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TITLE PAGE

A randomised, waiting list controlled trial of cognitive-behavioural therapy for persistent

postconcussional symptoms after predominantly mild-moderate traumatic brain injury

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ABSTRACT

Background

Persistent post-concussional symptoms (PCS) can be a source of distress and disability following traumatic brain (TBI). Such symptoms have been viewed as difficult to treat but may be amenable to psychological approaches such as cognitive behaviour therapy (CBT).

Objectives

To evaluate the effectiveness of a 12-session individualised, formulation-based CBT program.

Method

Two-centre randomised waiting list controlled trial with 46 adults with persistent PCS after predominantly mild-to-moderate TBI (52% with PTA ≤ 24 hours), but including some with severe TBIs (20% with PTA > 7 days).

Results

Improvements associated with CBT were found on the primary outcome measures relating to quality of life (using the Quality of Life Assessment Schedule and the Brain Injury Community Rehabilitation Outcome scale). Treatment effects after covarying for treatment duration were also found for postconcussional symptoms and several secondary outcomes including measures of anxiety and fatigue (but not depression or PTSD). Improvements were more apparent for those completing CBT sessions over a shorter period of time, but were unrelated to medicolegal status, injury severity or length of time since injury.

Conclusions

This study suggests that CBT can improve quality of life for adults with persistent PCS, and potentially reduce symptoms for some, in the context of outpatient brain injury rehabilitation services.

Trial registration

ISRCTN49540320

INTRODUCTION

Traumatic brain injury (TBI) is associated with a range of cognitive, emotional and physical

symptoms. In the context of mild TBI (MTBI), the label postconcussional symptoms (PCS) is

commonly applied to reported problems including difficulty with concentration and memory,

irritability, depression and anxiety, and physical symptoms such as fatigue, headaches, dizziness.[1]

Such symptoms are common in the first few days or weeks after injury, but typically improve and

remit. However, in a minority these symptoms persist.[2] When lasting beyond three months post-

MTBI, they are viewed as problematic and likely to be chronic[3] or even worsen[4] and have

previously been viewed as resistant to treatment.[5] Although the term PCS is typically associated

with MTBI, the same constellation of symptoms can be reported after more severe injuries, although

their aetiology and course may differ.[6] With this caveat, "PCS" will be used here, operationally, to

describe the set of self-reported persistent postconcussional or PCS-like symptoms across a spectrum

of TBI severity.

There is growing evidence indicating that psychosocial factors influence the persistence of PCS after

MTBI [7-11] and overlapping symptoms or outcomes such as anxiety, depression and employment

status in studies including individuals with severe TBIs.[12,13] Lishman[14] suggested that while

direct effects of brain injury may contribute towards early PCS, persistence may increasingly involve

broader psychosocial factors and mechanisms. Similar diathesis-stressor models have been described

[15,16] where symptoms are maintained via "vicious cycles" involving factors indirectly related or

independent of any direct effects of brain injury. If this is the case then psychological approaches to

management may be applicable and effective in helping address or manage symptoms across a

continuous spectrum of TBI severity, including more-than-mild TBIs, acknowledging that the

likelihood and degree of persisting difficulties related to brain injury increases as injury severity

increases.

Although supported by expert opinion, [17] evidence for the effectiveness of cognitive-behavioural

therapy (CBT) to tackle persistent PCS is limited.[18] Trials incorporating CBT for individuals with

TBI have tended to focus on specific symptoms such as depression,[19] insomnia,[20] or

headache.[21] Typically using single-case or case series designs, results do point to possible

improvements in these domains using CBT. There is some support for prophylactic interventions in

patients at risk for persistent PCS in some[22] but not all studies.[23] Only one RCT has specifically

focussed on persistent PCS [24] randomising 20 individuals with mild-moderate TBI (seen on average

between 5 and 6 years post-injury) to a waiting list group or an intensive neuropsychological

rehabilitation treatment program (5 hours per week for 11 weeks) combining CBT and cognitive

remediation. Improvements were demonstrated on a global symptom measure, in symptoms

associated with anxiety and depression, and some measures of cognitive functioning (although not

subjective functioning). The relative contribution of the CBT and cognitive remediation components

could not be assessed and it is unclear whether improvement could be achieved using CBT alone.

The current study tested the impact of individual, formulation-driven CBT, without explicit cognitive

remediation or cognitive rehabilitation. The study was conducted as a preliminary RCT (with waiting

list control) delivered in the context of two outpatient brain injury services. Eligibility criteria were

designed to help ensure that the results would be generalizable to routine clinical practice. It was

predicted that individuals would report reduced PCS and greater quality of life and health status after

receiving CBT when compared with those in the control group. Supplementary analyses examined the

impact on specific domains of PCS and quality of life, such as symptoms associated with depression,

PTSD, pain and fatigue.

METHODS

Participants were randomised to the immediate Intervention group or waiting list Control group: the

latter were offered treatment at the end of the waiting list period. There was no financial incentive or

other compensation offered to participate in the study. Ethical approval was received by the

committees overseeing the two trial sites. The trial was registered with the International Standard

Randomised Controlled Trial Number Register (ISRCTN49540320).

Recruitment

Participants were recruited through consecutive out-patients referrals to two National Health Service secondary/tertiary care brain injury clinics at the Lishman Brain Injury Unit at South London and Maudsley NHS Foundation Trust, London, UK, and the Brain Injury Rehabilitation Unit, at Edgware Community Hospital, London, UK, between March 2003 and June 2009.

Inclusion criteria were age between 18 and 65 at the time of initial assessment; evidence for (at minimum) a mild traumatic brain injury (MTBI)[25] at least six months before; and symptoms consistent with the ICD-10 criteria for Postconcussional Disorder (F07.2), as laid out in the Diagnostic Criteria for Research (DCR-10).[1]

Exclusion criteria were: non-fluent English; Mini Mental State Exam[26] scores of less than 20 and/or Frontal Assessment Battery[27] scores of less than 10; moderate-severe physical disability (Barthel[28] score less than 15); previous receipt of 4 or more sessions of CBT after their TBI; other neurological disorder independent of the TBI (e.g. non-post-traumatic epilepsy); drug/alcohol misuse meeting ICD-10[29] criteria for a dependence syndrome (F1x.2); and clinically-assessed risk of self-harm or severe psychiatric illness necessitating involvement of a Community Mental Health Team.

Potential participants were screened for eligibility at an initial neuropsychiatric or multidisciplinary assessment, and offered a neuropsychological assessment and feedback session if this had not been completed previously. In the absence of consistent contemporaneous injury severity information from GCS or post-traumatic amnesia (PTA), the latter was retrospectively estimated at initial assessment(s) using the Rivermead protocol[30] which provides reasonable accuracy in terms of overall categorical classification of TBI severity.[31] Median PTA duration was 24 hours (Intervention: 30 hours, range 0-1440 hours; Control: 16 hours, range 0-1800 hours).

All eligible participants were invited to participate. After providing informed consent, but before randomization, participants completed the first (T1) set of outcome measures with the study therapist.

Their general practitioner was requested that, as far as possible, any psychoactive medications were kept constant, and to inform the project team of any changes.

Randomisation

Independent randomization was carried out independently by a Clinical Trials Unit using four pre-planned categorical variables for minimisation with an 80/20 minimised/random weighting applied case-by-case: site (Maudsley: Edgware); injury severity (PTA ≤ 24 hours [mild]: > 24hrs PTA ≤ 7 days [moderate]: PTA > 7 days [severe]); length of time since injury (6 to < 12 months: 12-24 months >24 months), and medicolegal status (previous or current involvement, vs. no previous/current involvement). A minimum of 40 participants was recommended for randomization with 4 minimization factors: sample size was determined on that basis, with a minimum of 20 participants per group.

Flow through the study is shown in Figure 1, with 26 randomised to the Intervention arm and 20 to the Control arm. Median interval between injury and randomization was 25 months (Intervention: 28 months, range 6-171); Control: 23 months, range 8-175). Demographic details of the participants are shown in Table 1. There were no significant differences between the two groups at T1 on any minimisation variables (all p's > 0.4) or gender (χ^2 (1)=0.450; p=0.502), age (t (1)=-0.86; p=0.393), education (t (1)=-0.109; p=0.914) or current occupational level (χ^2 (2)=0.073; p=0.964) (Table 1). PTA duration did differ between the groups (Mann-Whitney U=225.5; p=0.44).

Table 1: Individual demographic and injury characteristics at T1 (before randomisation)

	Intervention Group	Waiting List Group	Total sample
	(n = 26)	(n = 20)	(N = 46)
Age	40.1±10.3	43.1±13.1	41.4±11.6
Gender (male)	58% (15)	50% (10)	54% (25)
Education (years)	15.0 ± 2.8	15.1 ± 3.4	15.0 ± 3.0

Qualifications (or equivalent) obtained						
No qualifications	4%	(1)	10%	(2)	7%	(3)
GCSE	31%	(8)	20%	(4)	26%	(12)
A-Level	12%	(3)	25%	(5)	17%	(8)
Undergraduate degree	35%	(9)	20%	(4)	28%	(13)
Postgraduate degree	19%	(5)	25%	(5)	22%	(10)
Employment status						
Full-time	27%	(7)	30%	(6)	28%	(13)
Part-time	19%	(5)	20%	(4)	20%	(9)
Unemployed	54%	(14)	50%	(10)	52%	(24)
Injury Type						
Road traffic accident	62%	(16)	55%	(11)	59%	(27)
Assault	12%	(3)	10%	(2)	11%	(5)
Other	27%	(7)	35%	(7)	30%	(14)
PTA duration (hours)	170 ± 354		138 ± 399		156 ± 370	
≤ 24 hours [mild]	46%	(12)	60%	(12)	52%	(24)
> 24 hours, ≤ 7 days [moderate]	31%	(8)	25%	(5)	28%	(13)
> 7 days [severe]	23%	(6)	15%	(3)	20%	(9)
Time since injury (months)	42 ± 39		34 ± 38		39 ± 39	
6 – 12 months	23%	(6)	35%	(7)	28%	(13)
> 12 months, ≤ 24 months	23%	(6)	15%	(3)	20%	(9)
> 24 months	54%	(14)	50%	(10)	52%	(24)
Previous or current involvement in litigation (yes)	73%	(19)	70%	(14)	72%	(33)
Current litigation (yes)	50%	(13)	40%	(8)	46%	(21)
Site of treatment (% Maudsley)	85%	(22)	90%	(18)	87%	(40)

Values are shown as either mean ± *SD* or percentage (n)

Outcome Measures

Outcome measures are shown in Table 2. Three co-primary outcome measures examined broad

aspects of PCS and their impact. Six secondary outcome measures were used to examine more

specific domains (such as those relating to depression or fatigue), as well as a supplementary quality

of life scale.

Table 2: Primary and secondary outcome measures

Primary outcome measures	Description	Index (cut-off if applicable)
Rivermead Post Concussion Symptoms Questionnaire (RPQ)[32]	16-item measure of the severity and impact of PCS	Total score (>12)[33]
Brain Injury Community Rehabilitation Outcome scale (BICRO-39)[34]	39-item measure of functioning and disability	Total and subscale scores
Quality of Life Assessment Schedule (QOLAS)[35]	10-item personalized measure of quality of life and health status.	Total score
Secondary outcome measures		
Hospital Anxiety and Depression Scale (HADS)[36]	14-item measure of anxiety and depression	Anxiety (HADS-A) and Depression (HADS-D) subscale totals (>10).
Impact of Event Scale - Revised (IES-R)[37]	22-item measure of PTSD	Total score (>32)[38]
Checklist of Individual Strength (CIS20R)[39]	20-item measure of fatigue	Total score (cut-off of ≥40 for the "subjective fatigue" subscale)[40]
McGill Pain Questionnaire (MPQ)[41]	Multi-dimensional assessment of pain	Present Pain Intensity
State-Trait Anger Expression Inventory-2 (STAXI-2)[42]	57-item measure of state/trait anger and anger expression	Anger Expression Index, raw score
EuroQoL from EQ-5D[43]	Visual analogue scale of global	

subjective health rating (0-100).

For the QOLAS, individuals are asked to identify their two greatest problems/issues in each of five different domains: physical health, cognitive problems, psychological issues, social issues, and daily activities. Each problem is rated on a 0-5 scale (from to "no problem" to "it could not be worse") and the items summed. Validity, reliability and sensitivity to change for the QOLAS has been shown in other clinical populations.[35,44] It was judged particularly suitable for assessing PCS due to the potential heterogeneity of symptoms, and to help identify those symptoms most troubling to an individual and prioritise them during treatment.. Blind expert rating of individual responses in the physical, psychological and cognitive domains indicated that 84.2 % were directly/closely related to DCR-10 description of Postconcussional Disorder;[1] 10.2% as indirectly/partially related; and only 5.5% not apparently/obviously related.

With the exception of the EuroQol, higher scores on all of the outcome measures indicate a greater degree of symptoms, distress or impact. All measures were self-report and therefore non-blind. Assessments were completed prior to randomization (T1) and at the end of treatment (Intervention arm) or after 4-months (Control arm) (T2).

Treatment methods: CBT format and content

The planned intervention comprised 12 weekly one-hour sessions of individual CBT, but the protocol allowed for longer inter-session intervals. Treatment was provided by the same therapist (SP), a clinical neuropsychologist with previous experience of CBT in the context of TBI, depression and chronic fatigue syndrome.

Given the heterogeneity of PCS, an individualised, formulation-driven approach within a semi-structured protocol was used. Agenda-based sessions, collaborative target-setting and homework tasks were central features of treatment. Details of the intervention are described in more detail elsewhere

[16] and in the supplementary online information. A session structure similar to that described by Miller and Mittenberg[45] was used. The first three sessions were broadly focussed on problem identification, psychoeducation based on a range of sources, socialising the patient to the CBT model, and formulation. Sessions 4-12 focussed on the individual target problems identified collaboratively with the therapist. In the final 3 sessions, time was increasingly focussed on relapse prevention and how to maintain therapeutic gains. Copies of the protocol and booklets are available from the authors on request.

Central to individual CBT case formulations were (a) that persistent PCS could be maintained or exacerbated by vicious cycles[15] (b) that these cycles could be understood by reciprocal relationships between thoughts, emotions and actions, and (c) that alleviation in symptoms, associated distress and functional limitations was possible by changing thinking and behaviour. Whilst it was recognised in the treatment protocol that persistent cognitive difficulties attributable to the injury might exist for some individuals (especially for more-than-mild injuries),[46] no attempt was made to provide explicit cognitive remediation or rehabilitation. With increasing injury severity and more-than-mild TBIs, psychological mechanisms were framed as likely to still play an important role in maintaining ongoing symptoms via "vicious cycles", presenting a therapeutic target for CBT to help reduce symptoms and their impact. Overt resistance to psychologically-orientated treatment did not appear to be a common issue for individuals completing the initial screening assessments but who did not wish to take part in the trial. Although the reasons for declining participation were not systemically collected, of the 21 individuals who declined, 9 were prepared to be referred for CBT outside the trial, and another 5 cited distance as an obstacle to attending regular treatment sessions.

Control waiting list group

Individuals randomised to the Control arm received a letter following randomisation with the date of an out-patient appointment to complete the T2 measures. Patients in this arm did not receive any form of additional information or psychological intervention from the service for the period that they were on the waiting list.

Participants in both groups were offered clinical follow-up after their CBT sessions finished.

Statistical Analyses

Analysis was planned on an intention-to-treat basis. In practice almost all individuals in the Intervention arm completed their course of CBT and T2 outcome measures: three individuals stopped after 8, 9 and 10 sessions respectively due to significant symptom improvement and a lack of outstanding treatment targets. Only one patient discontinued treatment after 6 sessions and was lost to follow up due to difficulties attending the sessions. No attempt was made to impute missing data for this one case.

Data were double-entered from the completed paper forms on to computer, and analysed using SPSS 15.0. For the main treatment effects, analysis of covariance (ANCOVA) was used,[47] comparing outcome between groups at T2 with T1 as a covariate: effect sizes are shown using partial eta squared and an estimate of Cohen's d ($\hat{d}$), calculated as the mean difference between the contrasts divided by square root of the mean squared error.

It was predicted that the Intervention arm would report fewer symptoms and better quality of life after CBT compared with those in the Control arm after their time on the waiting list. Given the exploratory nature of the study and the paucity of evidence from relevant previous controlled trials, primary outcome measures were analysed independently without correction for multiple comparisons. For secondary outcomes, corrections for multiple comparisons were applied using the Benjamini and Hochberg method.[48]

Mean duration of the T1-T2 interval for the Intervention arm was 29 weeks (SD=10.3, median=26, range 14-53) and 17 weeks for Control arm (SD=2.9, median=17, range 11-24) (Mann-Whitney -U=46.0; p<0.001), producing a potential confound to the analyses. The analyses therefore also included T1-T2 interval as a covariate.

RESULTS

Data for the primary and secondary outcome measures at T1 (baseline) and T2 are shown in Table 3.

The two groups were broadly similar at baseline in terms of symptoms and quality of life reported:

there were no significant differences between the Intervention and Control arms at T1 on any of the

primary or secondary outcome measures (p>0.05) after correcting for multiple comparisons on the

latter.

Table 3: Summary data for primary and secondary outcome measures

Outcome measure	Intervention Group (n = 25)		Control group (n = 20)	
	T1	T2	T1	T2
RPQ	34.1 ± 11.4 (100%)	26.3 ± 16.4 (76%)	33.4 ± 9.2 (100%)	27.8 ± 9.0 (95%)
QOLAS	35.6 ± 6.9	27.0 ± 10.5	36.4 ± 6.7	33.5 ± 8.2
BICRO-39	85.7 ± 23.1	80.6 ± 24.6	76.5 ± 16.5	80.5 ± 17.3
- Personal care	2.3 ± 6.3	1.7 ± 3.9	0.2 ± 0.7	0.3 ± 1.1
- Mobility	7.2 ± 7.4	6.5 ± 6.7	4.4 ± 3.7	5.4 ± 5.2
- Self-organisation	9.2 ± 8.7	9.0 ± 8.5	7.3 ± 8.0	7.7 ± 7.2
- Contact (partner/children)	5.7 ± 3.9	5.4 ± 4.4	3.1 ± 3.6	3.8 ± 3.3
- Contact (parents/siblings)	10.1 ± 3.1	10.5 ± 2.5	9.5 ± 4.7	10.0 ± 3.7
- Socialising	18.6 ± 5.7	18.8 ± 4.3	19.1 ± 5.0	7.0 ± 5.2
- Productive employment	16.5 ± 2.8	15.6 ± 3.6	15.7 ± 3.5	15.9 ± 3.7
- Psychological well-being	16.1 ± 6.4	13.2 ± 6.9	17.3 ± 5.8	17.4 ± 7.0
HADS-A	9.9 ± 4.8 (44%)	8.9 ± 4.9 (40%)	11.8 ± 3.9 (70%)	11.0 ± 4.1 (55%)
HADS-D	8.9 ± 4.0 (44%)	7.7 ± 5.0 (28%)	8.9 ± 3.3 (40%)	8.6 ± 4.5 (20%)
IES-R	27.0 ± 20.1 (32%)	20.8 ± 18.7 (28%)	29.5 ± 20 (40%)	25.3 ± 19.9 (40%)
CIS20R	98.2 ± 19.5 (76%)	86.8 ± 25 (40%)	104.2 ± 18 (75%)	100.8 ± 26.3 (75%)
STAXI-2	41.8 ± 14.1	36.7 ± 16	42.1 ± 17.6	40.4 ± 18.0
MPQ	1.6 ± 1.3	1.7 ± 1.3	1.2 ± 1.0	1.4 ± 0.9

| EuroQol | 57.2 ± 15.9 | $65.3 \pm 16.3^*$ | 46.7 ± 15.5 | 52.7 ± 18.4 |

Values are mean ± SD (% above cut-off, where applicable); table shows data for individuals with data at both T1 and

T2 (n=45); *n=24 due to missing data

Effects of treatment on primary and secondary outcome measures

A significant treatment effect was noted for quality of life as assessed by the QOLAS (Table 4 &

Figure 2). For postconcussional symptoms (RPQ) the findings were only significant when using T1-

T2 interval as a covariate (Table 4).

No treatment effect was noted in terms of global functioning (BICRO-30). Previous research into

broader community rehabilitation using the BICRO-39 has indicated individual scales may be more

sensitive to change than the total score:[49] these scale scores are also given in Table 4. Psychological

Well-Being and Socialising showed evidence of a treatment effect (the latter only when using T1-T2

interval as a covariate), with little overall change in the other subscales in either group.

Table 4: Analyses of primary and secondary outcome measures, with T1-T2 interval as covariate

			Without T1-T2 interval as covariate							With T1-T2 interval as covariate						
		T1 (adjusted)	T2 (adjusted)		Difference	95% CI		p	Effect Size		T2 (adjusted)		Difference	95% CI		p	Effect Size	
			Intervention	Control					†	‡	Intervention	Control					†	‡
Primary	RPQ	33.78	26.00	28.10	2.09	(-3.85 to 8.03)	$F(1, 42) = 0.51$	0.481	0.012	0.21	23.62	31.07	7.46	(0.42 - 14.50)	$F(1, 41) = 4.578$	0.038*	0.100	0.81
	QOLAS	35.93	27.32	33.11	5.79	(0.97 to 10.61)	$F(1, 42) = 5.886$	0.020*	0.123	0.73	25.64	35.21	9.57	(3.78 - 15.37)	$F(1, 41) = 11.120$	0.002*	0.213	1.26
	BICRO-39	81.57	77.87	83.97	6.10	(-4.35 to 16.55)	$F(1, 42) = 1.387$	0.245	0.032	0.36	77.56	84.35	6.80	(-6.19 - 19.79)	$F(1, 41) = 1.117$	0.297	0.027	0.40
BICRO subscales	Personal care	1.36	1.56	0.45	-1.12	(-2.96 to 0.73)	$F(1, 42) = 1.491$	0.229	0.034	-0.38	1.50	0.53	-0.97	(-3.28 to 1.35)	$F(1, 41) = 0.710$	0.414	0.017	-0.32
	Mobility	5.94	5.74	6.33	0.59	(-2.46 to 3.65)	$F(1, 42) = 0.154$	0.697	0.002	0.12	6.26	5.68	-0.57	(-4.32 to 3.1)	$F(1, 41) = 0.096$	0.759	0.002	-0.12
	Self-organisation	8.36	8.45	8.44	0.00	(-3.26 to 3.26)	$F(1, 42) = 0.000$	0.998	0.000	0.00	9.10	7.63	-1.47	(-5.52 to 2.58)	$F(1, 41) = 0.537$	0.468	0.013	-0.28
	Contact (partner/children)	4.53	4.52	4.95	0.43	(-1.21 to 2.06)	$F(1, 42) = 0.276$	0.602	0.007	0.17	4.15	5.41	1.26	(-0.76 to 3.27)	$F(1, 41) = 1.593$	0.214	0.037	0.50
	Contact (parents/siblings)	9.82	10.29	10.19	-0.10	(-1.25 to 1.04)	$F(1, 42) = 0.033$	0.857	0.001	-0.05	10.40	10.05	-0.34	(-1.80 to 1.11)	$F(1, 41) = 0.229$	0.635	0.006	-0.18
	Socialising	18.82	18.86	20.02	1.16	(-1.07 to 3.40)	$F(1, 42) = 1.108$	0.299	0.026	0.32	17.83	21.32	3.49	(0.89 to 6.09)	$F(1, 41) = 7.325$	0.010*	0.152	1.03
	Productive employment	16.13	15.26	16.23	0.97	(-0.49 to 2.44)	$F(1, 42) = 1.802$	0.187	0.041	0.41	15.34	16.12	0.78	(-1.08 to 2.63)	$F(1, 41) = 0.715$	0.403	0.017	0.32
	Psychological well-being	16.60	13.66	16.77	3.11	(0.42 to 5.81)	$F(1, 42) = 5.429$	0.025*	0.114	0.70	12.54	18.18	5.64	(2.49 to 8.80)	$F(1, 41) = 13.069$	0.001*	0.242	1.37
Secondary	HADS-A	10.74	9.43	10.37	0.94	(-1.41 to 3.30)	$F(1, 42) = 0.654$	0.423	0.015	0.25	8.25	11.84	3.59	(1.03 to 6.15)	$F(1, 41) = 8.003$	0.007*	0.163	1.06
	HADS-D	8.90	7.70	8.62	0.92	(-1.06 to 2.91)	$F(1, 42) = 0.882$	0.353	0.021	0.28	7.25	9.19	1.94	(-0.531 to 4.44)	$F(1, 41) = 2.513$	0.121	0.058	0.60
	IES-R	28.11	21.48	24.40	2.92	(-5.49 to 11.32)	$F(1, 42) = 0.044$	0.853	0.012	0.26	19.19	27.26	8.06	(-2.28 to 18.41)	$F(1, 41) = 2.479$	0.123	0.057	0.59
	CIS20R	100.82	89.04	97.98	8.94	(-3.48 to 21.36)	$F(1, 42) = 2.112$	0.153	0.048	0.44	85.26	102.70	17.44	(2.34 to 32.53)	$F(1, 41) = 5.441$	0.025*	0.117	0.89
	STAXI-2	41.93	36.80	40.30	3.50	(-2.65 to 9.65)	$F(1, 42) = 1.317$	0.258	0.030	0.34	34.31	43.41	9.10	(1.826 to 16.370)	$F(1, 41) = 6.384$	0.015*	0.135	0.95
	MPQ	1.40	1.61	1.51	-0.10	(-0.71 to 0.51)	$F(1, 42) = 0.112$	0.739	0.003	-0.10	1.57	1.56	-0.12	(-0.78 to 7.52)	$F(1, 41) = 0.001$	0.974	0.000	-0.12
	EuroQol	52.57	69.93	55.59	-7.34	(-17.46 to 2.77)	$F(1, 41) = 1.433$	0.238	0.050	-0.47	67.74	49.82	-17.92	(-28.57 to -7.26)	$F(1, 40) = 11.555$	0.002*	0.224	-1.31

*p<0.05; †partial eta squared; ‡Cohen's đ

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FIGURE 2 AROUND HERE

For secondary outcome measures, treatment effects were only noted after covarying for T1-T2 interval, for anxiety (HADS-A), fatigue (CIS20R), anger (STAXI-2) and a quality of life (EuroQol). These results remained significant after correcting for multiple comparisons. No treatment effects were found for depression (HADS-D), symptoms associated with PTSD (IES-R) or pain (MPQ).

Examination of the data indicated that, where T1-T2 interval was a significant covariate, *shorter* intervals were associated with *better* outcomes. This is illustrated by the data for PCS symptom severity (RPQ) (Figure 3) after dividing the Intervention arm into those completing treatment more quickly (n=13) and slowly (n=12) based on a median T1-T2 interval split of 188/189 days. Those taking longer to complete CBT show little change while those who completed CBT more quickly demonstrate larger improvements. This was in contrast to the possibility that a longer interval might allow additional time for individuals to show recovery, and be associated with better outcomes.

FIGURE 3 AROUND HERE

Uncorrected *post hoc* t-tests on unadjusted means were completed to explore within-group changes from T1 to T2 on those measures showing a significant difference between the Intervention and Control conditions on ANCOVA at T2 after covarying for treatment duration. For primary outcome measures, both the Intervention (t (24)=3.32, p=0.003) and Control (t (19)=4.00, p=0.001) groups showed statistically significant improvements on the RPQ over this period, with a similar results for the QOLAS (Intervention: t (24)=4.55, p<0.001; Control: t (19)=2.29, p=0.034). The Intervention group showed a significant improvement on the Psychological Well-Being subscale from the BICRO (t (24)=2.99, p=0.006), whereas the Control group did not (t (19)=0.12, p=0.197). Neither group showed evidence of significant improvement on Socialising on the BICRO (Intervention: t (24)=0.12, p=0.903; Control: t (19)=1.35, p=0.197). For secondary outcome measures, the Intervention group showed significant improvements on the CIS-20R (t (24)= 2.91, p=0.008), STAXI-2 (t (24)= 2.20, p=0.038) and EuroQol (t (23)=2.18, p=0.040), whilst the Controls did not show any improvements

on these variables (t (19)=0.71, p=0.785; t (19)= 0.90, p=0.382; t (19)= 1.58, p=0.132). Neither group showed evidence of significant improvement on HADS-A (Intervention: t (24)=1.01, p=0.323; Control: t (19)=1.18, p=0.253).

Factors moderating treatment effects

To explore the possible influence of other factors on improvements in the Intervention group, data from the QOLAS was examined, as it showed the largest effects of treatment. Those factors used as minimisation variables at randomisation were considered separately as additional categorical factors: data stratified by these variables is shown in Table 5. Neither injury severity (F (2)=0.33; p=0.72), length of time since injury (F (2)=1.09; p=0.36), medicolegal status (F (1)=1.20; p=0.29), nor treatment site (F (1)=0.02; p=0.90) were related to outcome. Current (as opposed to either previous or current) medicolegal involvement was also unrelated to outcome (F (1)=0.19; p=0.67).

Table 5: Summary data for QOLAS, stratified by TBI severity, length of time since injury and medicolegal status

	Intervention Group			Control group		
	n	T1	T2	n	T1	T2
TBI severity						
Mild	12	34.0 ± 8.3	24.5 ± 12.0	12	35.8 ± 6.8	32.7 ± 9.9
Moderate	7	37.1 ± 6.0	28.2 ± 8.2	5	37.4 ± 6.9	34.1 ± 3.9
Severe	6	36.9 ± 4.7	30.6 ± 10.1	3	37.2 ± 8.4	35.7 ± 8.0
Time since injury						
6 – 12 months	6	34.9 ± 7.9	21.7 ± 10.0	7	36.6 ± 4.7	33.9 ± 4.8
≤ 24 months	5	30.0 ± 7.0	24.1 ± 7.9	3	34.8 ± 4.4	27.5 ± 11.8
> 24 months	14	37.8 ± 5.5	30.4 ± 10.8	10	36.8 ± 8.6	35.0 ± 9.1
Medicolegal status						
Previous/current	19	36.7 ± 7.0	28.9 ± 9.4	14	36.0 ± 7.4	33.4 ± 8.9

| None | 6 | 32.0 ± 5.4 | 20.9 ± 12.4 | 6 | 37.3 ± 5.0 | 33.8 ± 7.2 |

Values are shown as mean ± SD; table shows data for individuals with data at both T1 and T2 (n=45)

DISCUSSION

This study, consistent with positive findings from a previous RCT,[24] found that individual CBT improved the quality of life for patients with persistent postconcussional symptoms. CBT also appeared to be effective in alleviating postconcussional symptoms in those patients who completed treatment more quickly. Possible benefits on a range of secondary outcomes including anxiety, fatigue and anger were also noted, albeit only after treatment duration was considered as a covariate. Some outcome measures showed medium to large effect sizes from before to after treatment, although the mean scores of the CBT group indicated that a majority of patients were not symptom- or problem-free at the end of treatment (as with the previous trial[24]), although 24% of treatment completers fell below a proposed clinical cut-off of 12 or less on the RPQ.[33] The current study also demonstrates that clinical improvement in PCS is possible using CBT without an explicit cognitive remediation component, and with a programme of only 12 hours of patient-therapist contact. Contrary to expectations, neither medicolegal status, injury severity nor length of time since injury had significant effects on improvements in the CBT group.

Of the outcome measures used, the QOLAS gave the most robust evidence for an effect of CBT, perhaps because it captured specific symptoms identified by the patient as directly relevant to their quality of life. Its identification of "key" problem areas also helped identify personal treatment targets for therapy, increasing its sensitivity to change and its clinical utility. The potential advantages for individualised patient reported outcome measures and their responsiveness to change has led to their appearance into brain injury rehabilitation via methods such as Goal Attainment Scaling.[49]

Contrary to expectations that persistent symptoms are static or may even worsen with time, individuals in the control group did show evidence of improvements over their waiting list period in terms of postconcussional symptoms (RPQ) and their impact on quality of life (QOLAS). The neuropsychiatric and neuropsychological assessment, feedback and prospect of either immediate or

delayed treatment may have contributed to this change, by providing a coherent possible explanation

for symptoms and offering hope that change and improvement was possible.[50]

There was no evidence for significant effects of treatment on depression or PTSD, both of which have

been linked with PCS.[9] This may have been due to floor effects as less than 50% of individuals

scored above clinical cut-offs on the HADS-Depression and RIES at the initial assessment. However,

the results imply that general improvement in PCS and quality of life were not simply mediated by

improved mood.

The impact of the time taken to complete CBT was noteworthy. Not using treatment duration as a

covariate removed positive group differences for postconcussional symptoms (as measured by the

RPQ), although those for quality of life (QOLAS and BICRO-Psychological Well-Being) remained.

Session frequency may be a factor, with evidence that increased intensity may be associated with

better outcomes in psychological therapies in other areas,[51] although this may not always be the

case.[52] Faster completion may also have indicated greater engagement to therapy. Although the

large majority completed therapy, a longer time to complete the CBT sessions was often due to

patients repeatedly cancelling and re-arranging appointments, perhaps reflecting lower commitment.

Whatever the source of this effect, it does indicate that there may be significant variability between

individuals with persistent PCS in their treatment response to CBT.

Caution should be exercised in the interpretation of the null results from the moderator analyses

examining the impact of variables possibly affecting treatment response, such as injury severity and

medicolegal status. The relationship between injury severity and treatment response may be complex:

a residential programme focussing on treating PTSD in military veterans found that individuals with

moderate-severe TBIs showed greater improvements in their PTSD symptoms compared with those

with mild TBIs.[53] Although compensation seeking has been associated with increased symptom

report even after early intervention,[54] medicolegal status itself may serve as a poor proxy for

phenomena such as poor effort or malingering in neuropsychological assessments.[55] The latter is

more likely to affect treatment outcome, but formal cognitive measures of effort were not available

for all participants. More generally, the smaller subgroups used and the corresponding decrease in

power may well have obscured modest influences on treatment outcome, and precluded examining

possible interactions such as medicolegal status playing a more significant role in influencing

outcome in mild but not severe TBI.[8] Nonetheless, the null results for variables that might be

expected to affect treatment outcomes suggests that they may not be *major* determinants of

improvement in CBT, at least for the individuals seen in the current trial.

Study limitations

The variation in time taken to complete the sessions of CBT (with the CBT group taking longer to

complete their treatment than the interval spent on waiting list in the control group) was undesirable,

although the alternative of having a fixed assessment interval regardless of the completeness of

treatment imposes its own difficulties. Use of a cross-over waiting list design complicates the

controlled assessment of the maintenance of therapy benefits and longer-term outcome. Data on other

variables that might have influenced outcomes (such as homework completion, or perceptions of the

usefulness of treatment) were not collected.

Another limitation stems from the use of a single therapist combined with individualised treatment.

Although fitting the needs of patients, rather using a highly manualised protocol, generalisability of

the findings may be limited by the therapist's expertise. Whilst typical of clinical practice, completion

of study questionnaires given by the treating therapist may also contribute a potential response bias.

Future larger/multicentre studies may usefully include a measure of therapist competence and

protocol adherence, and outcome measures given by a non-treating researcher.

Conclusions

The current trial adds to the sparse evidence that the impact of persistent PCS, especially on

individuals' quality of life, can be ameliorated even for individuals sustaining more-than-mild TBIs.

Individual differences in treatment response were noted, as indicated by the impact of treatment

duration, and some treatment effects were only statistically significant after controlling for this variable. An explicit, concomitant cognitive rehabilitation component did not appear necessary for these improvements: further work is needed to examine the value of this approach with or without CBT, whether certain symptoms respond differently to different treatment types, and how best to integrate different treatments both practically and theoretically. As research into psychological factors affecting the development and maintenance of persistent PCS continues, understanding the role of variables such as coping,[11] symptom and injury perceptions,[10,11] and broader personality traits[2] may also help in refining CBT interventions and identifying mediators and moderators of change.[16]

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according to T1-T2 interval

SUPPLEMENTARY ONLINE MATERIAL: ADDITIONAL INFORMATION ABOUT CBT PROTOCOL

Two psychoeducation information booklets were used. The first was a general introduction to PCS based on the material of Mittenberg, Zielinski and Fichera,[1] which reviewed common postconcussional symptoms, expected course of recovery, and the possible role of vicious cycles in maintaining persistent symptoms. The second booklet focussed on cognitive difficulties and reasons why they might persist after a brain injury (including the role of stress, effort and over-exertion);[2] the role of confidence in memory and other cognitive domains, and the impact of mood, rumination and worry;[3] and the possible effects of elements of perfectionism[4,5] and reinvestment.[6]

Where formulations identified overlap with relevant conditions such as anxiety and depression, cognitive-behavioural techniques used were based on the work of Beck,[7] and Hawton and colleagues,[8] and utilised modifications for chronic fatigue syndrome,[9] PTSD[10] and perfectionism[5] when appropriate.

Behavioural components included work on sleep hygiene[8] and behavioural activation,[11] with a particular focus on making sustainable increases in activity over time avoiding "boom-and-bust",[9] as well as addressing possible avoidance and procrastination[12].

Cognitive approaches included negative automatic and dysfunctional thoughts records in responses to symptoms, explored through Socratic questioning, and challenged/tested particular through verbal reattribution techniques or behavioural experiments. A focus on

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beliefs about symptom causes was typically avoided in favour of broader attributions and

implications such as catastrophic misinterpretations to cognitive errors.

Metacognitive beliefs were often an explicit target in the sessions. Amongst other homework

tasks, patients were often encouraged to keep positive data logs[13] to keep track of cognitive

or other successes, to modify attentional bias towards mistakes or failures and build

confidence.

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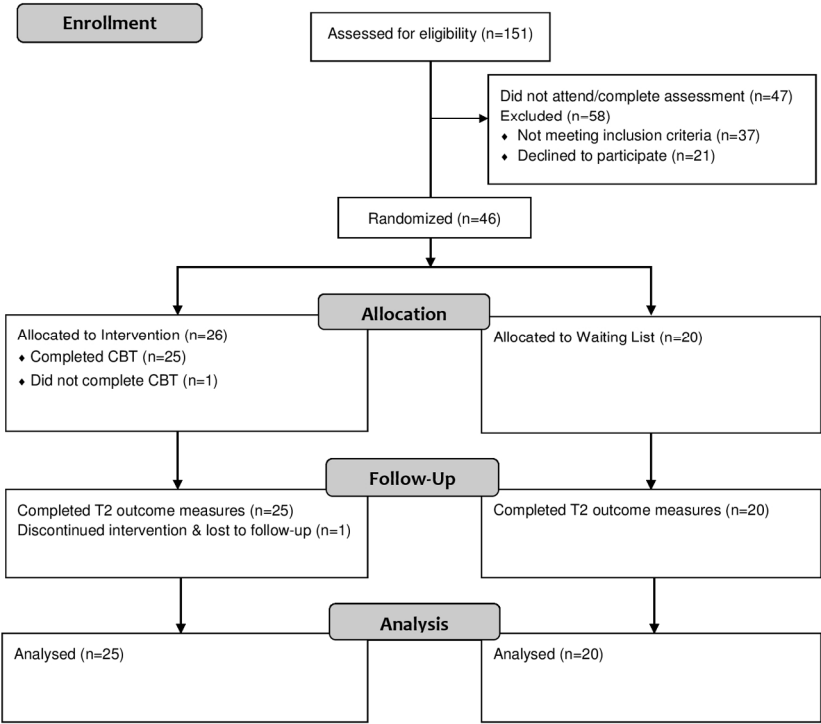45
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Figure 1: CONSORT flow diagram of patient progress through trial
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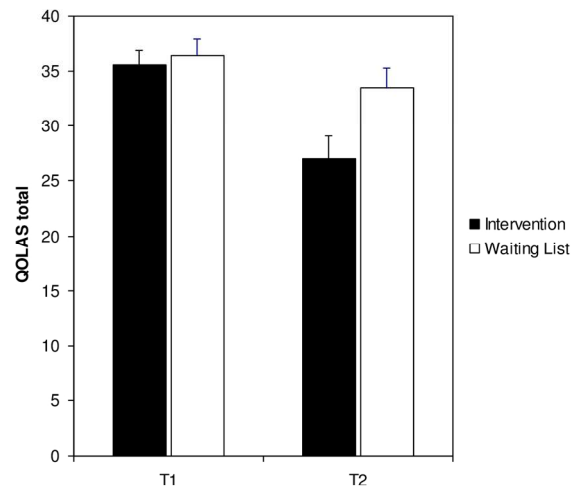
Figure 2: QOLAS total score at baseline (T1) and follow-up (T2)
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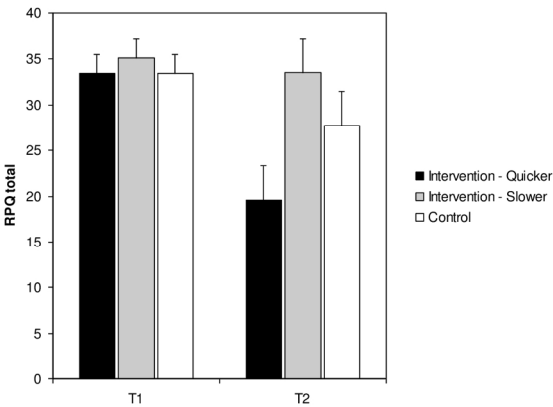51
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Figure 3: RPQ total score at baseline (T1) and follow-up (T2), with Intervention group median-split according to T1-T2 interval
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