the injury, we differentiated a positive course group (non-PPCS) and a negative course group (PPCS), with the help of a complaints questionnaire based on the ICD-10 criteria for post-concussion syndrome.

Although the number of TBI patients in our study is small – 55 evaluated at T1, then 26 reevaluated at T2 – the distribution according to sex, age and type of accident is consistent with the literature (Kurtz & Kurland, 1993; Cassidy et al., 2004).The male population is larger, as men account for more traffic accidents and falls.

At T2, only 26 subjects agreed to be reevaluated. The TBI patients with a PPCS represent more than a half of this sample: 15 versus 11 for non-PPCS. Both sub-groups (non-PPCS and PPCS) were homogeneous in terms of sex, age and socio-educational level.

In the literature, PPCS prevalence is estimated at about 15% of TBI (Wood, 2004). In our study, the overrepresentation of PPCS subjects can be explained by persistent complaints which generate a higher motivation to consult again, compared to subjects with a good outcome (McCullogh & Feinstein, 2003). Moreover, we chose the ICD-10 criteria for PPCS, which offers a more comprehensive description and a higher sensitivity than the DSM-IV-TR criteria, and this could explain the high prevalence of PPCS in our population (McCauley et al., 2007).

Neuropsychological results predicting PPCS

Although the non-PPCS subjects' cognitive results were not deficient, the data set collected via neuropsychological tests shows that the average scores in the different tests, from both groups – PPCS and non-PPCS – were lower at T1 than at T2.

At T1, just like T2, PPCS subjects' scores are significantly lower than those of the non-PPCS group. For the PPCS subjects, disorders persist at T2, with short-term memory, work memory, mental flexibility, and inhibition problems (see tables 3 and 4).

Although typical complaints of divided attention disorders have often been reported after a TBI, we were not able to confirm this with Baddeley's test. The scores on this test were noticeably identical at T1 and T2 in both groups. Unfortunately, tests that assess this function are rare and lack specificity. The TEA, the most efficient test, is rather complex and far too long to administer, and so it cannot possibly be one of the elements of a check-list.

In line with the literature, in particular dealing with sports-related concussions (Warden et al., 2001; Guskiewicz et al., 2003; Mc Crea et al., 2003; Bleiberg et al., 2004 ; Collins et al., 2003), our study shows that a TBI, espe-

cially when followed by brain concussion signs, impacts on cognitive functions at the early phase. However, disorders are minor.

The main conclusion is that there is, in the first week following a TBI, a cognitive deficit that is significantly more serious with future PPCS than with those who will have a good outcome.

Complaints predicting PPCS

Headache and other pains assessed with VAS decrease in non-PPCS and increase in PPCS patients between the first and second assessments. Alexander (1992) showed that patients who feel pain at the time of the accident are more likely to develop chronic pain several months later.

For PPCS patients, anxiety is higher and quality of life is lower at T1 and T2, as compared to the non-PPCS. Therefore the question is whether anxiety and pain play a part in the persistence of a PCS, and whether they are linked with cognitive test results. Thus the improvement of cognitive results at T2 in the non-PPCS group could very well be explained by their pain disappearing, as opposed to the PPCS group.

Several types of research have actually demonstrated the deleterious effects of pain on cognition (Uomoto & Esselman, 1993; Hart et al., 2000; Smith-Seemiller, 2003; Alfano, 2006; Sheedy et al., 2006). Bigler (2008) put forward the idea that chronic pains are related to dysfunction of some areas of the limbic cortex, resulting in a disregulation of the cognitive and emotional functioning system (we do know how an abnormality in the cingulate cortex can lead to stress).

However, the PPCS group, whose pain increases 3 months after the TBI, will, nevertheless, show improvement in their cognitive results. It is therefore difficult to establish a direct link between the existence of pain and cognitive deterioration.

At T2, in PPCS, we identified the most significant complaints. When comparing the two groups, on the first assessment, we observed that these complaints looked decisive for predicting a PPCS. Thus several somatic and mental signs – connected with the expression of pain – would be two indicators that we should take into account, in the acute phase of a TBI, as risk factors for PPCS.

These results have clinical applications. They can contribute to establishing a “check-list” aimed at identifying, in the emergency unit, those MTBI patients who are most likely to develop PPCS (Bourque, 2000; Vos et al., 2006). Such a check-list could be filled in by an emergency doctor or even a nurse. It should not take longer than ten minutes in order to be accepted in clinical practice. Indeed, the atmosphere of an emergency unit, the frequency of MTBI and the lack of concern regarding them, made a quick investigation mandatory. In addition, there is hardly ever a neurosurgical emergency, which is usually solved by ordering a CT-scan when in doubt. With this last concern, searching for the risk factors of PPCS becomes secondary.

If these risk factors exist, the emergency doctor could give the injured person an information document on what necessary precautions should be taken and the secondary risks of an MTBI, such as the one designed by the association called France Traumatisme Crânien, requested by the French Health Office – Ministère Français de la Santé – (Truelle et al., 2008). J. Ponsford has demonstrated how efficient such explanations can be in decreasing symptoms.

Thus, the emergency doctor could send the injured person to his/her referring physician, prescribe medical treatment for the symptoms, a limited work sick leave, even a progressive return to work. Several hospital experiments (often with empirical data) tend to show how useful – as far as the prevention or limitation of a PPCS are concerned – a short-term secondary visit (neurologist or emergency doctor) can be, perhaps with a neuropsychological exam, even a classical MRI, and, in certain cases, diffusion tensor imaging (Messé et al., 2010).

In conclusion, the early identification of risk factors for PPCS requires a multidimensional approach to MTBI: severe initial symptoms, accident circumstances, socio-demographic and psychological contexts, is recommended by the WHO Task Force (Cassidy et al., 2004) and several authors (Wood, 2004; Bigler, 2008).

Limitations

There were a number of limitations to this study.

First of all, the etiology of the so-called “persistent post-concussion syndrome” has never been agreed upon (Wood, 2004; Iverson, 2005). Most researchers suggest a combination of biological effects of the injury, psychological factors, psychosocial factors and chronic pain (Mittenberg et al., 2000). This multidimensional model stresses the complexity of the syndrome and its persistency. Moreover, post-concussion-like symptoms are common in healthy subjects, personal injury claimants, post-traumatic stress disorder, orthopaedic injuries, chronic pain or whiplash injury (Zasler, 2007).

Secondly, certain factors were not taken into account, as recommended by the WHO Collaborating Center Task Force on MTBI. This has to do with the existence of post-traumatic amnesia associated with a TBI, or even premorbid factors, at a socio-demographic or psychological level (Vos et al., 2006). Our choice was imposed by the simplicity, reliability and necessary speed of data collection, in order to predict a PPCS.

Thirdly, out-patients who recovered quickly were reluctant to report back for a second examination, particularly if that second examination was foreseen several months after the injury. This bias was obvious in this preliminary study. That was the reason why we decided, in further study, to pay the participants.

The last limitation is the limited number of participants, which justifies a much longer cohort study. However, it is noteworthy that, even with such limitation, we managed to identify some significant differences, at an early

stage, between PPCS and non-PPCS, leading to a check-list of health complaints and neuropsychological results as predictors of a subsequent PPCS.

CONCLUSION

In the first weeks after a MTBI, it seems that we can predict a negative outcome at 3 months. This prediction could rely on a self-administered questionnaire listing about 10 complaints, and a few quick and sensitive tests. In this way, persons after an MTBI with a potential risk for PPCS could be identified on admission to the emergency unit. In order to prevent a negative course, early initial care would consist of giving both an informative document and a follow-up scheme to the injured person, as several countries now do (www.lapublishing.com; www.francetraumatismecranien.fr).

This preliminary research was also aimed at defining the psychological tools that would be sensitive to PPCS. A more thorough study is in progress, evaluating correlations between neuropsychological, psychopathological and magnetic resonance imaging (MRI).

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