Delayed-onset posttraumatic stress disorder: a prospective evaluation

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Objective: Delayed onset posttraumatic stress disorder (PTSD) refers to PTSD that develops at least 6 months after the traumatic event. This study aimed to index the features of patients who develop delayed-onset PTSD.
Method: This study investigated delayed onset PTSD by prospectively assessing 103 motor vehicle accident survivors within 1 month of the motor vehicle accident for acute stress disorder, and subsequently assessing them for PTSD 6 months post-accident, and 2 years post-accident. Patients were initially assessed for symptoms of traumatic stress, anxiety, depression, and resting heart rate.
Results: Five patients displayed PTSD 2 years post-trauma without meeting PTSD criteria 6 months posttrauma. Delayed onset cases were characterized by elevated psychopathology scores and resting heart rate levels within the initial month and elevated psychopathology 6 months posttrauma.
Conclusions: These findings suggest that cases of delayed onset PTSD suffer subsyndromal levels of posttraumatic stress prior to the diagnosis of PTSD. These findings challenge the notion of PTSD developing after a period without symptoms.
Key words: delayed onset, diagnosis, posttraumatic stress disorder.

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The DSM-IV identifies delayed onset posttraumatic stress disorder (PTSD) when the condition develops at least 6 months after the traumatic event. There are case studies of delayed onset PTSD developing as long as 30 years after the alleged precipitating event [1]. In terms of the frequency of this form of PTSD, delayed onset appears to be a rarely diagnosed condition. Large scale studies of civilian trauma have reported delayed onset PTSD in a very small minority of cases (range: 4.4%–6.2%, 2–4). The rarity of this condition has made empirical study of delayed onset PTSD difficult to conduct.

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Several mechanisms have been proposed to explain delayed onset PTSD. It has been argued that delayed onset PTSD may occur as a result of over-stimulation of endogenous stress-responsive hormones that may result in over-consolidation of trauma memories, which may form the basis of subsequent development of PTSD [5]. Alternately, it has been proposed that subsequent re-appraisals of the traumatic experience may heighten the perception of threat, and this different interpretation may contribute to PTSD development [6]. Finally, the culmination of stressful events that occur following the precipitating traumatic event may compound the initial stress reaction and cause PTSD development [7].

In terms of empirical findings, a number of studies have noted that individuals who are eventually diagnosed with delayed onset PTSD have reported subsyndromal levels of PTSD at initial assessments [2,8]. That is, these studies suggest that delayed onset PTSD cases fail to meet full criteria for PTSD initially but subsequently satisfy criteria because their condition

deteriorates to the point of meeting diagnostic threshold. Relatedly, evidence that many cases of delayed onset PTSD do suffer elevated levels of PTSD symptoms in the acute period suggests that many cases of so-called delayed onset PTSD may represent delayed identification rather than delayed onset of PTSD [9]. Other studies have found that people who develop delayed onset PTSD do experience more persistent stressors after the trauma, which may contribute to PTSD development [3,9,10]. There is also evidence that neuroticism and prior emotional difficulties are associated with people who develop delayed onset PTSD [3,11]. The current confusion over the mechanisms that mediate delayed onset PTSD and the scarcity of empirical studies of this disorder point to the need for further prospective investigation of delayed onset PTSD. One inherent limitation in the study of delayed onset of PTSD is that there are typically few patients who satisfy this profile, and accordingly analyses are limited by small sample sizes. This factor reinforces the need for further study of this atypical form of trauma response.

This study describes a prospective study of motor vehicle accident survivors who were assessed within 1 month of the motor vehicle accident, and subsequently assessed for PTSD at 6 months and 2 years post-accident. This prospective design permitted identification of patients who did not display PTSD 6 months post-accident but did have PTSD at the 2-year assessment (i.e. developed delayed onset PTSD). The goal of the study was to index the extent to which psychopathological indicators are evident in patients who eventually develop delayed onset PTSD. The initial data acquisition included assessment of (i) acute stress disorder (ASD) (ii) posttraumatic stress symptoms, anxiety, and depression, and (iii) resting heart rate levels. Heart rates were indexed because of evidence that people who eventually develop PTSD have higher resting heart rates in the initial week after a trauma than those who do not develop PTSD [12,13]. On the premise that delayed onset PTSD is characterized by elevated posttraumatic stress throughout the posttraumatic period, we hypothesized that delayed onset cases of PTSD would be characterized by higher levels of posttraumatic stress and higher levels of physiological arousal throughout their course of posttraumatic adjustment.

Method

Patients

One hundred and seventy-one motor vehicle accident survivors who were consecutive admissions to Westmead Hospital were assessed for acute stress disorder (ASD) within one month posttrauma, 134 were reassessed at 6 months posttrauma, and 103 were reassessed 2 years posttrauma. The participation of 103 individuals at the 2-year follow up represents a retention rate of 60% of the initial sample and 77% of the sample assessed at 6-months posttrauma. The sample comprising the initial, 6-month, and 2-year assessments are described in full elsewhere [14–17]. The final sample included 59 males and 44 females who ranged in age from 17 to 63 years (M = 30.96, SD = 12.18). Forty-nine patients were drivers, 21 passengers, 21 motorbike riders, 9 pedestrians, and 3 were cyclists. Those who participated in the 2-year posttrauma assessment did not differ from non-participants in terms of age, initial ASD diagnosis, length of hospital stay, injury severity score [18], or trauma-initial assessment interval.

All three assessments were carried out by clinical psychologists with more than 5 years experience in assessing traumatized individuals. Initial diagnosis of ASD was made using the acute stress disorder interview (ASDI 19). This structured clinical interview is based on DSM-IV criteria and possesses sound test-retest reliability (r = 0.95), sensitivity (91%), and specificity (93%). The 6-month and 2-year assessments involved the administration of the PTSD module from the composite international diagnostic interview (CIDI) which has acceptable internal consistency and concurrent validity for DSM-III-R [20]. In addition, at the initial and 6 month assessments patients were administered the impact of event scale [21], the Beck Depression Inventory [22], and the State trait anxiety inventory – State scale [23]. At the 6-month and 2-year assessments, patients were also administered an interview that indexed psychological treatment, the status of compensation proceedings, employment status, and chronic pain.

Treating physicians, who were unaware of patients' diagnostic status, indexed patients' heart rate using a Hewlett Packard M1205A omnicare monitor (model 24/24C) during hospitalization. Electrocardiogram leads were connected to the monitor, which digitally recorded heart rate. Heart rate was measured on the day of hospital discharge and was sampled continuously for 1 minute as the patient lay in their hospital bed and was subsequently recorded in the patient's medical record as beats per minute. To increase the reliability of indexing resting heart rate, two 1-minute periods were sampled and the average of these two measures was calculated to obtain mean resting heart rate values. These measures were obtained at times when the patients were resting and not immediately following or preceding any medical procedure.

Results

Diagnostic Results

There were 16 (15%) cases of ASD and 19 (19%) cases of subsyndromal ASD (defined as satisfying three of the four symptom clusters). In terms of incidence of PTSD, there were 23 (22%) cases at 6 months, and 25 (24%) cases at 2 years. There were only 5 female patients (5%) who met criteria for PTSD at 2 years but did not meet criteria at 6-months post-accident (delayed onset cases). Three patients had PTSD at 6 months but did not have PTSD at 2 years. Subsequent analyses focus on differences between patients who (i) had delayed onset PTSD (ii) had acute PTSD (defined as acute PTSD across the 6-month and 2-year assessments), and no PTSD (defined as no PTSD across the 6 month and 2 years assessments).

Acute Functioning

Table 1 presents the demographic characteristics and psychopathology scores of delayed onset PTSD, acute PTSD, and no PTSD patients. In terms of the initial assessment, one-way analyses of variance (ANOVA) indicate that those with acute PTSD had higher scores on each of the psychopathology measures than both delayed onset and no PTSD patients. Delayed onset patients also had higher IES-Intrusion scores than patients without PTSD. Delayed onset PTSD and acute PTSD patients had higher resting heart rates than no-PTSD patients. In terms of ASD diagnostic status, Table 2 demonstrates that more acute PTSD patients had at least subsyndromal ASD than no PTSD patients (χ^2 [1] = 18.40, $p < 0.001$).

Six Month Functioning

Oneway ANOVAs of psychopathology scores obtained at the 6 month assessment (see Table 1) indicate that those with acute PTSD had higher scores on each of the psychopathology measures than both delayed onset and no PTSD patients. Delayed onset patients had higher IES-Intrusion, BDI, and STAI-State scores than no PTSD patients. In terms of subsyndromal PTSD (defined as satisfying at least two symptom clusters of PTSD criteria) at 6 months, Table 2 demonstrates that more acute PTSD patients had at least subsyndromal PTSD than delayed onset PTSD patients (χ^2 [1] = 15.32, $p < 0.001$), who in turn had higher incidence than no PTSD patients (χ^2 [1] = 10.47, $p < 0.001$).

Chi-square analyses indicated that more acute PTSD patients received therapy than no PTSD (χ^2 [1] = 15.37, $p < 0.001$) and delayed onset PTSD (χ^2 [1] = 10.99, $p < 0.001$) patients. More acute PTSD (χ^2 [1] = 11.42, $p < 0.001$) and delayed onset PTSD (χ^2 [1] = 3.89, $p < 0.05$) patients reported chronic pain than no PTSD patients.

Two-year Functioning

Chi-squared analysis indicated that two years after the accident more acute PTSD patients reported ongoing compensation than patients without PTSD (χ^2 [1] = 7.07, $p < 0.05$). Delayed onset PTSD patients were less likely to have received treatment than acute PTSD patients (χ^2 [1] = 4.24, $p < 0.05$).

Discussion

The finding that only 5% of cases developed delayed onset PTSD reinforces previous reports of the scarcity of this diagnosis [2–4]. The pattern of these individuals was to immediately respond to the accident with elevated levels of posttraumatic stress that did not satisfy diagnostic criteria. Although four of the five patients displayed no ASD, they displayed higher level of re-experiencing symptoms in the initial month than patients without PTSD. The level of psychopathology associated with the delayed onset patients became more marked at the 6-month assessment when they displayed more re-experiencing, state anxiety, and depression than no-PTSD patients. This development is reflected in the finding that two of the five delayed onset PTSD patients had subsyndromal PTSD by 6 months. These findings converge with earlier reports that so-called delayed onset PTSD is predominantly describing individuals who do suffer acute posttraumatic stress but do not display or report sufficient symptoms to meet diagnostic criteria [2,9].

Table 1. Patient characteristics

Variable	Delayed-onset PTSD (n = 5)	No PTSD (n = 75)	Acute PTSD	*F* (2, 101) (n = 23)	p
Demographics					
Age	29.40 (12.93)	30.68 (11.93)	31.86 (12.81)	0.12	ns
Trauma-assessment interval	3.80 (3.49)	7.24 (5.52)	7.82 (6.30)	1.06	ns
Injury severity score	3.40 (2.30)	7.36 (6.32)	6.13 (5.19)	1.29	ns
Acute functioning					
ASDI total	5.20 (4.32)[a]	3.16 (2.75)[a]	11.09 (4.77)[b]	48.66	0.001
BDI	9.00 (10.42)[a]	7.27 (7.71)[a]	22.20 (7.04)[b]	21.39	0.001
IES intrusions	18.80 (3.89)[a]	8.62 (10.26)[b]	25.27 (9.57)[c]	16.00	0.001
IES avoidance	12.60 (2.88)[a]	8.28 (10.26)[a]	31.25 (15.19)[b]	12.01	0.001
STAI-state	41.20 (10.67)[a]	38.92 (14.35)[a]	56.87 (10.80)[b]	9.45	0.001
Heart rate	83.00 (7.58)[a]	74.31 (8.74)[b]	82.18 (14.60)[a]	5.13	0.01
6-month functioning					
BDI	12.25 (11.93)[a]	4.31 (5.31)[b]	16.35 (12.08)[c]	14.44	0.001
IES intrusions	13.50 (8.34)[a]	4.81 (5.81)[b]	22.28 (9.57)[c]	35.76	0.001
IES avoidance	14.25 (4.64)[a]	6.06 (6.41)[a]	31.25 (15.19)[b]	34.28	0.001
STAI-state	47.40 (10.08)[a]	31.66 (12.36)[b]	53.79 (12.84)[c]	18.74	0.001

Note. Standard deviations appear in parentheses. Trauma-assessment interval measured in days. ASDI = acute stress disorder interview. IES = impact of event scale. BDI = Beck depression inventory. STAI = state trait anxiety inventory. Superscripts of different letters indicate significant group differences.

Table 2. Categorical results

Variable	Delayed-onset PTSD (n = 5)	No PTSD (n = 75)	Acute PTSD (n = 23)	χ^2 (2)	p
Demographics					
Sex (male/female)	0/100	71/29	26/74	21.34	0.001
Psychiatric history	20	13	39	7.49	0.05
Acute functioning					
ASD diagnosis	20	7	43	18.21	0.001
6-month functioning					
Subsyndromal/full PTSD	40	4	100	83.87	0.001
Compensation pending	76	51	69	2.15	ns
Employed	100	89	92	0.55	ns
Received treatment	0	32	77	11.40	0.005
Chronic pain	100	56	100	15.00	0.001
2-Year Functioning					
Compensation Pending	80	38	70	18.87	0.001
Received treatment	20	30	70	37.88	0.001

Note. All values reported as percentages.

The finding that delayed onset PTSD and acute PTSD patients had higher resting heart rate levels than patients without PTSD is consistent with evidence that sympathetic activation in the initial week after a trauma is associated with subsequent PTSD [12,13]. This observed pattern suggests that delayed onset PTSD patients responded in a physiologically similar manner to acute PTSD patients, and supports the notion that delayed onset PTSD involves similar mechanisms as those mediating acute PTSD. Shalev argues that elevated heart rate levels in the acute phase reflect strong fear conditioning that leads to subsequent hyperarousal and other PTSD disturbances [13]. It is possible that delayed onset PTSD is mediated, in part, by elevated sympathetic activation, and that this may be a useful means to assist identification of acutely traumatized people who are at risk of presenting with delayed onset PTSD. This finding suggests that future prospective studies should index other biological markers of PTSD, such as reduced cortisol levels [24], to determine other psychophysiological patterns of people who develop delayed onset PTSD.

The finding that delayed onset PTSD cases were differentiated from no PTSD cases by higher levels of pain at 6 months is consistent with evidence that delayed onset PTSD is associated with ongoing stressors that occur during the posttrauma period [3,9–11]. It is possible that initial posttraumatic stress reactions were subsequently compounded by additional stressors experienced by delayed onset PTSD patients.

Interestingly, no delayed onset PTSD patients had received treatment at 6 months and only one had received treatment at 2 years posttrauma. The lack of psychological assistance during the posttrauma period may have contributed to these patients deteriorating over time. The finding that 32% of no PTSD patients received treatment compared to no delayed onset PTSD patients at 6 months posttrauma raises the possibility (that cannot be answered with the current data) that psychological intervention may have assisted some of the no PTSD patients. Alternately, it is possible that delayed onset PTSD patients may be reluctant to report symptoms and this avoidance may reduce the likelihood of them seeking assistance [25]. This interpretation is not supported by the relatively low IES-avoidance scores reported by delayed onset PTSD patients.

Compensation following motor vehicle accidents may influence reporting of symptoms. Whereas there were no differences between groups in terms of litigation status 6 months posttrauma, fewer no PTSD patients were involved in litigation at 2 years than delayed onset and acute PTSD patients. This pattern appears to reflect a reduction in litigation involvement in no PTSD cases compared to the maintenance of litigation in cases with persistent PTSD. The greater involvement in litigation may be explained by factors associated with the documented higher rates of chronic pain and psychological distress observed in the acute and delayed onset PTSD patients. Alternately, it is possible that pending litigation contributed to enhanced symptom reporting or increased stress that contributed to ongoing PTSD [26].

We recognize that inferences from this study are very limited by the small number of delayed onset PTSD cases. This limitation appears to be inherent in the study of delayed onset PTSD because of the scarcity of these

cases. We emphasize that inferences based on only five cases of delayed onset PTSD need to be made very cautiously because of the small sample size. This scarcity of delayed onset PTSD underscores the need for replication of prospective studies to establish robust findings across the small samples of delayed onset PTSD. Generalizing these findings to other trauma populations is also limited because of the current focus on motor vehicle accident survivors. These limitations notwithstanding, the data supports propositions that delayed onset PTSD may be a misnomer because the onset of the posttraumatic stress appears to be immediately after the traumatic event. Future work that prospectively studies delayed onset cases of PTSD, and especially addresses the issue of help-seeking in this population, may enhance the management of this minority response to traumatic experience.

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