



Original Contribution

Psychiatric morbidity following electrical injury and its effects on cognitive functioning

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Abstract

Objective: This study examines the prevalence of psychiatric morbidity in a large sample of electrical injury (EI) patients in three phases of recovery and its effects on cognitive functioning.

Methods: Eight-six self-referred EI patients received psychiatric and neuropsychological evaluations. Descriptive statistics were conducted to examine the prevalence of psychiatric morbidity. Polytomous logistic regression was used to identify predictors of psychiatric diagnosis. Between-subjects analysis of variances (ANOVA) was conducted to examine the effects of psychiatric morbidity on cognitive functioning.

Results: Seventy-eight percent of subjects warranted a psychiatric diagnosis. Long-term patients compared to acute patients were more likely to be diagnosed with two diagnoses than not having any diagnosis (OR=14.30, 95% CI 1.40–38.71). Patients with two diagnoses performed worse than both patients with a single or no diagnosis on all cognitive outcome measures ($P<.05$). Voltage level, chronic pain and litigation status did not predict psychiatric morbidity.

Conclusions: Psychiatric difficulties commonly emerge and persist following EI. EI patients with psychiatric conditions exhibited poorer cognitive performance as compared to EI patients with no post-injury psychiatric difficulties. Health care professionals need to devote careful attention to psychiatric and cognitive status when treating survivors of EI.

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1. Introduction

Psychiatric difficulties commonly emerge after traumatic injuries such as burns and traumatic brain injury (TBI), with prevalence of psychiatric disorders estimated as high as 50% in some studies. For instance, in a 30-year follow-up study of 60 TBI patients, Koponen et al. [1] found that 48.3% were diagnosed with a *Diagnostic and Statistical Manual*

(DSM-IV) [2] Axis I disorder that began after the TBI. Similarly, in a follow-up study of 45 adult burn patients, Madianos et al. [2] found that the prevalence of psychiatric disorders reached 46% at both baseline and follow-up assessments. Thus, traumatic injuries constitute a significant threat to an individual's recovery and carry the potential of long-term changes in psychiatric status.

There has been paucity of research examining the psychiatric manifestations of another form of trauma, electrical injury (EI), despite occupational injury survey data that show that 2287 US workers died and 32,807 workers sustained days away from work due to EI between 1992 and 1998 [3]. Scattered reports suggest that as in other forms of trauma, EI is associated with significant psychiatric

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difficulties including major depressive disorder, anxiety disorders and post-traumatic stress disorder [PTSD] [4–7]. The rates of psychiatric morbidity following EI vary between studies, ranging from 57% [4] to 87.5% [8]. These differences appear to be largely due to varying sample characteristics, diversity of measures used to assess psychiatric status and differences in the statistical control for confounding variables. Specifically, Hooshmand et al. [8] administered the Minnesota Multiphasic Personality Inventory (MMPI) to a sample of 16 EI patients who were evaluated between 3 months to 1 year following their EI. The findings revealed that 14 of the 16 patients suffered from severe depression, and in 11 of the patients, the depression was severe enough to cause significant interference with occupational functioning.

In a study comparing 10 electrical burn patients to small samples of nonelectrical burn patients, chronic pain patients and inpatients on the adult psychiatry unit, Mancusi-Ungaro et al. [7] reported that the electrical burn patients showed the highest levels of distress on the clinical scales of the MMPI and endorsed more depressive symptoms relative to the other clinical samples. However, the small samples precluded any tests of statistical significance of the reported difference between the groups.

Only one study has utilized structured psychiatric diagnostic classification using the *DSM* following EI. Kelley et al. [4] reported that among a sample of 73 EI patients, 57% had psychiatric symptoms that warranted diagnosis of either depression or PTSD, and within this subgroup 31% had both diagnoses. However, this sample consisted of patients who were examined more than 3 months (and in some instances 9 years) following their EI, possibly limiting their generalizability to the EI patient population as a whole. Indeed, only one study has directly investigated the role that post-injury time interval may have on neuropsychiatric presentation after EI. Barrash et al. [9] examined the neuropsychological status in 18 survivors of high-voltage EI who were classified as *acute* (<1 month), *short-term* (1–6 months) or *long-term* (>6 months) related to when they were evaluated after their injury. Deficits in verbal learning and delayed recall of verbal information were present across various time intervals, and depression, anxiety and irritability were reported to be “widespread beyond the acute period”.

To date, there are no published studies that have systematically examined the prevalence of psychiatric disorder in a carefully screened large sample of EI patients in both acute and post-acute phases of recovery. Moreover, in order to further our understanding of the neuropsychiatric sequelae of EI and improve rehabilitation interventions with this population, it is also important to examine the relationship between psychiatric morbidity and neurocognitive performance. Indeed, impairment in memory, attention and executive functioning has been commonly associated with a variety of mood and anxiety disorders in the general psychiatric population [10]. Relatively fewer studies have examined the relationships between these variables in

clinical populations that suffered traumatic injuries. For instance, Rapoport et al. [11] reported a moderate positive correlation between self-report measures of depression and cognitive measures of attention, processing speed and verbal memory in a population of older adults who sustained TBI. Similarly, Ammar et al. [12] found poorer verbal memory performances among EI patient with PTSD as compared to EI patients without PTSD. These findings suggest that increased psychiatric difficulties may be associated with poorer cognitive outcomes among individuals suffering from traumatic injuries, and specifically among EI patients.

The current study examined the prevalence of psychiatric morbidity in a large sample of EI patients in three phases of recovery (acute, post-acute and long-term). We also investigated the relationship between psychiatric status and objective and subjective indices of cognitive functioning.

2. Method

2.1. Subjects

Subjects were 86 consecutive EI patients assessed by the local Electrical Trauma Research Program (ETP) between 1995 and 2005. Individuals eligible for this study were EI patients who received emergency and acute care services at the local medical center, as well as patients who were acutely managed at other institutions and later referred for post-acute multidisciplinary medical evaluation and treatment at our institution. One individual was excluded because his time since injury was greater than 3 S.D. from the group’s mean time since injury. The sample was predominately male (87%). The average age of subjects was 39.45 years (S.D.=9.81). Subjects had an average of 12.66 years of education (S.D.=2.10), with estimated intellectual ability in the average range (mean WAIS FSIQ=96.07, S.D.=13.56). At the time of the evaluation subjects were an average of 20.66 months post-injury (S.D.=20.15). Twenty-six (31%) of the subjects were in the acute stage of recovery (≤ 3 months), 27 (32%) were in the post-acute phase of recovery (>3 to ≤ 24) and 32 (38%) were in the long-term phase of recovery from EI (>24 months). Nineteen subjects (22%) had undergone surgery following their EI (five in the acute recovery phase group, six in the post-acute recovery phase group and eight in the long-term recovery phase group). Fifty-three (62%) of the subjects were hospitalized (18 in the acute recovery phase group, 16 in the post-acute recovery phase group and 19 in the long-term recovery phase group). Seventeen subjects (20%) were taking psychiatric medication at the time of their evaluation (two in the acute phase group, seven in the post-acute phase group and eight in the long-term group). None of the subjects was on thyroid medication and none had abnormal brain imaging findings. None of the subjects was reported to have a concurrent personality disorder. Forty-eight (56%) of the subjects were in litigation (see Table 1 for a detailed presentation of clinical, accident and injury-related variables of EI sample).

t1.1 Table 1

t1.2 Clinical, accident and injury-related variables of EI subjects

t1.3	EI Subjects, n (%)
t1.4 Accident-related variables	
t1.5 Low-voltage injuries	46 (54)
t1.6 High-voltage injuries	39 (46)
t1.7 Head contact with electricity source	3 (4)
t1.8 Injury-related variables	
t1.9 Loss of consciousness	25 (29)
t1.10 Traumatic head injury	1 (1)
t1.11 Surgery	19 (22)
t1.12 Hospitalization	53 (62)
t1.13 Clinical variables	
t1.14 Current chronic pain	43 (50)
t1.15 Current pain medication	17 (20)
t1.16 Current testosterone supplementation	1 (1)
t1.17 Current psychotropic medication	17 (20)
t1.18 Previous psychiatric diagnosis (depression and substance use)	5 (6)
t1.19 History of learning disability	4 (5)
t1.20 History of attention-deficit/hyperactivity disorder	1 (1)
t1.21 Previous exposure to a traumatic event	2 (2)

159 2.2. Procedure

160 The current study was approved by the Institutional
 161 Review Board Human Subject Committee at our medical
 162 center. Prospective subjects were informed that their
 163 participation in the study is voluntary and written informed
 164 consent to participate in the study was obtained. Each indi-
 165 vidual received a comprehensive psychiatric evaluation by a
 166 board-certified psychiatrist as part of their work-up for the
 167 ETP. The evaluation included a patient interview, a review of
 168 previous psychiatric evaluations in order to establish the
 169 existence of a previous psychiatric diagnosis or previous
 170 usage of psychotropic medication when applicable, and the
 171 use of the Structured Clinical Interview for the DSM-III-R
 172 Axis I diagnosis–McLean version [13]. In the present use of
 173 this tool, the wording of the diagnostic criteria has been
 174 modified to reflect *DSM-IV* [14]. As part of their evaluation
 175 at the ETRP, each individual was also assessed by a board-

certified neuropsychologist and was administered a standard 176
 battery to examine cognitive abilities such as memory, 177
 attention, processing speed and executive functioning. The 178
 neuropsychological measures included the California Verbal 179
 Learning Test–2nd Edition [15], Stroop Color Word Test 180
 [16] and the Trail Making Test [17]. All subjects included in 181
 the study showed no evidence of malingering based on their 182
 scores on formal symptom validity tests. 183

2.3. Data analysis 184

Descriptive statistics were utilized to characterize the 185
 incidence of psychiatric diagnosis. A time since injury 186
 variable was created with three ordinal categories each 187
 denoting a recovery phase period: acute ≤ 3 months, post- 188
 acute >3 months and ≤ 24 months, and long-term >24 189
 months (extending taxonomy first described by Pliskin et al. 190
 [18]). Polytomous logistic regression [19] was used to assess 191
 the predictive power of recovery phase from injury (time 192
 since injury) on psychiatric morbidity when other important 193
 injury-related predictors (reported voltage level, litigation 194
 status, previous psychiatric history, previous use of psycho- 195
 tropic medication, current level of pain, current use of pain 196
 medication) and demographic variables (sex, age, race, years 197
 of education) were included in the model. In order to create 198
 the most parsimonious model, terms were eliminated from 199
 the initial model using a backwards elimination procedure in 200
 which nonsignificant ($P > .10$) terms were removed until a 201
 final equation was reached. The model included a three-level 202
 dependent variable (no diagnosis, single diagnosis, two 203
 diagnoses) with no diagnosis as the referent group. 204
 Additionally, polytomous logistic regression was used to 205
 calculate odds ratios (ORs) and 95% confidence intervals 206
 (95% CIs) for each of the predictor variables. Next, between- 207
 subjects analysis of variances (ANOVA) was conducted to 208
 examine differences in cognitive performance between EI 209
 subjects with no psychiatric diagnosis, a single psychiatric 210
 diagnosis or two psychiatric diagnoses. For this analysis, we 211
 computed a composite z-score for three cognitive domains 212

t2.1 Table 2
 t2.2 Predictors of psychiatric diagnosis among EI subjects

t2.3 Adjusted variable	Single diagnosis (n=44) vs. no diagnosis (n=19)			Two diagnoses (n=22) vs. no diagnosis (n=19)		
	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
t2.5 Age			.09			.13
t2.6 ≤ 40 (n=47)	1.00	Referent		1.00	Referent	
t2.7 >40 (n=38)	2.84	0.84–9.57		3.02	0.73–12.50	
t2.8 Education			.76			.04
t2.9 ≤ 12 (n=56)	1.00	Referent		1.00	Referent	
t2.10 >12 (n=29)	0.83	0.25–2.76		0.19	0.04–0.91	
t2.11 Months since injury			.08			.32
t2.12 ≤ 3 (n=26)	1.00	Referent		1.00	Referent	
t2.13 >3 and ≤ 24 (n=27)	3.57	0.88–14.51		2.59	0.40–16.76	
t2.14 Months since injury			.85			.02
t2.15 ≤ 3 (n=26)	1.00	Referent		1.00	Referent	
t2.16 >24 (n=32)	1.41	0.35–5.60		14.30	1.40–38.71	

t2.17 Note: Each variable is adjusted.

t3.3	Diagnoses in EI subjects, <i>n</i> (%)	Acute (≤3 months)	Post-acute (>3 to ≤24 months)	Long-term (>24 months)
t3.4	With psychiatric diagnosis	17 (65)	23 (85)	26 (81)
t3.5	Without psychiatric diagnosis	9 (35)	4 (15)	6 (19)
t3.6	Type of psychiatric diagnosis			
t3.7	Depression	1 (4)	10 (37)	4 (13)
t3.8	PTSD	4 (15)	5 (19)	4 (13)
t3.9	Anxiety disorder	2 (8)	0 (0)	1 (3)
t3.10	Depression+PTSD	4 (15)	3 (11)	11 (34)
t3.11	Depression+anxiety	0 (0)	1 (4)	3 (9)
t3.12	Somatization	0 (0)	1 (4)	2 (6)
t3.13	Adjustment disorder	5 (19)	1 (4)	0 (0)
t3.14	Cognitive disorder NOS	1 (4)	0 (0)	3 (9)
t3.15	Mental disorder NOS	1 (4)	2 (7)	1 (3)

213 (verbal memory, executive functioning and attention) by first
 214 computing within-test *z*-scores followed by between-test
 215 *z*-scores. In addition, the effects of psychiatric morbidity on
 216 self-reported complaints of emotional, cognitive and phys-
 217 ical symptoms were examined using ANOVA. Finally,
 218 Pearson correlations were used to examine the relationships
 219 between the different composite *z*-scores and time since

injury as a function of psychiatric diagnosis. All analyses 220
 were performed using SAS [20]. 221

3. Results 222

3.1. Prevalence of psychiatric morbidity 223

Seventy-eight percent of the total sample of EI subjects 224
 warranted a psychiatric diagnosis based on their evaluation. Of 225
 those with a psychiatric diagnosis, 52% had a single psy- 226
 chiatric diagnosis and 26% had two psychiatric diagnoses. 227

3.2. Predictors of psychiatric morbidity and symptoms profile 228

Recovery phase since EI was a significant predictor of 229
 psychiatric morbidity with long-term subjects significantly 230
 more likely to be diagnosed with two psychiatric diagnoses 231
 than not having a diagnosis as compared to acute subjects 232
 (OR=14.30, 95% CI 1.40–38.71). Specifically, EI subjects 233
 who are longer than 2 years post EI were 14 times more likely 234
 to be diagnosed with two psychiatric diagnoses relative to 235
 EI subjects who are in the post-acute phase of recovery 236
 (>3 months and ≤24 months). There was a trend for post- 237
 acute subjects to be more likely diagnosed with a single 238
 psychiatric diagnosis than not being diagnosed as compared 239
 to acute subjects (OR=3.57, 95% CI 0.88–14.51). Post-acute 240
 subjects were 3.5 times more likely to be diagnosed with a 241
 single psychiatric diagnosis relative to EI subjects who are in 242
 the acute phase of recovery (≤3 months) (*P*=.08; see Table 2). 243

t4.1 Table 4
 t4.2 Performance on objective and subjective cognitive outcome measures as a function of psychiatric diagnosis

t4.3	Outcomes	Psychiatric diagnosis group						<i>P</i> value
		Never diagnosed		Single diagnosis		Two diagnoses		
		Mean	S.D.	Mean	S.D.	Mean	S.D.	
t4.6	Verbal memory composite	0.40 ^a	0.84	0.07 ^a	0.81	−0.45 ^b	0.79	.01
t4.7	CVLT							
t4.8	Total learning across trials	53.63 ^a	12.66	51.34 ^a	11.63	44.32 ^b	12.47	.03
t4.9	Long delay free recall	11.37 ^a	3.02	10.61 ^a	3.76	8.45 ^b	4.23	.03
t4.10	Logical memory							
t4.11	Immediate free recall	18.25 ^a	6.89	14.28 ^b	6.19	11.23 ^c	4.77	.002
t4.12	Delayed free recall	13.66 ^a	6.11	11.94 ^a	5.36	9.63 ^b	4.58	.06
t4.13	Executive functioning composite	0.22 ^a	0.72	0.22 ^a	0.79	−0.59 ^b	0.83	.001
t4.14	Stroop Interference Test							
t4.15	Color–word	37.84 ^a	12.61	38.45 ^a	13.54	26.41 ^b	8.55	.001
t4.16	Trail Making Test							
t4.17	Trails B	75.02 ^a	43.08	79.88 ^a	47.67	119.18 ^b	75.13	.02
t4.18	Attention composite	0.30 ^a	0.81	0.17 ^a	0.76	−0.55 ^b	0.90	.001
t4.19	Stroop Interference Test							
t4.20	Word	92.63 ^a	21.32	87.84 ^a	20.53	72.45 ^b	21.30	.01
t4.21	Color	70.21 ^a	16.02	67.36 ^a	16.38	53.45 ^b	14.50	.001
t4.22	Trail Making Test							
t4.23	Trails A	32.10 ^a	18.26	33.95 ^a	16.03	46.90 ^b	30.74	.04
t4.24	Neuropsychological Symptoms Checklist							
t4.25	Emotion	1.07 ^a	1.53	3.07 ^b	2.39	3.31 ^b	2.56	.02
t4.26	Somatic	3.80 ^a	4.41	10.62 ^b	7.76	10.19 ^b	8.26	.01
t4.27	Cognitive	1.67 ^a	3.20	5.28 ^b	4.98	6.31 ^b	5.12	.02

Note: Psychiatric diagnosis groups (no diagnosis, single diagnosis, two diagnoses). Composite scores were first created by computing within-test *z*-scores followed by between-test *z*-scores. Therefore, the composite scores will not sum to 0 as typically expected.

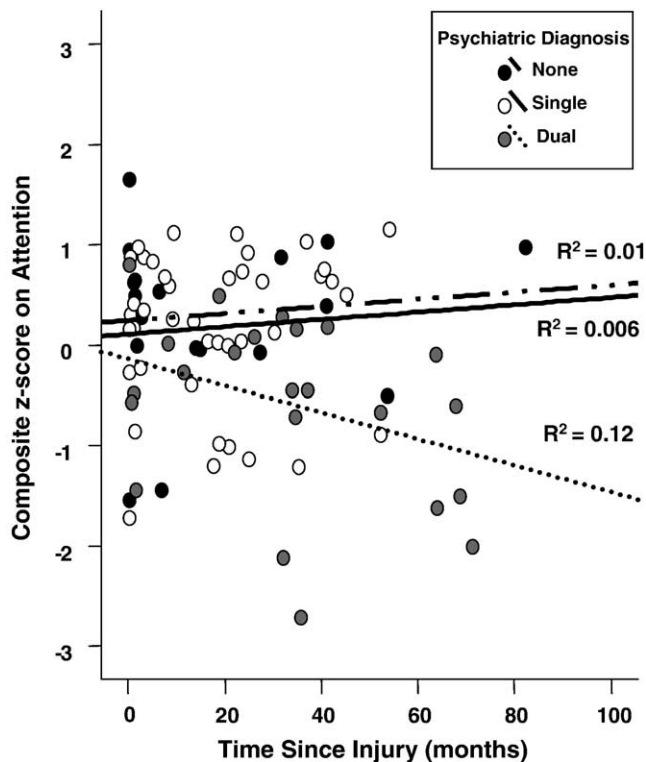


Fig. 1. Relationship between the composite z-score on attention and time since injury as a function of psychiatric diagnosis.

244 Reported voltage level, previous psychiatric history, previous
245 use of psychotropic medication, current level of pain, use of
246 pain medications and litigation status were not significant
247 predictors of psychiatric morbidity

248 Exploratory descriptive analyses of psychiatric incidence
249 were conducted for the three recovery-phase subgroups (see
250 Table 3). Among EI subjects in the acute phase of recovery,
251 65% were given a psychiatric diagnosis. The most prevalent
252 symptom profile was adjustment disorder (19%), followed
253 by PTSD alone (15%) and depression and PTSD (15%).
254 Depression alone was prevalent in 4% of the sample. In the
255 post-acute phase of recovery, the most prevalent symptom
256 profile was depression alone (37%) followed by PTSD alone
257 (19%) and depression and PTSD (11%). Among subjects in the
258 long-term recovery phase from EI, the most prevalent symptom
259 profile was co-occurrence of depression and PTSD (34%),
260 followed by depression alone (13%) and PTSD alone (13%).

261 3.3. Psychiatric morbidity and objective cognitive functioning

262 EI subjects with two diagnoses performed worse than
263 both subjects with a single or no diagnosis on all cognitive
264 outcome measures including verbal memory, executive
265 functioning and attention ($P < .05$; see Table 4).

266 3.4. The association between time since injury and objective 267 cognitive functioning as a function of psychiatric morbidity

268 There was a trend ($r = -.34$, $P = .10$) for a correlation between
269 the composite z-score on the attention domain and time since

injury (as a continuous measure) for subjects with two 270
diagnoses (see Fig. 1). This association was not approaching 271
significance in the no-diagnosis and one-diagnosis groups. 272

273 3.5. Psychiatric morbidity and self-reported cognitive, 274 emotional and somatic concerns

275 EI subjects with two psychiatric diagnoses as well as EI 276
subjects with one psychiatric diagnosis reported more 277
subjective cognitive, emotional and somatic concerns, as 278
compared to EI subjects with no psychiatric diagnosis (see 279
Table 4). In both the low- and high-voltage EI groups, the 280
most frequently endorsed somatic symptoms (i.e., symptoms 281
that were endorsed by more than a third of sample) were 282
blurred vision, difficulties with balance and acceleration 283
(e.g., trouble walking, tremors, dropping things), difficulties 284
with mechanoperception (e.g., numbness, “tingling” skin, 285
burning skin, loss of feeling) and symptoms of muscle 286
weakness, twitching and spasms.

287 4. Discussion

288 4.1. Prevalence and predictors of psychiatric morbidity

289 This study examined the prevalence of DSM-IV psychia-
290 tric disorders in a large sample of EI patients in all phases of
291 recovery (acute, post-acute and long-term). The results
292 demonstrate that psychiatric disorders are indeed common
293 following EI. Our findings showing an incidence of
294 psychiatric diagnosis in 78% of the sample are generally
295 consistent with those reported by Kelley et al. [4] and are
296 higher than those reported in TBI and burn populations [1,2].

297 Our findings also suggest that recovery phase from EI is a
298 significant predictor of psychiatric morbidity. Specifically,
299 EI subjects in the long-term phase of recovery were 14 times
300 more likely to be diagnosed with more than one psychiatric
301 diagnosis as compared to EI subjects in the acute and post-
302 acute phases of recovery. Thus, EI patients in our study who
303 are at a later phase in the recovery process are at a
304 significantly greater risk for developing psychiatric compli-
305 cations which is consistent with a prior study documenting
306 increased rate of emotional complaints in post-acute EI
307 patients as compared to acute EI patients [18]. As reported in
308 previous studies (e.g., Refs. [18,21]), there was no relation-
309 ship between EI outcome variable, in this study presence of
310 psychiatric disorder, and voltage level and litigation. More-
311 over, post-EI psychiatric morbidity was not associated with
312 previous history of psychiatric disorder, previous use of
313 psychotropic medication, and current chronic pain symp-
314 toms or usage of pain medications. Notably, the lack of
315 predictive relationship between previous use of psychiatric
316 medication and current presence of psychiatric disorder
317 suggests that psychiatric morbidity worsens overtime
318 regardless of whether or not there has been prior treatment
319 with psychiatric medication.

320 Considering symptoms profiles, there was an increase in
321 prevalence of combined PTSD and depression diagnoses

322 from the acute to long-term phases of recovery. The apparent
 323 progression in psychiatric difficulties among EI patients is of
 324 note given the literature demonstrating an inverse pattern
 325 among other clinical populations with traumatic injuries. For
 326 instance, in a cross-sequential study of 188 TBI patients,
 327 Ashman et al. [22] reported a decline in frequency of
 328 depression, substance abuse and PTSD over a period of
 329 3 years post-injury. Similarly, in a review of the literature,
 330 Patterson et al. [23] reported that anxiety and depression
 331 were the most prevalent disorders in burn patients, with
 332 prevalence rates between 25% and 65% during the first year
 333 after injury with most symptoms subsiding after that period.
 334 Thus, EI results in a distinct pattern of increased psychiatric
 335 morbidity over time as compared to other traumatic injuries
 336 for which there is a reported reduction in prevalence of
 337 psychiatric symptoms over the course of recovery.

338 The differences in the prevalence and progression of
 339 psychiatric morbidity following EI as compared to other
 340 traumatic injuries highlight the distinctiveness of this clinical
 341 population. These findings raise important questions as to
 342 whether EI has unique properties that render it more likely to
 343 trigger chronic and progressive psychiatric distress as
 344 compared to other types of traumatic injuries. In that regard,
 345 Mancusi-Ungaro et al. [7] suggested that the experience of
 346 electric shock is the prototype of one-trial fear conditioning
 347 and thus may be a potent inductor of PTSD symptomatology.
 348 Another possibility is that the increased rate of psychiatric
 349 complications observed over time reflects central nervous
 350 system dysfunction that may be attributed to the delayed
 351 onset or progressive effects of EI. Additionally, the use of
 352 electroconvulsive therapy (ECT) is a model of how exposure
 353 to an electrical field can alter brain functioning. ECT has
 354 long been successfully used for the treatment of major
 355 depression [24]. Although the exact mechanisms mediating
 356 the therapeutic effects of ECT on emotional functioning are
 357 currently unknown, several hypotheses have been offered
 358 including current-induced changes in membrane electro-
 359 permeabilization [25] and alteration of neurotransmitter
 360 systems, including serotonin, dopamine and GABA
 361 [26,27]. In that sense, EI might be considered as a case of
 362 ECT gone awry, in which accidental exposure to higher
 363 voltages causes a brain insult that results in chronic and
 364 progressive alterations in neural substrates involved in
 365 emotional functioning.

366 4.2. Psychiatric morbidity and cognitive functioning

367 Our findings further suggest that EI patients with two
 368 psychiatric diagnoses exhibited poorer cognitive perfor-
 369 mances on indices of verbal memory, executive functioning
 370 and attention relative to EI patients with a single psychiatric
 371 diagnosis. These findings are consistent with a previous
 372 study indicating decreased verbal memory among EI patients
 373 with PTSD as compared to EI patients without PTSD [12].
 374 The findings also demonstrated a trend for a correlation
 375 between performance on attention measures and time since

injury in the combined diagnosis group only. This indicates 376
 that EI patients with multiple psychiatric diagnoses show a 377
 trend for a progressive decline in attentional skills well into 378
 the long-term phase of recovery. 379

This pattern of increased rate of cognitive impairment 380
 among EI patients with multiple psychiatric diagnoses is not 381
 surprising given the extensive literature documenting the 382
 association between psychiatric morbidity and cognitive 383
 dysfunction in the general clinical population. Therefore, the 384
 observed relationship between increased psychiatric morbidity 385
 and poorer cognitive outcomes is not unique to the EI 386
 clinical population and may represent shared phenomenological 387
 aspects between EI and other clinical populations. 388

The current findings also demonstrate that EI patients 389
 diagnosed with a single or multiple psychiatric conditions 390
 report increased rate of cognitive, emotional and somatic 391
 concerns. This pattern of increased rate in self-reported 392
 cognitive complaints among the two diagnoses group is 393
 consistent with the study's findings indicating more 394
 pronounced impairment on objective measures of cognitive 395
 functioning in the two-diagnosis group as compared to the 396
 single and no-diagnosis groups. 397

The biologic mechanisms that underlie the increased rate 398
 of psychiatric morbidity observed in long-term EI patients 399
 are not yet well understood. While questions still remain as 400
 to how electrical exposure affects central nervous system 401
 function, the presence of poorer cognitive outcomes in the 402
 more psychiatrically disturbed EI subgroup could be 403
 suggestive of more widespread and persistent central 404
 nervous system dysfunction in neural systems that underlie 405
 cognitive as well as emotional functioning. 406

407 4.3. Clinical implications, limitations and future research

There are several implications to the current study. First, 408
 the findings establish that psychiatric changes are prevalent 409
 among EI patients and that this prevalence increases over 410
 time. These findings compel health care professionals 411
 working with EI patients at all phases of recovery to 412
 devote careful attention to evaluating the patients' psychi- 413
 atric status and to initiate appropriate interventions. Mental 414
 health professionals working with this clinical population 415
 should also be mindful of the possibility that EI patients 416
 presenting with comorbid psychiatric conditions and are 417
 further along into their recovery period may present with 418
 significant cognitive impairment encompassing several 419
 cognitive domains including memory, attention and execu- 420
 tive functioning. Thus, neuropsychological evaluation and 421
 cognitive rehabilitation, when appropriate, should be 422
 considered an important part of caring for EI patients at 423
 all phases of recovery. 424

The main limitation of the current study is that it used a 425
 convenience sample of EI patients pursuing a multidisci- 426
 plinary medical evaluation and treatment. As a result, it is 427
 possible that our sample was biased towards a higher rate of 428
 psychiatric distress compared to the EI population as a whole. 429

430 This limitation must be kept in mind when considering
431 the findings.

432 Future research should directly investigate the clinical
433 and phenomenological aspects of EI as compared to other
434 traumatic injuries. In addition, the effects of EI on brain
435 functioning, and in particular its effects on neural substrates
436 involved in emotion regulation, need to be thoroughly
437 examined through structural and functional neuroimaging
438 investigations.

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