Effects of Mood Induction on the Pain Responses in Patients with Migraine and the Role of Pain Catastrophizing

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Migraine has close associations with depression and anxiety. Catastrophizing, an alarmist reaction to pain, has been proposed as one of the mediators in the relationship between headache and emotional distress. However, much experimental evidence is needed to make such a view more validated. The aims of this study are to examine the effects of mood induction on the pain responses and to investigate the role of pain catastrophizing in the relationship between pain and mood amongst a sample of patients with migraine. For this purpose, 60 patients with migraine were recruited from a headache clinic in Tehran-Iran and were randomly assigned into one of three groups: negative mood induced group, positive mood induced group and control group. The following instruments and measures were used in this study: mood induction by presenting different types of films (positive, negative), a computerized cognitive task to elicit pain, Beck Depression Inventory and Pain Catastrophizing Scale. The results showed that while the induction of depressed mood increased the pain intensity, the induction of positive mood reduced it significantly ($p < 0.05$). Further analyses revealed that catastrophizing is as a confounding factor in the relationship between pain and mood. Once catastrophizing scores were entered into the analyses as a covariate, the significant effect of mood on the pain intensity reduced. In conclusion, both mood and catastrophizing are important factors in understanding the migraine pain. Clinical implications of these findings are discussed in the paper. Copyright © 2014 John Wiley & Sons, Ltd.

Key Practitioner Message:
- Pain-related catastrophizing and mood induction are important factors in understanding pain intensity amongst patients with migraine pain.
- Catastrophizing as a confounding factor in the relationship between pain and mood may partially mediate the relationship between mood and pain.
- Therapeutic interventions should focus on the reduction of depression and catastrophizing.

Keywords: migraine, depression, mood induction, pain catastrophizing

INTRODUCTION

Headache is one of the most common medical complaints resulting in presentation to a doctor’s office, and it is frequently associated with substantial personal and societal burdens (Chai, Rosenberg, & Peterlin, 2012). Globally, it has been estimated that the prevalence of current headache disorder among adults is 47% (World Health Organization, 2012).

Headache International Society (HIS) has divided the headaches into two categories: primary and secondary. In primary headaches, there is inherent dysfunction of the nervous system, and this genetic readiness potentially increases the vulnerability to headache attacks. However, the secondary headache is a condition in which pain is a secondary event to physical processes (Olesen, 2004).

Migraine is the most common primary headache disorder (Tepper & Tepper, 2011). It occurs in about 18% of females and 6% of males (Bray, Heath, & Militello, 2013). About 94% of patients that come to headache clinics are diagnosed with migraine (Tepper & Tepper, 2011). Also, migraine is the prevalent headache in Iran, and about one fifth of Iran’s population suffers from it (Nazari & Eghbali, 2012). The disease is more common among women, and the higher prevalence of migraine headaches in women during the productive years together with bearing social and economic responsibilities has turned migraine into a highly significant issue in for the women’s health (Nazari & Eghbali, 2012).

Migraine is a disabling recurrent headache, which is broadly divided into migraine with or without aura.
Migraine is characterized as moderate to severe, often unilateral and pulsating characterized headache attacks, which is aggravated by physical activity and accompanied by vegetative symptoms such as nausea, vomiting, photophobia and phonophobia. According to the definition of the HIS, the duration of attacks ranges from 4 to 72 h. From a pathophysiological point of view, migraine is understood as a paroxysmal central nervous system dysfunction. It has also been suggested that some types of migraine have a genetic basis (May, 2008).

Pain has been defined as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage' or described in terms of such damage (International Association for the Study of Pain (IASP) (Merskey, 2007). The biopsychosocial model of pain has been proposed to explain pain (George et al., 2008). This model emphasizes the role of cognitive and psychological factors such as depression and anxiety in processing and adjustment to pain. The comorbidity of migraine with depression, anxiety and stress has been documented in several studies (e.g., Gerrits et al., 2012; Ligthart, Gerrits, Boomsma, & Penninx, 2013; Mehdizadeh & Ghaffarinejad, 2011; Moon, Smith, Lahr, & Cutrer, 2013; World Health Organization, 2012). According to a number of population-based studies from North America and Europe, among migraine sufferers, the odd ratio of those having depression is higher than of those without this condition (the odd ratio ranges from 1.3 to 5.8) (Breslau et al., 2003). Depression is the most common disorder amongst patients with migraine; it has been seen in about 80% of migraine sufferers (Fuller-Thomson, Schrumm, & Brennenstuhl, 2013; Gerrits et al., 2012). Depression can cause headaches that cannot be differentiated from migraine (Kindler et al., 2012; Patten et al., 2008; Silberstein, Lipton, & Dalessio, 2001), and furthermore, depression can lead to higher levels of perceived pain (Williams et al., 2012) and disability (Asghari, Julaiæiha, & Godarsi, 2008; Huijnen et al., 2010).

However, cognitive errors have important roles not only in pain experience but also in how patients react to pain and adjust to it. Extensive studies have shown that higher levels of pain-related catastrophizing were a significant predictor of more severe pain (Asghari & Golak, 2005; Velly et al., 2011), physical disability and depression (Asghari & Golak, 2005; Bergbom, Boersma, Overmeer, & Linton, 2011). Also, pain catastrophizing has a mediating role in the relation between pain intensity and depression, and it may confound this relation (Wood et al., 2013). Baseline catastrophizing and depression were the main predictors for pain (as measured at baseline) 6 to 12 months later in patients (Meeus et al., 2012).

Many mood-induced studies have been performed to investigate the effect of depressed and/or elated mood on the pain responses (Weisenberg, Raz, & Hener, 1998; Willoughby, Hailey, Mulkana, & Rowe, 2002), pain tolerance and pain intensity (Strand et al., 2006). However, most of the research has been conducted amongst healthy pain-free individuals or amongst patients that suffered from chronic low back pain (Tang et al., 2008). Migraine is a recurrent headache; therefore, the results from other pain conditions in other sites of body such as low back pain, which mostly is as a persistent pain, may not be generalizable to patients with migraine headache, especially as prevalence is increasing in Iran, particularly in women (Nazari & Eghbali, 2012). Therefore, it is necessary to continue research in this area on patients with migraine.

In this study, using a group of patients with migraine, we measured the intensity of pain before and after mood induction to clarify to what extent the results of mood induction amongst these patients are similar to results previously reported on patients with chronic persistent pain (e.g., low back pain). So, the objectives of this study were to determine (1) whether the experimentally induced depressed and elated moods can change perceived pain intensity, (2) what the role of pain catastrophizing is in the relation between pain and mood in patients with migraine and whether catastrophizing can confound this relationship. We hypothesized that there is a significant relationship between pain and mood, in which mood change can increase or decrease the perceived pain intensity, similar to the results previously reported on patients with chronic low back pain. However, when the catastrophizing scores are taken into account, the significant magnitude of this relationship would be reduced and catastrophizing would have a confounding role in the relationship between mood and pain.

METHOD

Design

To investigate the addressed questions in the present study and after examining inclusion/exclusion criteria, all eligible participants were asked to complete baseline self-report measures of catastrophizing, depressed or cheerful mood and pain intensity prior to receiving pain and mood induction. Following this, a clinically relevant pain provoking task (computerized task of cognitive exhaustion) to elicit pain was used, and after this stage, the mood induction procedure by showing a film was applied, with patients with migraine randomly allocated to depressed, control or elated mood conditions. Dependent variables were pain ratings during the computerized challenge and mood induction. Measures were taken immediately after the baseline level, after the mood induction and after the computerized task. Figure 1 depicts the procedure and measures used in the study. A $3 \times 2 \times 2$ repeated-measures design was used to analyze data. In these analyses, data from the mood induction type
(depressed, control, elated) served as the between-subjects variable and manipulation (baseline: mood/pain 1, after mood induction: altered mood/pain 3) and task (after the first computerized task: pain 2, after the second computerized task: pain 4) served as the within-subjects variables. The study had received full ethical approval.

Participants

All of participants were patients with migraine seeking medical treatment for migraine at an outpatient headache clinic in Tehran-Iran. Patients were included in the study if they were (1) aged between 18 and 65, (2) had a formal diagnosis of migraine by a neurologist, (3) had a complaint of migraine for 6 months or longer and (4) had a high school certificate (12 years of formal education). Patients excluded from the study were those with (1) severe depression or anxiety, (2) severe daily headache that may interfere with their cooperation and (3) pregnancy. Data related to demographic status (age, educational level, marital status and duration of migraine) were also collected.

Of the 110 patients who were approached and screened for the purpose of this study, 30 did not meet the criteria specified above (severe depression or anxiety = 15; pregnancy = 5; severe daily headaches and disability = 10).

Data analyses were planned only for those in whom the mood responses were consistent with the induction condition they were assigned to. As such, whilst a total of 80 participants completed the study, final sample comprised 60 patients with chronic migraine: 20 in the depressed group, 20 in the control group and 20 in the elated group. This sample size has been selected with regard to the previous experimental studies (e.g., Tang et al., 2008).

Procedure

After selecting the participants, they were told that the aim of the study is to evaluate ‘the effect of different films on migraine pain and task performance’. Following this, each patient was asked to sign a consent form if s/he was interested to participate in this study. To minimize the effect of substances on their response to the experiment, we asked the participants to abstain from nicotine, pain/mood medications and caffeinated food and beverages 3 h prior to attending the appointment. The testing session was approximately 1 h long and took place in a sound-attenuated experimental room. All participants took part individually in the absence of a spouse/partner and with minimal interaction between the participants and the experimenter.

After obtaining informed consent, participants were asked to complete a series of questionnaires including pain catastrophizing, depressed/cheerful mood and pain intensity based on their experience of migraine. Before commencing the experiment, the participants were given 5 min to familiarize themselves with the surroundings and they were asked to browse a neutral themed magazine for 5 min to control the effect of the mood induction procedure itself and to establish the baseline of various mood and pain measures. Then, the participants were asked to provide a rating of the levels of their cheerfulness (as ‘cheerfulness rating’), depression (as ‘depression rating’) and pain (as ‘pain rating 1’), using a 0–100 rating scale with the referent being ‘right now’. A close examination of data at this stage (ANOVA analysis) showed that the baseline pain rating score of some participants (nine participants) made a significant difference within the subjects. For maintaining homogeneity among participants in the experiment, these patients were excluded from the study.

Computerized Cognitive Exhaustion Task

At the next step (step 2), a pain-provoking task was used (computerized cognitive exhaustion task), and again, immediately after its fulfilment, all participants were asked to rate their pain intensity (pain 2). This task was based on the theory, proposed by Sedek and Kofta (1990), of the effect of cognitive exhaustion on learned helplessness and stress. The computerized task consisted of four discrimination questions with eight tasks for each of them (32 slides). Participants were asked to find the correct answer to eight tasks in each question. The task instruction
has been clearly explained by using an example, and also, it was told that there was not any time limitation on answering. In the first three questions, they could rationally find the correct answer during problem solving attempts, but in the last question, they were exposed to a source of inconsistent, self-contradictory task information. The flow of such information makes the hypothesis-testing activity futile. Prolonged and inefficient activity of this kind leads, in turn, to the emergence of a state of cognitive exhaustion and stress. Previously, this task provoked helplessness and stress with a high degree of validity (0.92) and reliability among a Persian population (0.96) (Shokri, 2003). This result is due to the fact that stress has an inevitable role in migraine, and it has been shown that 50–80% of people with migraine believed that stress has an important role in their migraine headaches. These patients also reported that the incidence of migraine is higher if they had experienced stressful situations in the previous year (Radat, 2013), so it was concluded that this task can be efficient in provoking or increasing pain. For this purpose, a pilot study was conducted on 10 patients with migraine, and they were asked to rate their pain before and after task performance on a continuum 0–10, and according to mean elicited pain intensity, the computerized task is an appropriate tool to elicit pain.

For ethical reasons, the participants were explained that they could leave the experiment at any time. At this stage of the study, another five participants were excluded from the study because of the fact that, while the rate of their pain increased, but this increase was less than 20 scores, so it was not according to the pre-determined mood change criterion.

**Mood Induction by Film**

After a 5-min break, the participants were shown a film for mood induction according to their group assignment. Like previous research in this area (e.g., Dennis & Solomon, 2010; Herring, Burleson, Roberts, & Devine, 2011; Radstaak et al., 2011; Tang et al., 2008), the length of mood induction procedure was 12 min. For those who were assigned to the depressed group, a sad film clip (Dennis & Solomon, 2010) and for those who were assigned to the elated group, a happy one was shown (Herring et al., 2011). The members of the control group did not watch any films; they were asked to review a neutral magazine for 12 min.

When the film time was over, the participants were asked to provide a second cheerfulness rating and a second depression rating as the manipulation checks, and in addition, to evaluate the effect of mood on pain ratings, the participants were then asked to give additional pain rating (pain 3). At this stage, six more participants were excluded from the experiment because their mood rating did not change compared with step 1. At the end of the experiment (step 4), the cognitive exhaustion task was performed again and the participants were asked to rate their pain intensity (pain 4). Before leaving the session, we made sure that all participants were in normal conditions, and then, they received a small reward for their participation.

**Questionnaires**

Three standardized questionnaires were administered before the experiment to index the participants’ pain and psychological characteristics:

**Beck Depression Inventory**

To index mood disturbance, we asked the participants to complete the Beck Depression Inventory (BDI) based on how they had been feeling over the past week. The BDI is a 21-item, self-report rating inventory that measures characteristic attitudes and symptoms of depression (Beck et al., 1961). Total depression score ranges from 0 to 63. Scores of >15 identify depressed cases (as obtained in a study amongst Iranian population) (Montazeri, Vahdaninia, Ebrahimi, & Jarvandi, 2003). This inventory has a very high level of validity and reliability (0.86) (Beck, Steer, & Garbin, 1988), and the Persian version of this inventory also has high reliability (0.87) (Rajabi, Attari, & Haghighat, 2001).

**Pain Catastrophizing Scale**

The Pain Catastrophizing Scale (PCS) is used to assess individual responses to pain and to predict the levels of pain and distress among clinical patients (Sullivan, Bishop, & Pivik, 1995). This scale has 13 items with three subscales of rumination, magnification and helplessness. Reliability and validity of this scale among clinical and non-clinical population is high (α 0.90) (Fernandes, Storheim, Lochting, & Grotle, 2012). Among Persian people, PCS has very high test–retest reliability and internal consistency (r = 0.87) (Rahmati, 2011).

**Pain Numerical Rating Scale**

Patients reported their pain intensity, using a numerical rating scale. The NRS asks patients to rate their pain intensity right now on a 0 to 100 (101 point) scale where 0 indicates ‘no pain’ and 100 means ‘pain as bad as it could be’. The psychometric properties of the NRS have been confirmed among different groups of patients with pain (Jensen & Karoly, 2001).
Effects of Mood Induction on pain and the Role of Pain Catastrophizing

**Treatment of Data**

Considering the central focus of this study (i.e., the effects of experimentally induced mood states on pain rating), pre-determined mood and pain change criterion was adapted to judge whether a participant's data should be used for analysis. Data from manipulation of depressed and elated mood or from pain provoking condition was considered acceptable for further analysis if (1) the mood/pain ratings changed in the predicted direction and (2) the magnitude of the change was equal to 20 scores or more (on a 0–100 rating scale). For the control group, data were accepted for a further analysis if there was no significant change in the mood/pain scores (i.e., change < 20 scores in either direction). A total of 11 participants did not meet these pre-determined criteria, so their data were excluded from further analysis (five and six participants were excluded at the second and third step, respectively) in addition to nine participants at step 1. To sum, the final sample for this study reduced to 60 participants.

A series of one-way analysis of variance (ANOVA; with Tukey’s/Dunnnett’s T3 post hoc tests) were performed to detect if there were any significant differences among the three groups on the continuous variables of age, time since pain started, pain intensity, pain catastrophizing and depression.

For the pain ratings, a $3 \times 2 \times 2$ repeated-measures ANOVA was performed, with the induction type (depressed, control, elated) as the between-subjects variable and manipulation (baseline: mood/pain 1, after mood induction: altered mood/pain 3) and task (after the first computerized task: pain 2, after the second computerized task: pain 4) as the within-subjects variables. This allows us also to examine the impact of the computerized cognitive exhaustion task on the pain ratings and a three-way interaction among these variables. Additionally, the effect sizes were calculated using the formula, Cohen’s $d=M1-M2/spooled$ (Cohen, 1992), to index the magnitude of changes in the pain outcome measures before and after the mood induction. Correlation analyses (Pearson) were also performed to examine the relationship between the changes in mood and the changes in the pain responses.

**RESULTS**

**Participant Characteristics**

As a group, this sample had a mean age of 34.52 years (SD = 9.75). All were female, and the majority of them were married (80%). In almost 85% of the participants, the interval between two migraine attacks was less than 15 days. Mean duration of the onset of migraine headache was reported to be 19.33 months (SD = 18.01). The average pain intensity is 12.23 (SD = 1.26) on a 0–100 numerical rating scale.

As it can be seen from Table 1, the three groups of participants did not differ significantly from each other in terms of demographic (age and duration of migraine), baseline pain intensity and depression, while there was significant difference among three groups in pain catastrophizing. All main effects did not approach significance ($p = 0.18–0.88$), with the exception of the pain catastrophizing score ($p = 0.048$).

**Mood Ratings as Manipulation Checks**

Figure 2a and 2b depicts the changes in the cheerfulness and depression ratings by induction type before and after the mood manipulation. For the depression rating, there was a significant interaction between induction type and manipulation [$F(2, 57) = 220, p < 0.001$]. As expected, the depressed group reported an increase in their negative mood [$t(19) = -12.51, p < 0.001$]. However, the control group showed no significant change [$t(19) = 1.37, p = 0.189$], and the elated group showed a decrease [$t(19) = 12.103, p < 0.001$] in the depression level following the mood induction.

There was a significant main effect for induction type [$F(2, 57) = 27.61, p < 0.001$], meaning that amongst depressed group, the average change of pre-mood to

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**Table 1. Demographic data and the baseline measures of pain, age, depression, catastrophizing for the participants (n = 60)**

<table>
<thead>
<tr>
<th></th>
<th>Depressed group (20)</th>
<th>Control group (20)</th>
<th>Elated group (20)</th>
<th>$F(2, 57)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>35.05 (7.80)</td>
<td>33.45 (10.30)</td>
<td>35.05 (11.30)</td>
<td>0.17 (ns)</td>
</tr>
<tr>
<td>Pain duration (month)</td>
<td>13.85 (6.90)</td>
<td>24.25 (25.01)</td>
<td>19.90 (16.60)</td>
<td>1.72 (ns)</td>
</tr>
<tr>
<td>Pain intensity (time 1)</td>
<td>11.75 (1.32)</td>
<td>11.55 (1.06)</td>
<td>13.4 (1.43)</td>
<td>0.12 (ns)</td>
</tr>
<tr>
<td>Pain catastrophizing (time1)</td>
<td>26.95 (11.32)</td>
<td>23.3 (12.56)</td>
<td>29.05 (9.64)</td>
<td>1.33</td>
</tr>
<tr>
<td>Depression (BDI) (time 1)</td>
<td>11.95 (7.39)</td>
<td>10.8 (8.76)</td>
<td>13.95 (4.8)</td>
<td>0.98 (ns)</td>
</tr>
</tbody>
</table>

The means are reported with standard deviations put in parentheses. Pain intensity from NRS (range between 0 and 100), PCS = Pain Catastrophizing Scale (range from 0 to 65); BDI = Beck Depression Inventory (range from 0 to 63).

*ns* = not significant.
post-mood induction was higher, compared with that of the control and the elated group. Furthermore, the overall of depression rating was higher after the mood induction compared with that mentioned before.

Similarly, for the cheerfulness rating, there was a significant interaction between induction type and manipulation \( F(2, 57) = 39.79, p < 0.001 \), meaning that as expected, the depressed group reported a decrease \( t(19) = 6.52, p < 0.001 \), the control group no significant change \( t(19) = -1.405, p = 0.176 \) and the elated group an increase \( t(17) = -4.39, p < 0.001 \) in cheerfulness following the mood induction. There was also a significant main effect for induction type \( F(2, 57) = 9.98, p < 0.0001 \), meaning that the average of the pre-mood to post-mood induction cheerfulness ratings of the elated and the control groups were higher compared with that of the depressed group.

**Effects of Mood Induction on Pain Ratings**

Figure 3 and Table 2 depict the changes in the pain ratings over the course of the experiment. In the \( 3 \times 2 \times 2 \) ANOVA (induction type—manipulation—task), there was a significant main effect for manipulation \( F(1, 57) = 161.496, p < 0.0001 \) and for task \( F(1, 57) = 52.37, p < 0.0001 \); the task effect showed that the pain rating across groups was significantly higher after computerized task compared with the one before \( p < 0.0001 \).

The main effect of manipulation was modified by a significant manipulation–induction type effect \( F(2, 52) = 3.752, p = 0.029 \). This interaction was accounted for by a significant increase in pain ratings (pain 1 and pain 3) in the depression group \( t(19) = -11.18, p < 0.001 \), pre-post manipulation effect size = \(-1.95\) and change in the control group \( t(19) = -5.25, p < 0.001 \), pre-post manipulation.
Effects of Mood Induction on pain and the Role of Pain Catastrophizing

Table 2. Mean (standard deviation) of pain rating in the three study groups at four steps (before and after mood induction)

<table>
<thead>
<tr>
<th>Pain Step</th>
<th>Depressed group (20)</th>
<th>Control group (20)</th>
<th>Elated group (20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain 1</td>
<td>11.75 (1.32)</td>
<td>11.55 (1.063)</td>
<td>13.40 (1.43)</td>
</tr>
<tr>
<td>Pain 2</td>
<td>24.00 (1.32)</td>
<td>23.65 (9.43)</td>
<td>27.9 (1.12)</td>
</tr>
<tr>
<td>Pain 3</td>
<td>38.7 (1.43)</td>
<td>21.35 (8.44)</td>
<td>15.15 (9.20)</td>
</tr>
<tr>
<td>Pain 4</td>
<td>48.15 (1.42)</td>
<td>27.50 (1.24)</td>
<td>20.00 (1.63)</td>
</tr>
</tbody>
</table>

Association Between Changes in Moods and Changes in Pain Ratings

To ascertain the association between the induced variations in mood and the subsequent changes in the pain responses, differences in mood ratings, pain ratings before and after the mood induction were calculated (by subtracting the pre-induction value from the post-induction value) and subjected to Pearson correlational analysis.

As it is evident from Table 3, all correlations were statistically significant and moderate in strength (r range = 0.63–0.76). Increase in the cheerfulness rating was negatively correlated with increase in pain ratings. However, increase in the depression rating was positively correlated with increase in pain ratings.

Role of Pain Catastrophizing in Pain Rating

In another analysis, pain catastrophizing variable was controlled as a covariate. In a 3 × 2 × 2 ANCOVA (induction type—manipulation—task), there was a significant main effect for manipulation [F(1, 56) = 14.50, p < 0.0001], and when pain catastrophizing scores were taken into account, in the interaction between manipulation and catastrophizing, there was a significant difference between participants in pain rating in manipulation [F(1, 56) = 1.35, p = 0.025]. While in this condition, the interaction between manipulation and induction type was not statistically significant and the magnitude of significance decreased [F(2, 56) = 3.05, p = 0.055]. These results showed that the pain catastrophizing is important in the relationship between pain and mood.

Although there was a significant main effect for task [F(1, 56) = 5.19, p = 0.02], when the effect of pain-related catastrophizing was taken into account, the significant differences between participants regarding their pain rating in task [F(1, 56) = 0.26, p = 0.60] was no longer significant. However, the interactive effect of task-induction type was significant [F(1, 56) = 79.49, p < 0.001], and also, the main effect for the covariate factor (pain catastrophizing)
DISCUSSION

The results of this experiment indicate that mood can alter the reported level of pain among patients with migraine in the predicted directions. While induction of depressed mood resulted in significantly higher pain ratings, the induction of happy mood resulted in significantly lower pain ratings; in the control group, however, there was no significant change in pain rating. As to the second hypothesis, the results showed that pain catastrophizing confounds the relationship between pain and mood intensity among patients with migraine. Once catastrophizing was taken into account, the significant effects of mood on pain intensity were reduced.

The above findings were, as expected, consistent with the considerable body of previous epidemiological and descriptive research that suggested a strong association between chronic pain and depressed mood. In addition, they support the suggestion that happy mood can significantly reduce pain perception (Boettger, Schwier, & Bär, 2011; Strand et al., 2007; Tang et al., 2008; Terhaar et al., 2010).

The relationship between migraine and depression, however, is likely to be bidirectional (Breslau et al., 2000; Breslau et al., 2003). Breslau and colleagues (Breslau et al., 2000; Breslau et al., 2003) have shown that those reporting depression at baseline have a higher risk of first-onset migraine during the 2-year follow-up period and that those with migraine at baseline have an increased risk of developing first-onset major depression during follow-up. Furthermore, there are some other studies that indicated that depression can cause headaches non-differentiable from migraine (Kindler et al., 2012; Patten et al., 2008) and depression can lead to higher levels of perceived pain (Williams et al., 2012). In sum, it can be said that depressed mood can lead to more pain intensity and pain, in turn, it can lead to more severe depressed mood. This conclusion can be confirmed especially when it is observed that improvement in depression correlates with a reduction in migraine frequency and its intensity, which leads to greater improvement of depression (Moon et al., 2013).

Depression has been shown to be associated with pain, regardless of the anatomical site (i.e., head, back and lower back). Depression is also a risk factor for developing multiple pain sites (Ligthart et al., 2013). This suggests that a considerable part of the comorbidity of migraine and other types of pain may be explained by depression (Ligthart et al., 2013). These findings emphasize the importance of accounting for depression in studies of migraine and pain comorbidity. Awareness of this comorbidity may facilitate adequate treatment of types of conditions. Altogether, these results are consistent with our findings. Although the current study did not examine the bidirectional relationship between pain and mood, it showed the presence of strong relationship between mood and pain intensity in patients with migraine. Certainly, longitudinal studies will be needed to certify the bidirectional relationship between mood and migraine pain. However, some results in this area are not consistent. Using the same design and pain measures, some studies have reported that depressed mood decreases pain tolerance but has no effect on pain ratings (Willoughby et al., 2002). However, other studies have reported that positive mood and not negative mood can change the pain tolerance and/or pain ratings (Weisenberg et al., 1998). One justification for these inconsistent results is that the participants of those studies were healthy pain-free individuals who participated in experimental induced pain procedures (e.g., cold pressor pain) (e.g., Weisenberg et al., 1998; Willoughby et al., 2002), while the participants of the current study and some other previous studies were clinical patients.

Another finding of this study was that catastrophizing is a confounding variable in the relationship between mood and pain intensity in patients with migraine. Our findings about the role of catastrophizing seems to be consistent with the previous research suggesting that pain catastrophizing has a mediating role in the relationship between pain intensity and depression amongst patients with heterogeneous chronic pain (Geisser, Robinson, Keefe, & Weiner, 1994; Wood et al., 2013). According to our study, once catastrophizing was entered into the analyses, the significant association between pain and depression is reduced. Moreover, consistent with previous work (Wood et al., 2013), pain catastrophizing can lead to increased pain independent of depressed mood. It can be concluded that induction of negative mood (as a negative situation) can lead to activation of dysfunctional attitudes towards pain (i.e., catastrophizing) and consequently increases the perceived pain intensity. These findings suggest that when people with migraine are in a negative situation, these situations will activate the cognitive error such as catastrophizing. This issue, in turn, will lead to a negative evaluation of situation and reduce their ability to cope with pain. This finding demonstrates that cognitive factors have important role in relationship between pain intensity and mood in patients with migraine and supports a cognitive-behavioural mediation model (Rudy, Kerns, & Turk, 1988).

Since the association between mood and pain is multifactorial (Breslau et al., 2003; Stam et al., 2010; Surah,
Baraniaharan, & Morley, 2014), other factors such as genetic factors, neurochemical abnormalities, stress and anxiety are thought to play a key role in the association. That is why the catastrophizing may partially mediate the relationship between mood and pain, which the current results were consistent with it.

The current study is the first one to evaluate the effect of experimentally induced depressed and elated moods on pain responses amongst Iranian people with migraine headache. This study differs from the previous studies due to the following three reasons:

1. A number of relevant studies (as reviewed above) tested the same mood effect in healthy pain-free volunteers. It seems reasonable to hypothesize that depressed mood may have a more profound effect on pain responses in chronic pain patients with migraine than in healthy pain-free individuals;
2. Most of the previous research has been performed amongst low back pain as a persistent chronic pain, but the present study has been performed on migraine as a recurrent chronic pain;
3. In this study, the important role of catastrophizing was investigated in relation to mood pain in patients with migraine that was not focused on by previous researches.

Limitation and Future Researches

There is a number of limitations that must be considered when interpreting these results: first, in the current study, the patients with migraine were recruited from a pain clinic and they suffered from migraine, therefore, no any other forms of pain in addition to their headaches was not considered. According to the previous study, the awareness of this comorbidity may facilitate adequate treatment of types of conditions in regard to depression (Ligthart et al., 2013). As recent research has indicated, catastrophizing depends significantly on pain type and age (Ruscheweyh et al., 2011), so other types of pain in different age groups should be considered. Second, this sample represents females with migraine pain and may not be representative of all people in the community with migraine. Third, although migraine is a recurrent pain that makes it possible low baseline pain intensity, it is recommended that future studies should select a patient group with migraine with higher baseline pain intensity and then compare the findings. Fourth, in the current study, the patients with severe depression and severe daily headache were excluded because of controlling some confounding and unwanted factors. So, the participants who completed the study may not be representative of the wider population of people with migraine, that in future studies should be considered.

Additional research is needed to replicate our findings, especially with a neutral group, which will watch a neutral film, beside the other experimental groups, and to explore other pain-related variables that may contribute to the mood and pain rating in patients with migraine. Future research might include more assessment of mediating or moderating role of catastrophizing and potential mediating and confounding variables (such as stress, attentional processes, automatic negative thinking and rumination) and look into the possible interaction between depressed mood and the level of arousal associated in the modulation of pain to control the mechanisms that may be involved in the way in which mood modulates the experience of pain.

CONCLUSIONS

In conclusion, the present study clearly demonstrates that experimentally induced changes in mood influence self-reported pain of a group of patients with chronic migraine and also confirmed the importance of pain catastrophizing as a confounding in relation between pain and mood. These findings have important implications for pain assessment and treatment. Translational research of this kind offers opportunities to better understand both the mechanisms involved in the psychological modulation of chronic pain and the ways in which pain can be reduced and better tolerated, and they emphasize the importance of using appropriate interventions to reduce catastrophizing and to improve coping strategies to modify the pain experience in adults with migraine. As depression and catastrophizing are associated, in the most, with more severe pain and sensitivity to pain, physical disability, poor treatment outcomes and potentially with early mortality (Kwon & Chang, 2013), a variety of interventions mainly in the form of cognitive behavioural treatment from cognitive, behavioral to medication seem to be effective in treatment of migraine headache (Dindo et al., 2012; Jensen et al., 2012).

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