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Catastrophizing as a Key Factor for Understanding Posttraumatic Trigeminal Neuropathy: A Preliminary Study

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Purpose: Limited literature exists regarding psychological relevance to pain experience and disability in patients with painful posttraumatic trigeminal neuropathy (PPTTN), which is well-known for its treatment-refractory features and considerable impact on the quality of life. Thus, this study aimed to examine the biopsychosocial risk factors for pain disability in patients with PTTN.

Methods: A comprehensive set of self-administered questionnaires was used to assess biopsychosocial features in patients with PPTTN. The questionnaires comprised the Brief Pain Inventory (sensory dimension), Symptom Checklist-90-Revised (affective dimension), Pain Catastrophizing Scale (PCS, cognitive dimension), and Pittsburgh Sleep Quality Index (sleep quality). Sensory clinical examinations were also conducted.

Results: Data were obtained from 32 patients with PPTTN who had a median pain duration of 16 months. Injuries to the inferior alveolar nerve and lingual nerve accounted for 71.9% and 28.1% of all injuries, respectively. Most patients showed high levels of pain catastrophizing (71.9%) and poor sleep quality (87.5%). Unlike affective distress and sleep quality, the mean scores of the three subscales and the global scores of PCS were significantly higher in patients with high pain interference than those with low pain interference. Pain severity and the PCS "helplessness" subscale were significant risk factors for pain interference in patients. Significance was observed for the final model with two predictors, explaining 86.5% of the pain interference variance. Additional analyses revealed that the PCS scores were not correlated with sensory features of PPTTN. However, they were associated with affective distress and subjective sleep quality.

Conclusions: The study findings indicate that the key role of pain-specific helplessness as a determinant of pain disability associated with PPTTN may provide insight into an enhanced understanding and management of pain disability in patients with PPTTN.

Keywords: Catastrophization; Psychology; Trigeminal nerve injuries

INTRODUCTION

Dental practitioners seek to uphold "do good, do no harm" as a core component of the Hippocratic Oath [1]. However, completely avoiding harm seems impossible due to the inherent characteristics of invasive dental surgery and the prominent sensory sensitivity of the orofacial region, which is innervated by the trigeminal nerve. One well-known but unwanted sequelae of invasive dental treatments is trigeminal nerve injury, which can result in chronic pain and/or altered sensation in the orofacial region [2]. Depending on the differences in the definition and criteria of signs and symptoms, the chronic pain condition following traumatic trigeminal injury is also referred

to as posttraumatic trigeminal neuropathic pain (PTTN) [3], painful posttraumatic trigeminal neuropathy (PPTTN) (third edition of the International Classification of Headache Disorders [ICHD-3]), chronic postsurgical pain [4], and persistent dentoalveolar pain [5]. In this study, PPTTN refers to a painful (dysesthesia) or nonpainful condition (numbness and paresthesia) that develops after iatrogenic trigeminal nerve damage. Because the inferior alveolar nerve (IAN) and lingual nerve (LN) are the two branches of the trigeminal nerve typically affected by a third molar extraction, implant placement, local anesthesia injection, and endodontic treatment [6], PTTN or PPTTN has been acknowledged to substantially impact a patient's oral physiologic functioning, psychosocial functioning, and overall quality of life [7-9] According to a study by Cruyssen that analyzed 1331 PTTN cases [10], most patients with PTTN reported difficulty in activities of daily living, such as eating, drinking, talking, sleeping, as well as kissing and facial expressions. Emotional stress, such as depression and anxiety, was also reported to increase pain intensity [10]. Furthermore, increasing interest in preclinical and clinical studies on biological pathophysiology and sensory profiling using electrophysiological and psychophysical tools has been observed [11,12]. However, the psychological relevance assessment of PPTTN has received little attention. Given the significant relationship between chronic neuropathic pain and various Axis II components, including emotion, sleep, and cognition [13-15], a comprehensive evaluation of the relevance of various psychological aspects to pain outcomes related to PPTTN is necessary. Interestingly, among various psychological factors, several clinical studies have demonstrated that pain catastrophizing, a pain-specific psychological factor, is one of the strongest predictors of chronic pain [15-19]. Pain catastrophizing refers to a dysfunctional and negative mentality when coping with actual or anticipated pain. It comprises three distinct but related subscales: rumination ("I always worry about whether it will end"), magnification ("It is awful, and I fear that it will never get better"), and helplessness ("I feel like I can't go on") [20,21]. The relative contributions of these three separate subscales to the pain outcomes associated with PTTN are limited. Therefore, to better characterize the psychological consequences of PPTTN, this study aimed to examine the psychological risk factors—including general psychological factors, such as emotion and sleep, and pain-specific psychological factors, such as pain catastrophizing—for pain experience and disability associated with PPTTN. Particularly, the study focused on the association of the three subscales of pain catastrophizing with pain severity and interference related to PPTTN. This study hypothesizes that general and pain-specific psychological factors are significantly associated with pain severity and interference in patients with PPTTN.

MATERIALS AND METHODS

This clinical study was conducted in accordance with the 1964 Helsinki Declaration on medical ethics and protocol. Ethical approval was provided by the Institutional Review Board (IRB) of Dankook University Dental Hospital (IRB no. DKUDH IRB 2019-09-001), and informed consent was obtained from all patients.

1. Study Participants

This cross-sectional study assessed the clinical and psychosocial characteristics of patients who sought consult at the Orofacial Pain Clinic of Dankook Dental Hospital between 2020 and 2021 following an iatrogenic trigeminal nerve injury. A total of 38 patients who complained of sensory changes, including numbness and/or pain, after invasive dental treatments affecting IAN or LN were consulted and screened for PTTN by an orofacial pain specialist (KHK).

Based on the medical interviews and results of the clinical sensory tests, PPTTN diagnosis was established in accordance with ICHD-3 [22]. According to the ICHD-3 criteria, PPTTN diagnosis requires a history of unilateral or bilateral orofacial pain caused by an identifiable trauma to the trigeminal nerve(s), as well as clinically evident positive (hyperalgesia and allodynia) and/or negative (hypoesthesia and hypoalgesia) signs localized to the affected nerve-innervated area(s). As a temporal requirement, pain should manifest no later than six months following the trauma. To be eligible for inclusion, individuals should have a history of traumatic nerve injury following invasive dental treatment, with subsequent onset of altered sensation, including hypoesthesia and/or hyperesthesia (e.g., allodynia and hyperalgesia) and/or paresthesia, mainly limited to the innervation

of the injured nerve. The included participants were at least 18 years old and had no history of systemic or neurological conditions that increased the likelihood of peripheral neuropathy. Patients were excluded from the study if they had concurrent orofacial pain conditions other than PPTTN, including painful temporomandibular disorders and burning mouth syndrome.

2. Clinical Examination

The patients' medical records were retrospectively evaluated for their demographic profiles, medical history, subjective symptoms, and objective signs. Patients were asked to rate the intensity of their numbness using a numeric rating scale (NRS), with the endpoint of 0 suggesting no sensory deficits and 10 denoting the worst hypoesthesia imaginable. Additionally, a qualitative evaluation was conducted for the positive symptoms. Based on the NRS ranging from 0 (no pain) to 10 (the worst pain imaginable), the positive symptoms included superficial pain (e.g., burning sensation); deep pain (e.g., pressing or tightness); paroxysmal pain (e.g., shooting or electric); evoked pain upon touch, pressure, or exposure to cold; and paresthesia (e.g., tingling or pin and needle sensation). Clinical sensory testing, including brush stroke, pin-prick, and thermal discriminating tests, was performed in all patients to localize the area with abnormal sensations and assess neuropathy. In the case of IAN injuries, cone-beam computed tomography images were taken to evaluate nerve damage. The unaffected side contralateral to the affected side was used as a control. An experienced specialist (HKK) interviewed and examined all the patients.

3. Self-reported Questionnaires

In the current study, four self-reported questionnaires designed to evaluate the patients' psychosocial aspects were utilized.

4. Brief Pain Inventory (BPI)

The BPI, a short and simple questionnaire, is among the most extensively used evaluation tools for assessing clinical pain [23]. Pain severity and its impact on function, called pain interference, are evaluated based on rating scales ranging from 0 (denoting no discomfort) to 10 (representing the worst discomfort imaginable). The "worst," "least," "average," and "now" pain components comprise the measure of pain intensity of the BPI. The pain interference scale of the BPI comprises seven questions and measures how much pain has impacted sleep, walking, job performance, mood, enjoyment of life, and relationships with others. In the Korean version of the BPI, the item measuring "walking ability" was replaced by "chewing ability" in relation to orofacial pain [24]. The responses were based on the week before the BPI was completed.

5. Pain Catastrophizing Scale (PCS)

PCS, created by Sullivan et al. [20], evaluates how frequently participants encounter thoughts and feelings related to their pain. PCS comprises 13 items on a 5-point scale, with 0 meaning "never" and 4 meaning "always." The total score and three subscale scores for rumination (focusing on pain-related thoughts), magnification (a tendency to exaggerate the extent of pain), and helplessness (a reactive appraisal of pain) were obtained using the PCS.

6. Symptom Checklist-90-Revised (SCL-90R)

The respondent's degree of psychological health was assessed using 90 items from the SCL-90R [25]. On a 5-point Likert-type scale (0=not at all; 4=very), responders were asked to rate how much each of the 90 items in the survey annoyed them in the preceding seven days. From the 90 items, the global symptom index (GSI) and nine symptom dimensions, including somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism, were evaluated.

7. Pittsburgh Sleep Quality Index (PSQI)

Various sleep quality factors were assessed using the PSQI, which comprises a set of 19 self-reported questions [26]. Seven component scores were extracted from the 19 items, with each value ranging from 0 to 3. The total PSQI score, ranging from 0 to 21, was derived from the sum of these seven component scores. Increased scores denote reduced sleep quality.

8. Statistical Analysis

The Kolmogorov–Smirnov test was used to evaluate data normality. Mean and standard deviation (SD) were used for descriptive statistics of continuous data with a normal distribution, whereas median and interquartile range (IQR) were used for data that were not normally distributed. Moreover, numbers and proportions were used for categorical values. Patients were categorized into two groups based on clinical features (e.g., sex, etiology for injury, and injured nerve) and sensory characteristics (numbness, pain severity, and pain interference), using the median value as the cutoff for continuous and nondichotomous data. Independent t-tests and analyses of variance were conducted to compare the biopsychosocial features between the groups. The relationships between variables were assessed using Pearson's correlation analysis. Multiple regression analysis with a stepwise approach was conducted to explore predictors of pain interference. All data were log-transformed for statistical analyses. Data analyses were conducted using IBM SPSS Statistics for Windows, Version 21.0 (IBM Co.). Statistical

Table 1. Sample characteristics (n=32)

Variable	Descriptions	Statistics
Demographics		
Sex (Female:male)	19 (59.4):13 (40.6)	
Age (y)	45.8 (12.37)	
Duration (mo)	16.0 (9.25-27.75)	
Clinical features		
Etiology (extraction:installation)	16 (50.0):16 (50.0)	
Injured nerve (IAN:LN)	23 (71.9):9 (28.1)	
Sensory features		
NRS		
Numbness	5 (3.62-7.00)	
BPI		
Pain severity	4.9 (2.30)	
Pain interference	5.1 (2.75)	
Cognitive features		
PCS		F=5.267 (p=0.007)
Magnification	5.5 (3.77)	
Rumination	9.0 (1.00-13.75)	
Helplessness	12.0 (7.25-15.50)	
Global score	25.5 (10.75-37.75)	
Affective features		
SCL-90R		F=1.428 (p=0.184)
Somatization	43.0 (39.00-48.75)	
Obsessive-compulsive	39.0 (32.25-43.00)	
Interpersonal sensitivity	38.5 (36.00-45.00)	
Depression	40.0 (36.00-44.75)	
Anxiety	41.5 (39.00-43.75)	
Hostility	40.0 (38.50-43.00)	
Phobic anxiety	43.0 (40.00-45.00)	
Paranoid	38.0 (38.00-41.50)	
Psychoticism	40.0 (38.00-44.50)	
GSI	39.0 (32.25-42.75)	
Subjective sleep quality		
PSQI		
Global score	11.0 (4.38)	

IAN, inferior alveolar nerve injury; LN, lingual nerve injury; NRS, numeric rating scale; BPI, Brief Pain Inventory; PCS, Pain Catastrophizing Scale; SCL-90R, Symptom Check List-90-Revised; GSI, global symptom index; PSQI, Pittsburgh Sleep Quality Index.
Values are presented as number (%), mean (standard deviation), or median (interquartile range).
For non-normal distributed data, the median and interquartile range were utilized, and log-transformation was performed for further statistical analyses. Data including PCS and SCL-90R were log-transformed for one-way analysis of variance. The Tukey post-hoc analysis result for the three subscales of PCS was as follows: helplessness>magnification and rumination.

significance was set at p=0.05.

RESULTS

1. Demographics and Clinical Features of Included Patients

Among the 38 patients screened for PTTN, 32 were included in the study. Four patients with persistent dentoalveolar pain, as well as two patients with PPTTN and painful osteoarthritis of the temporomandibular joints, were excluded from the study. The demographic and clinical features are presented in Table 1. The mean age (SD) of the included patients was 45.8 (12.37) years. The median duration of neuropathy (IQR) was 16.0 (9.25–27.75) months. The proportion of females (59.4%) was greater than that of males (40.6%). Trigeminal nerve injuries were attributed to implant installation and extraction, and no significant difference in the incidence was found for each (50.0% vs. 50.0%). Most injuries (71.9%) occurred in the IAN, with 28.1% of the patients having LN injuries.

The symptomatic sites in patients with IAN damage included the unilateral mentum, lower lip, and/or ipsilateral anterior mandibular teeth. The symptom site in cases of LN

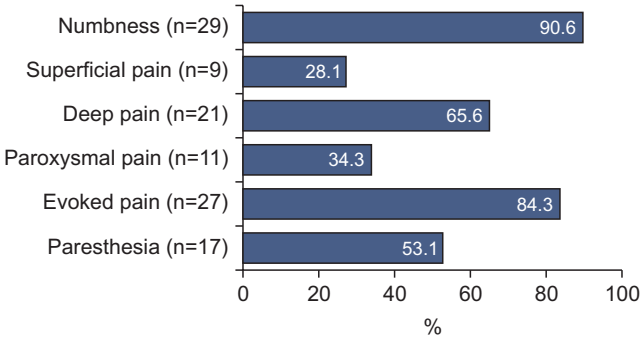

Fig. 1. Frequency and proportion of altered sensation (n=32).

Table 2. Comparison of biopsychosocial features based on sex, etiology, and injured nerve(s)

Variable	Sex			Etiology			Injured nerve		
	Female (n=19)	Male (n=13)	p-value	Extraction (n=16)	Implant (n=16)	p-value	IAN (n=23)	LN (n=9)	p-value
NRS									
Numbness	1.3 (0.71)	1.6 (0.23)	0.356	1.6 (0.38)	1.3 (0.71)	0.215	1.3 (0.62)	1.8 (0.31)	0.215
BPI									
Pain severity	1.5 (0.54)	1.1 (0.92)	0.176	1.3 (0.61)	1.4 (0.85)	0.688	1.4 (0.76)	1.3 (0.67)	0.688
Pain interference	1.5 (0.76)	1.2 (0.89)	0.257	1.3 (0.84)	1.4 (0.81)	0.647	1.4 (0.75)	1.1 (0.98)	0.647
PCS									
Magnification	1.4 (0.81)	1.4 (1.01)	0.938	1.4 (0.87)	1.4 (0.92)	0.953	1.4 (0.87)	1.1 (0.91)	0.953
Rumination	1.7 (1.13)	1.5 (1.14)	0.685	1.7 (1.08)	1.6 (1.19)	0.764	1.7 (1.10)	1.5 (1.24)	0.764
Helplessness	2.1 (0.92)	2.2 (0.75)	0.846	2.1 (0.92)	2.2 (0.79)	0.833	2.2 (0.70)	1.9 (1.16)	0.833
Global score	2.9 (0.93)	2.8 (1.01)	0.886	2.9 (0.90)	2.8 (1.03)	0.872	2.9 (0.90)	2.7 (1.11)	0.872
SCL-90R									
Somatization	3.8 (0.17)	3.7 (0.13)	0.230	3.7 (0.16)	3.7 (0.16)	0.662	3.8 (0.15)	3.7 (0.17)	0.662
Obs-com	3.6 (0.22)	3.6 (0.19)	0.844	3.6 (0.21)	3.6 (0.21)	0.645	3.6 (0.19)	3.7 (0.26)	0.645
Int-sen	3.7 (0.23)	3.6 (0.16)	0.731	3.7 (0.21)	3.7 (0.21)	0.853	3.6 (0.18)	3.7 (0.26)	0.853
Depression	3.7 (0.23)	3.7 (0.17)	0.843	3.7 (0.21)	3.7 (0.21)	0.771	3.7 (0.18)	3.7 (0.28)	0.771
Anxiety	3.7 (0.16)	3.7 (0.14)	0.699	3.7 (0.16)	3.7 (0.16)	0.888	3.7 (0.14)	3.7 (0.20)	0.808
Hostility	3.7 (0.21)	3.7 (0.11)	0.803	3.7 (0.20)	3.7 (0.15)	0.904	3.7 (0.13)	3.7 (0.27)	0.904
Pho-anx	3.7 (0.12)	3.7 (0.10)	0.471	3.7 (0.06)	3.8 (0.14)	0.213	3.7 (0.12)	3.7 (0.07)	0.213
Paranoid	3.7 (0.16)	3.6 (0.11)	0.748	3.6 (0.16)	3.7 (0.12)	0.535	3.7 (0.11)	3.7 (0.21)	0.535
Psychoticism	3.7 (0.18)	3.7 (0.13)	0.699	3.7 (0.17)	3.7 (0.16)	0.766	3.7 (0.14)	3.7 (0.22)	0.766
GSI	3.6 (0.22)	3.6 (0.16)	0.963	3.6 (0.20)	3.7 (0.20)	0.796	3.6 (0.17)	3.7 (0.27)	0.796
PSQI									
Global score	2.3 (0.45)	2.2 (0.60)	0.758	2.3 (0.47)	2.2 (0.55)	0.491	2.2 (0.49)	2.4 (0.54)	0.491

NRS, numeric rating scale; BPI, Brief Pain Inventory; PCS, Pain Catastrophizing Scale; SCL-90R, Symptom Check List-90-Revised; Obs-com, obsessive-compulsive; Int-sen, interpersonal sensitivity; Pho-anx, phobic anxiety; GSI, global symptom index; PSQI, Pittsburgh Sleep Quality Index; IAN, inferior alveolar nerve injury; LN, lingual nerve injury.
Values are presented as mean (standard deviation).
All data were log-transformed for independent t-test.

injury was the anterior two-thirds of the tongue on the injured side. All medical questions, for both negative and positive symptoms, focused on the site that caused the maximum discomfort. The median value (IQR) of numbness was 5.0 (3.62–7.00), and the mean (SD) values for pain severity and interference were 4.9 (2.30) and 5.1 (2.75), respectively. Fig. 1 shows the frequency and proportion of altered sensations reported by patients with PPTTN. Various sensory abnormalities were noted by the patients. Approximately 90.6% of patients reported numbness. For positive symptoms, the most frequent complaint was evoked pain (84.3%), followed by deep pain (65.6%), paresthesia (53.1%), paroxysmal pain (34.3%), and superficial pain (9%).

2. Exploration of Biopsychosocial Features of the Included Patients

The biopsychosocial features of the patients are presented in Table 1. The descriptive values of PCS include the three subscales and the global PCS score. The median (IQR) value of the global PCS score was 25.5 (10.75–37.75). The proportion of patients classified as high catastrophizers, according to Akhter's [27] categorization (a global PCS score≥15), was 71.9% (n=23), whereas 28.1% of patients were classified as low catastrophizers (a global PCS score<15). The mean value (SD) of magnification was 5.5 (3.77), and the median values (IQR) of rumination and helplessness were 9.0 (1.00–13.75) and 12.0 (7.25–15.50), respectively. Comparing the three PCS subscales with each other revealed significant differences, with helplessness showing the maximum score (p=0.007). Conversely, the nine SCL-90R subscales did not significantly differ from one another (p=0.184). The median GSI score (SD) was 39.0 (32.25–42.75). The number (ratio) of patients with GSI scores of 40 or more, 50 or more, and 60 or more were 13 (40.6%), 4 (10.5%), and 2 (6.2%), respectively.

The mean global PSQI score (SD) of all the patients was 11.0 (4.38), and approximately 87.5% of the patients had poor sleep quality (global score>5).

Table 2 shows the comparison of the sensory, cognitive, affective, and subjective sleep quality features based on sex, etiologies (implant extraction vs. installation), and injured nerves (IAN vs. LN) (Table 2). None of the descriptive values for numbness, BPI, PCS, SCL-90R, or PSQI differed based on sex, etiology, or injured nerves. Similarly, none of the descriptive values for numbness, BPI, PCS, SCL-90R, or PSQI exhibited a significant association with age (Table 3). However, symptom duration showed a significant correlation with numbness (Table 3, r=–0.535, p=0.002), indicating that numbness weakened over time. However, pain severity, related pain interference, and psychosocial factors did not differ based on symptom duration (Table 3, all p>0.05).

Table 4 shows the comparison of the sensory, cognitive, affective, and subjective sleep quality features based on numbness severity, pain severity, and pain interference. The numbness severity did not impact pain severity, pain interference, SCL-90R scores, or PSQI scores. The mean global

Table 3. Correlation of sensory, cognitive, affective, and sleep features with age and symptom duration

Variable	Age	Symptom duration
	r (p-value)	
Sensory features		
NRS		
Numbness	−0.067 (0.715)	−0.535 (0.002*)
BPI		
Pain severity	0.056 (0.759)	0.175 (0.338)
Pain interference	0.127 (0.488)	0.194 (0.288)
Cognitive features		
PCS		
Magnification	0.103 (0.575)	0.206 (0.258)
Rumination	−0.145 (0.429)	0.099 (0.591)
Helplessness	0.018 (0.924)	0.218 (0.231)
Total score	−0.051 (0.782)	0.197 (0.279)
Affective features		
SCL-90R		
Somatization	−0.192 (0.292)	0.105 (0.568)
Obsessive-compulsive	−0.111 (0.546)	−0.164 (0.370)
Interpersonal sensitivity	−0.116 (0.527)	−0.058 (0.752)
Depression	−0.120 (0.514)	−0.100 (0.586)
Anxiety	−0.097 (0.596)	−0.045 (0.805)
Hostility	−0.199 (0.275)	−0.139 (0.446)
Phobic anxiety	0.083 (0.652)	0.168 (0.359)
Paranoid	−0.132 (0.471)	−0.165 (0.368)
Psychoticism	−0.021 (0.909)	0.004 (0.983)
GSI	−0.095 (0.604)	−0.031 (0.867)
Sleep feature		
PSQI		
Global score	−0.281 (0.119)	−0.163 (0.374)

NRS, numeric rating scale; BPI, Brief Pain Inventory; PCS, Pain Catastrophizing Scale; SCL-90R, Symptom Check List-90-Revised; GSI, global symptom index; PSQI, Pittsburgh Sleep Quality Index. All data were log-transformed for Pearson's correlation analysis. r indicates correlation coefficient. *p<0.01.

Table 4. Comparison of cognitive, affective, and sleep features based on sensory features

Variable	Numbness			Pain severity			Pain interference		
	Low (n=20)	High (n=12)	p-value	Low (n=20)	High (n=12)	p-value	Low (n=17)	High (n=15)	p-value
Numbness	1.2 (0.57)	1.9 (0.14)	<0.001**	1.5 (0.59)	1.4 (0.59)	0.822	1.4 (0.71)	1.5 (0.38)	0.448
BPI_PS	1.5 (0.50)	1.2 (1.02)	0.390	1.0 (0.75)	1.9 (0.16)	0.001**	1.0 (0.79)	1.8 (0.21)	<0.001**
BPI-PI	1.4 (0.59)	1.2 (1.11)	0.414	1.0 (0.83)	2.0 (0.20)	<0.001**	0.8 (0.78)	2.0 (0.15)	<0.001**
PCS_M	1.5 (0.82)	1.2 (0.99)	0.417	1.1 (0.93)	1.8 (0.63)	0.035*	0.9 (0.88)	1.9 (0.53)	0.001**
PCS_R	1.8 (0.91)	1.3 (1.39)	0.211	1.4 (1.17)	1.9 (1.01)	0.225	1.2 (1.14)	2.1 (0.93)	0.026*
PCS_H	2.3 (0.45)	1.8 (1.20)	0.069	1.9 (0.95)	2.5 (0.51)	0.079	1.7 (0.93)	2.6 (0.45)	0.004**
PCS_G	3.1 (0.50)	2.4 (1.35)	0.058	2.6 (1.06)	3.2 (0.61)	0.079	2.4 (1.04)	3.4 (0.54)	0.004**
Somatization	3.7 (0.16)	3.7 (0.16)	0.997	3.7 (0.15)	3.8 (0.17)	0.319	3.7 (0.13)	3.8 (0.18)	0.659
Obs-com	3.6 (0.17)	3.7 (0.25)	0.334	3.6 (0.15)	3.7 (0.26)	0.138	3.6 (0.16)	3.7 (0.24)	0.121
Int-sen	3.6 (0.18)	3.7 (0.25)	0.611	3.6 (0.15)	3.7 (0.27)	0.223	3.6 (0.16)	3.7 (0.25)	0.541
Depression	3.7 (0.18)	3.7 (0.25)	0.592	3.6 (0.14)	3.7 (0.28)	0.137	3.6 (0.14)	3.7 (0.26)	0.208
Anxiety	3.7 (0.13)	3.7 (0.19)	0.484	3.7 (0.10)	3.8 (0.20)	0.081	3.7 (0.10)	3.8 (0.19)	0.071
Hostility	3.7 (0.12)	3.8 (0.24)	0.359	3.7 (0.09)	3.8 (0.26)	0.163	3.7 (0.10)	3.8 (0.23)	0.231
Pho-anx	3.7 (0.11)	3.7 (0.11)	0.850	3.7 (0.10)	3.7 (0.13)	0.848	3.7 (0.11)	3.7 (0.11)	0.711
Paranoid	3.6 (0.10)	3.7 (0.20)	0.459	3.6 (0.07)	3.7 (0.20)	0.071	3.6 (0.08)	3.7 (0.18)	0.178
Psychoticism	3.7 (0.13)	3.7 (0.20)	0.784	3.7 (0.10)	3.8 (0.22)	0.074	3.7 (0.10)	3.7 (0.20)	0.202
GSI	3.6 (0.17)	3.7 (0.25)	0.654	3.6 (0.14)	3.7 (0.26)	0.123	3.6 (0.14)	3.7 (0.24)	0.258
PSQI_G	2.3 (0.46)	2.3 (0.59)	0.993	2.3 (0.46)	2.2 (0.59)	0.740	2.3 (0.45)	2.2 (0.58)	0.903

BPI, Brief Pain Inventory; PS, pain severity; PI, pain interference; PCS, Pain Catastrophizing Scale; M, magnification; R, rumination; H, helplessness; G, global score; Obs-com obsessive-compulsive; Int-sen, interpersonal sensitivity; Pho-anx, phobic anxiety; GSI, global symptom index; PSQI, Pittsburgh Sleep Quality Index.
Values are presented as mean (standard deviation). Cutoff values of numbness, pain severity, and pain interference were 5 (median value), 4.9 (median value), and 5.1 (median value), respectively.
All data were log-transformed for independent t-tests. *p<0.05, **p<0.01.

score and helplessness subscale score of PCS were higher in the reduced numbness group than in the increased numbness group; however, this difference was not statistically significant (p=0.058 for the global score; p=0.069 for the helplessness subscale). The group with increased pain severity (p=0.035) and pain interference (p=0.001) had greater magnification scores than the group with reduced pain severity and interference. The group with superior pain severity had more helplessness-related thoughts than those with reduced pain severity but with no statistically significant difference (p=0.079). In addition to magnification, pain-related daily interference showed a significant impact on rumination (p=0.026), helplessness (p=0.004), and global PCS score (p=0.004). Contrary to pain catastrophizing thoughts, no difference was found in the GSI score, all nine SCL-90R components, as well as subjective sleep quality between the groups with increased and reduced pain severity and interference.

To predict the relevant risk factors for pain disability in patients with PPTTN, the three PCS subscales and the pain severity of BPI were input as independent variables, while the pain interference of BPI was input as a dependent variable in the multiple regression analysis (Table 5). The results revealed that pain severity (β=0.721, p<0.001) and the helplessness subscale (β=0.282, p=0.004) are two significant risk factors for pain interference in patients with PPTTN. Multicollinearity between independent variables was evaluated using VIF, which was within 2.0. The final model with two predictors was significant, explaining 86.5% of pain interference variance (F=100.579, p<0.001). In the follow-up correlation analyses, no significant relationship was found between the global score and three subscales of PCS and the five neuropathy features (superficial pain, deep pain, paroxysmal pain, evoked pain, and paresthesia) (Table 6). Unlike the neuropathy features, the affective (r=0.404, p=0.022 for depression; r=0.407, p=0.021 for anxiety; r=0.373, p=0.035 for GSI) and sleep (r=0.462, p=0.008) features showed significant correlations with the global PCS score (Table 6).

Table 5. Multiple regression results predicting pain interference from pain severity and pain cognition

Variable	Parameter estimate	Standard error	Standardized beta coefficient	Test statistic	p-value	95% confidence interval
Pain severity	0.805	0.100	0.721	8.035	<0.001	0.600, 1.010
Helplessness	0.272	0.087	0.282	3.140	0.004	0.095, 0.449
Constant	−0.332	0.149		−2.222	0.034	−0.637, −0.026
R^2			0.874			
Adjusted R^2			0.865			

The three subscales of Pain Catastrophizing Scale (magnification, rumination, and helplessness) and the pain severity of Brief Pain Inventory were input as independent variables. Pain interference was input as a dependent variable in the analysis.
Magnification (p=0.209) and rumination were excluded from the model using a stepwise selection.
All data were log-transformed for multiple regression analysis. F=100.579 (p<0.001).

Table 6. Correlation of sensory, affective, and sleep features with pain catastrophizing

Variable	PCS_M	PCS_R	PCS_H	PCS_G
	r (p-value)			
Neuropathy features				
Superficial pain	0.001 (0.999)	0.040 (0.829)	0.140 (0.446)	0.084 (0.647)
Deep pain	0.257 (0.155)	0.244 (0.178)	0.276 (0.126)	0.336 (0.060)
Paroxysmal pain	0.037 (0.840)	0.007 (0.969)	0.001 (0.999)	0.017 (0.927)
Evoked pain	0.048 (0.793)	0.042 (0.819)	0.106 (0.563)	0.061 (0.738)
Paresthesia	0.132 (0.470)	0.090 (0.625)	0.009 (0.961)	0.006 (0.974)
Affective features				
Depression	0.396 (0.025*)	0.386 (0.029*)	0.424 (0.016*)	0.404 (0.022*)
Anxiety	0.434 (0.013*)	0.443 (0.011*)	0.385 (0.029*)	0.407 (0.021*)
Somatization	0.274 (0.130)	0.296 (0.100)	0.254 (0.160)	0.266 (0.141)
GSI	0.389 (0.028*)	0.349 (0.050)	0.384 (0.030*)	0.373 (0.035*)
Sleep feature				
PSQI_G	0.152 (0.406)	0.463 (0.008**)	0.438 (0.012**)	0.462 (0.008**)

PCS, Pain Catastrophizing Scale; M, magnification; R, rumination; H, helplessness; GSI, global symptom index; PSQI, Pittsburgh Sleep Quality Index; G, global score.
All data were log-transformed.
Correlations between variables were performed using Pearson's correlation analysis. r indicates the correlation coefficient. *p<0.05, **p<0.01.

DISCUSSION

This study aimed to assess the psychological risk factors for pain experience and disability associated with PPTTN to better characterize the psychological sequelae of PPTTN. These psychological risk factors include general psychological factors, such as emotion and sleep, and pain-specific psychological factors, such as pain catastrophizing. Particularly, the study focused on the relative contributions of the three subscales of pain catastrophizing and pain interference related to PPTTN. Consistent with previous studies [7,28], the present study found that pain catastrophizing, its three subscales, and the PCS global score, as well as pain severity, were associated with pain interference due to PPTTN. Considering the dimensionality of pain reflecting pain interference (reactive dimension) as a consequence and reflection of the intensity of pain (sensory dimension), it is not surprising that pain severity was the strongest risk factor for pain interference related to PPTTN. Numbness partially improved over time; however, in the current study, no improvement in pain severity was noted. This finding suggests that pain sensation rather than numbness and psychological factors might be a critical factor in pain-related outcomes due to PPTTN. In the study, the proportion of poor sleep quality was up to 87.5%; however, the relationship between poor sleep quality and pain interference was not significant.

Notably, helplessness proved to be the strongest psychological sequela of PPTTN among the pain-related psychological factors. Magnification and rumination, the other two

PCS subscales, were removed from the stepwise regression analysis. The study findings confirm the key role of helplessness in explaining pain-related daily outcomes related to PPTTN, along with pain severity. The study results add to a growing body of research that indicates helplessness as a strong predictor of pain experience related to neuropathic pain [15,28]. In a previous study with 80 patients who had various neuropathic pains, including diabetic neuropathy, post-herpetic neuralgia, and postsurgical neuropathic pain, catastrophic thinking was closely linked to pain-related disability, and the helplessness subscale was the only pain catastrophizing dimension contributing to distinctive variance in pain prediction [28].

The uniqueness of PCS as a measure of the pain-specific psychosocial dimension is attributed to the fact that the three subscales reflect distinct aspects of pain-coping strategies. Previous studies have suggested the theoretical concept that pain catastrophizing might have two dimensions [21,29]. Rumination and magnification, which are dispositional aspects of pain, are regarded as the initial evaluation of the pain stressor, whereas helplessness is the subsequent evaluation in which the person develops a feeling of powerlessness and lack of control over the situation [21,29]. Based on the theoretical background and the findings of the current study, helplessness is the most significant pain-related psychological factor affecting pain outcomes in patients with a median symptom duration of 16 months (IQR=6.25–27.75). This clinical finding can be explained by the learned helplessness theory [17], which suggests that once pain becomes chronic, uncontrollable, and inescapable despite continuous coping efforts and treatments, patients may feel depressed cognitions of helplessness and use negative coping strategies due to a perceived lack of control [17,30]. Although a relationship was found between PCS and pain outcomes, it did not show significant associations with the pain ratings of various neuropathic sensory domains (Table 6). Consistent with these findings, Sullivan et al. [28] reported that pain catastrophizing was associated with affective pain ratings but not spontaneous and evoked pain ratings. In the present study, the global score and three subscales of PCS showed significant relationships with emotional distress, such as depression and anxiety, and poor sleep quality. Consistent with previous research [15,18,31], this finding suggests that pain catastrophizing, as a pain-specific psychosocial construct, is positively associated with general psychological components. However, it is also uniquely significant in pain-related outcomes independent of general psychological constructs. A previous systemic review that focused on brain changes during chronic pain using pain imaging has shown that pain catastrophizing is linked to changes in brain regions involved in pain processing, attention to pain, emotion, and motor activity, as well as impaired top-down pain inhibition [32]. Collectively, these findings suggest that the negative impact of catastrophizing on pain outcomes might be linked to changes in brain regions involved in processing chronic pain. Based on the findings of the present study, the implications of focusing on pain catastrophizing in managing PPTTN should be considered. However, only two randomized clinical trials have explored the effect of cognitive behavior therapy (CBT) targeting chronic peripheral neuropathic pain related to spinal cord injury [33] and burning mouth syndrome [34]. The systematic review on the "effect of psychological intervention for neuropathic pain," which included the two RCT studies mentioned above [35], did not establish the therapeutic efficacy of CBT. These results might be explained by the lack of research focusing on the specific psychosocial aspects associated with each type of neuropathic pain. Strong scientific evidence is lacking for CBT in patients with neuropathic pain [35]. However, previous studies targeting the efficacy of CBT with a 6–10 week duration in patients with chronic headache and chronic pain related to temporomandibular disorders have demonstrated that the treatment was associated with a reduction in pain catastrophizing, suggesting a possible clinical benefit of CBT intervention targeting pain catastrophizing in PPTTN [36,37]. Unfortunately, there is limited trial data in the field of PPTTN focusing on psychological interventions targeting pain catastrophizing. In particular, psychological interventions that emphasize coping with helplessness should be highlighted.

Standard pharmacological treatments frequently fail to alleviate neuropathic pain and are associated with significant side effects [38]. Reducing pain catastrophizing through psychological intervention might be helpful in the pharmacological management of PPTTN. In previous

research, high levels of catastrophizing were linked to an increased likelihood of pharmacotherapeutic inefficacy and an increased risk of treatment discontinuation during the 6-month follow-up period, resulting in poor pain outcomes in patients with neuropathic pain due to peripheral neuropathy [15]. These results suggest that increased catastrophizing has a maladaptive impact on pain outcomes by negatively affecting medication efficacy and compliance, as well as Axis II of pain. Furthermore, assessment and management of catastrophizing may reduce unnecessary medication overuse and improve medication compliance.

This study has several limitations. First, the preliminary study findings should be interpreted cautiously due to the small sample size. However, this study was performed on the hypothesis that the contribution of general and pain-specific psychosocial constructs to the pain-related outcomes of PPTTN might differ. Although the sample size is small, the significant association between pain catastrophizing, particularly helplessness, and pain disability in patients with PPTTN presents important clinical implications. Thus, a larger confirmatory study is required. Second, as a cross-sectional design, the outcomes of the present study cannot determine the causal relationships between pain catastrophizing and pain-related outcomes. However, considering the median pain duration of 16 months and the reactive dimension of helplessness, it can be assumed that chronic neuropathic pain following iatrogenic nerve injuries induces helplessness. Further research is required to investigate whether the relative impacts of the dispositional catastrophizing components (rumination and magnification) and the reactive catastrophizing dimension (helplessness) on pain outcomes depend on the duration of the pain. In spite of these limitations, this study has several strengths. For example, although the sample size was small, it consisted of a homogeneous cohort that served as representatives of PPTTN related to invasive dental treatments.

In conclusion, in the current study, the independent and unique role of pain-specific helplessness in the pain-related outcomes of the sample with PPTTN might provide insight into an improved understanding and management of pain disability in patients with PPTTN. Thus, it is important to emphasize the key role of helplessness in determining pain-related disability associated with PPTTN, as well as the establishment of coping strategies to manage helplessness.

CONFLICT OF INTEREST

Hye-Kyoung Kim has been the Editor-in-Chief of the *Journal of Oral Medicine and Pain* since April 1, 2022. Mee-Eun Kim serves as an editor of the *Journal of Oral Medicine and Pain* but was not involved in the decision to publish this article. Except for this, no potential conflict of interest relevant to this article was reported.

DATA AVAILABILITY STATEMENT

The datasets used in the current study are available from the corresponding author upon reasonable request.

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None.

AUTHOR CONTRIBUTIONS

Conceptualization: HKK, MEK. Investigation: HKK. Methodology: HKK. Supervision: MEK. Data analysis and interpretation: HKK, MEK. Writing original draft: HKK. Writing review-editing: HKK, MEK.

REFERENCES

1. Hajar R. The physician's oath: historical perspectives. Heart Views 2017;18:154-159.
2. Baad-Hansen L, Benoliel R. Neuropathic orofacial pain: facts and fiction. Cephalalgia 2017;37:670-679.
3. International Classification of Orofacial Pain, 1st edition (ICOP). Cephalalgia 2020;40:129-221.
4. Rosenberger DC, Pogatzki-Zahn EM. Chronic post-surgical pain - update on incidence, risk factors and preventive treatment options. BJA Educ 2022;22:190-196.
5. Nixdorf D, Moana-Filho E. Persistent dento-alveolar pain disorder (PDAP): Working towards a better understanding. Rev Pain 2011;5:18-27.
6. Tay AB, Zuniga JR. Clinical characteristics of trigeminal nerve injury referrals to a university centre. Int J Oral Maxillofac Surg 2007;36:922-927.
7. Smith JG, Elias LA, Yilmaz Z, et al. The psychosocial and affective burden of posttraumatic neuropathy following injuries to the trigeminal nerve. J Orofac Pain 2013;27:293-303.

8. Gustin SM, Wilcox SL, Peck CC, Murray GM, Henderson LA. Similarity of suffering: equivalence of psychological and psychosocial factors in neuropathic and non-neuropathic orofacial pain patients. Pain 2011;152:825-832.
9. Jensen MP, Chodroff MJ, Dworkin RH. The impact of neuropathic pain on health-related quality of life: review and implications. Neurology 2007;68:1178-1182.
10. Van der Cruyssen F, Peeters F, Gill T, et al. Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries. J Oral Rehabil 2020;47:1212-1221.
11. Garcia-Larrea L. Objective pain diagnostics: clinical neurophysiology. Neurophysiol Clin 2012;42:187-197.
12. Pfau DB, Geber C, Birklein F, Treede RD. Quantitative sensory testing of neuropathic pain patients: potential mechanistic and therapeutic implications. Curr Pain Headache Rep 2012;16:199-206.
13. Torta R, Ieraci V, Zizzi F. A Review of the emotional aspects of neuropathic pain: from comorbidity to co-pathogenesis. Pain Ther 2017;6(Suppl 1):11-17.
14. Ferini-Strambi L. Neuropathic pain and sleep: a review. Pain Ther 2017;6(Suppl 1):19-23.
15. Toth C, Brady S, Hatfield M. The importance of catastrophizing for successful pharmacological treatment of peripheral neuropathic pain. J Pain Res 2014;7:327-338.
16. Samwel HJ, Evers AW, Crul BJ, Kraaimaat FW. The role of helplessness, fear of pain, and passive pain-coping in chronic pain patients. Clin J Pain 2006;22:245-251.
17. Samwel HJ, Kraaimaat FW, Crul BJ, Evers AW. The role of fear-avoidance and helplessness in explaining functional disability in chronic pain: a prospective study. Int J Behav Med 2007;14:237-241.
18. Edwards RR, Dworkin RH, Sullivan MD, Turk DC, Wasan AD. The role of psychosocial processes in the development and maintenance of chronic pain. J Pain 2016;17(9 Suppl):T70-T92.
19. Meints SM, Edwards RR. Evaluating psychosocial contributions to chronic pain outcomes. Prog Neuropsychopharmacol Biol Psychiatry 2018;87(Pt B):168-182.
20. Sullivan MJL, Bishop SR, Pivik J. The pain catastrophizing scale: development and validation. Psychol Assess 1995;7:524-532.
21. Sullivan MJ, Thorn B, Haythornthwaite JA, et al. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain 2001;17:52-64.
22. IHS Classification ICHD-3. The International Classification of Headache Disorders 3rd edition [Internet]. International Headache Society; c2021 [cited 2023 Mar 17]. Available from: ttps://ichd-3.org/
23. Cleeland CS, Ryan KM. Pain assessment: global use of the brief pain inventory. Ann Acad Med Singap 1994;23:129-138.
24. Yun YH, Mendoza TR, Heo DS, et al. Development of a cancer pain assessment tool in Korea: a validation study of a Korean version of the brief pain inventory. Oncology 2004;66:439-444.
25. Derogatis LR. SCL-90-R: Administration, scoring and procedures manual-I for the (revised) version and other instruments of the psychopathology rating scale series. Clinical Psychometric Research; 1977.
26. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213.
27. Akhter R, Benson J, Svensson P, Nicholas MK, Peck CC, Murray GM. Experimental jaw muscle pain increases pain scores and jaw movement variability in higher pain catastrophizers. J Oral Facial Pain Headache 2014;28:191-204.
28. Sullivan MJL, Lynch ME, Clark AJ. Dimensions of catastrophic thinking associated with pain experience and disability in patients with neuropathic pain conditions. Pain 2005;113:310-315.
29. Martorella G, Côté J, Choinière M. Pain catastrophizing: a dimensional concept analysis. J Adv Nurs 2008;63:417-426.
30. Crisson JE, Keefe FJ. The relationship of locus of control to pain coping strategies and psychological distress in chronic pain patients. Pain 1988;35:147-154.
31. Jang HH, Kim ME, Kim HK. Pain catastrophizing mediates the effects of psychological distress on pain interference in patients with orofacial pain: a cross-sectional study. J Oral Facial Pain Headache 2018;32:409-417.
32. Malfliet A, Coppieters I, Van Wilgen P, et al. Brain changes associated with cognitive and emotional factors in chronic pain: a systematic review. Eur J Pain 2017;21:769-786.
33. Heutink M, Post MWM, Bongers-Janssen HMH, et al. The CONECSI trial: results of a randomized controlled trial of a multidisciplinary cognitive behavioral program for coping with chronic neuropathic pain after spinal cord injury. Pain 2012;153:120-128.
34. Miziara ID, Filho BC, Oliveira R, Rodrigues dos Santos RM. Group psychotherapy: an additional approach to burning mouth syndrome. J Psychosom Res 2009;67:443-448.
35. Eccleston C, Hearn L, Williams AC. Psychological therapies for the management of chronic neuropathic pain in adults. Cochrane Database Syst Rev 2015;2015:CD011259.
36. Litt MD, Shafer DM, Ibanez CR, Kreutzer DL, Tawfik-Yonkers Z. Momentary pain and coping in temporomandibular disorder pain: exploring mechanisms of cognitive behavioral treatment for chronic pain. Pain 2009;145:160-168.
37. Thorn BE, Pence LB, Ward LC, et al. A randomized clinical trial of targeted cognitive behavioral treatment to reduce catastrophizing in chronic headache sufferers. J Pain 2007;8:938-949.
38. Cavalli E, Mammana S, Nicoletti F, Bramanti P, Mazzon E. The neuropathic pain: an overview of the current treatment and future therapeutic approaches. Int J Immunopathol Pharmacol 2019;33:2058738419838383.