

Six Months Post Myocardial Infarction Depression: Is Acute PTSD a Predisposing Condition?

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Abstract

Background: Depression has been recognized as one of the most critical psychological issues following a Myocardial Infarction (MI), its presence associated with readmissions and death, augments healthcare costs and increases utilisation of services. For theoretical and clinical reasons, Post-traumatic Stress Disorder (PTSD) should be considered as a predisposing psychological condition for depression. However, its impact on depression's intensity and presence 6 months after a MI has not been clearly assessed.

Methods: Out of the 870 eligible patients in three Canadian hospitals, 339 completed the research protocol. Patients completed a depression (BDI-II) and a PTSD (MPSS-SR) inventory 48 hours to 14 days post MI to assess the prevalence of both disorders. They again completed the BDI-II six months after their MI to investigate the predisposing effect of PTSD on depression.

Results: Based on the symptomatology cut-off point of their respective measurement instruments, the prevalence of comorbid PTSD-depression was 11.5%. Patients with PTSD symptomatology one month post-MI report high level of depression symptomatology 6 months after the MI. The level of depression at 6 months for comorbid patients was not different from the depressed or traumatized patients' level at 1 month.

Conclusion: The results suggest that the presence of PTSD symptomatology at 1 month is a predisposing condition for the development of depression and its evaluation in a post MI investigation routine is recommended.

Keywords: Acute post-traumatic stress disorder; Depression; Comorbidity; Myocardial infarction; Prevalence

Introduction

Myocardial Infarction (MI) is an acute, life-threatening event that can lead patients to develop a wide range of complications and may ultimately cause death. Among the consequences following an MI, depression has been recognized as one of the most critical psychological issues [1-4]. Not only is it a frequent disorder, with studies reporting prevalence as high as 40% [5], but its presence in the months following an MI is associated with adverse complications. Depression consequent to a MI predicts cardiac readmissions and death, augments healthcare costs and increases utilisation of services [6,7]. Patients with subclinical levels of depression present a similar prognostic and clinical picture [8].

An effective way to prevent the detrimental effects of depression is to detect as early as possible the predisposing conditions accountable for its development. Therefore, timely identification of variables associated with the depressive state of a patient in the months following a MI could help improve his/her chance of survival [9], improve quality of life [10,11] and reduce the burden on the health care system [9].

Among the variables associated with the presence of depression is the presence of Post-traumatic Stress Disorder (PTSD) symptomatology [12,13]. PTSD, an anxiety disorder affecting up to 1 in 5 patients after a MI [14,15], can develop after the exposure to a potential traumatic event. Its symptoms, which include re-experiencing, avoidance and emotional numbing among others, must be present at least one month after the event to establish the diagnosis [16].

There are theoretical and clinical reasons why PTSD should be considered as a predisposing psychological condition for depression. First, PTSD symptoms can be so distressing and debilitating that they can facilitate the development of depressive symptomatology [17]. For

instance, emotional numbing, which acts as a protection mechanism after the trauma, can nurture depressive feelings by impeding the experience of positive emotions [18]. Following a potentially traumatic event, a certain number of patients will adopt avoidance behaviours after the event [19]. The resulting isolation prevents individuals from benefitting from available sources of social support and low social support is known to be an important risk-factor for depression [20]. However, to our knowledge, no reports have been published on the associative relationship between PTSD and ensuing depression in a post MI context.

Another reason supporting the relevance of the PTSD as an explanatory variable for the presence of depression in the months following the traumatic event is its part in the PTSD-depression comorbidity at the time of the trauma. Studies that have investigated the presence of PTSD-depression comorbidity in non cardiac context reported a prevalence varying from 12% to 14% [21,22]. Moreover, those studies demonstrated that patients with PTSD-depression comorbidity immediately after the trauma are more likely to have higher level of depression in the ensuing months. However, to what extent these results

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apply to the post MI context requires further investigation. We found only two studies reporting the prevalence of post MI PTSD-depression comorbidity, both at 8%, and only one investigated the impact of comorbidity on depression [12,23]. In this study, Ginzburg [12] reports that more than 20% of the patients with comorbidity 1 week after the MI, fulfilled the diagnostic criteria for depression 7 months later. However, this study measures symptoms of depression and PTSD only 1 week after the MI, incidentally not respecting the basic duration criteria for a diagnosis of depression and for PTSD.

Therefore, the current study prospectively investigates the relation between PTSD and depression among MI patients. Specifically, it assesses post MI patients 1 and 6 months after their coronary event in order to:

- 1- Assess the prevalence of comorbid symptomatology of PTSD and depression 1 month post MI;
- 2- Determine the impact of the 1 month post MI PTSD symptomatology on the intensity of depressive symptoms 6 months post MI;
- 3- Determine the impact of comorbid symptomatology of PTSD and depression 1 month post MI on the intensity of depressive symptoms 6 month post MI.

Such knowledge is fundamental as better recognition of PTSD may improve overall care for patients with cardiac conditions [24]. Moreover, the effects of comorbidity between PTSD and depression are still unclear [22] and need to be empirically validated in the specific post MI context [12].

Methods

Participants and procedure

Participants in this study are drawn from a larger study (refer to Roberge et al. [25], for complete details about participants, procedure and measurement instruments). The recruitment took place from June 2002 to April 2005 in three Montreal hospitals. In order to be part of the study, patients had to: 1) be 18 years old or older; 2) have sufficient oral and written language skills (English or French); 3) have a confirmed MI diagnosis base on troponin levels and electrocardiogram results and 4) have no diagnosis of any moderate-to-severe cognitive deficit nor any severe comorbid health problems (e.g.: cancer, AIDS).

Identification of eligible patients was achieved through the consultation of the medical files in the coronary units. Patients who met the criteria were then met by research assistant who presented the goals of the study and the research protocol. Participating patients had to sign a consent form to be part of the study. Patients who refused to participate were not asked additional questions as ethic committees prohibited it.

Before they left the hospital, sociodemographic and medical information were obtained from participants. Complementary medical information unknown to patients (e.g.: left ventricular ejection fraction) was gathered from the medical file. Additional questionnaires, to be returned 1 month after the MI, were given to participants along with a preaddressed prestamped envelope. Patients who did not return their questionnaires at due time were phoned daily until they completed and returned the questionnaires. Patients were contacted 6 months later to complete the same questionnaires, with the exception of the Life Events Stress Scale. Ethics Committees of the three institutions approved the research protocol.

Measurement instruments

Sociodemographic and medical data: This instrument collected participants' sociodemographic data (e.g.: age, sex, level of education) and different medical information (e.g.: consultation with a psychologist or psychiatrist, family history of coronary heart disease). The questionnaire was designed for the current study.

Life events stress scale (LESS) [3]: This inventory is a modified French and English version of the Life Events Stress Scale [26]. The adapted version is a list of ten potential traumatic event experienced in the last six months and their consequential level of stress on the patient. Psychometric properties are currently unknown.

Modified PTSD symptom scale-self-report (MPSS-SR) [27]: This 17-item questionnaire allows the patients to self-report the frequency and the severity of their posttraumatic stress symptoms, on a five-point Likert scale. The total score is obtained by adding the totals of the two scales. Its internal consistency is excellent (frequency, $\alpha=0.92$; severity, $\alpha=0.95$) as is its 5-week test-retest reliability (frequency, $r=.98$, $p<.001$; severity, $r=.98$, $p<.001$) [28]. It has been validated as a reliable measure of PTSD [28]. Caseness for the presence of PTSD symptomatology is determined by using a MPSS-SR cut-off of 22 [28].

Beck depression inventory, second edition (BDI-II) [29]: Based on the DSM-IV criteria, this 21-item self-administered questionnaire is commonly used to assess depression symptoms. Its internal consistency ($\alpha=0.92$) and 1 week test-retest reliability ($r=.93$) are excellent [30]. Caseness for depression symptomatology in the actual study is determined by using a BDI-II cut-off of 11 since it presents considerable sensitivity (100%) and specificity (75%) [31].

Statistical analyses: Descriptive analyses were carried out in order to establish participants' characteristics and the prevalence of symptomatology of both disorders (depression and PTSD) and their comorbidity.

In order to evaluate the impact of PTSD symptomatology and the PTSD-depression symptomatology comorbidity on the 6-month depression intensity, regression analysis was used. Univariate analysis, specifically correlations ($r>.40$) and t -tests ($p<.05$), were first conducted on clinically and theoretically important variables (e.g.: age, number of stressful event in the last 6 months) as whether or not they should be include in the regression model. The final model includes the following variables: age, sex, level of education, BDI-II and MPSS-SR scores as well as the interaction score of both measures. BDI-II scores were transformed using a logarithmic transformation to meet the assumptions specific to the regression and the interaction score was computed using the Z value of each result.

As secondary analysis, patients were grouped according to the presence/absence of symptomatology on both measures at 1 month, therefore creating four different groups (not clinical, depression, PTSD and comorbidity). One-way ANOVA and chi-squares analyses were conducted to respectively investigate any difference in the intensity of depression symptoms and in the proportion of patients with a subclinical level of depression between the four aforementioned groups.

Results

Participants

Out of the 1344 patients hospitalized during the recruitment period, 474 did not meet the inclusion criteria and 370 declined participation when invited to. On the 500 patients who consented to the study, 161

did not return their questionnaires or were unreachable for either the 1 or 6 months post MI follow-up, resulting in a final sample size of 339 participants, representing 39% of the eligible responders (see Figure 1 for details).

Other than age (mean age respectively of 58 versus 62; $t=2.89$; $p<.05$), there was no significant difference on the sociodemographic variables nor on PTSD and depression symptomatology between the

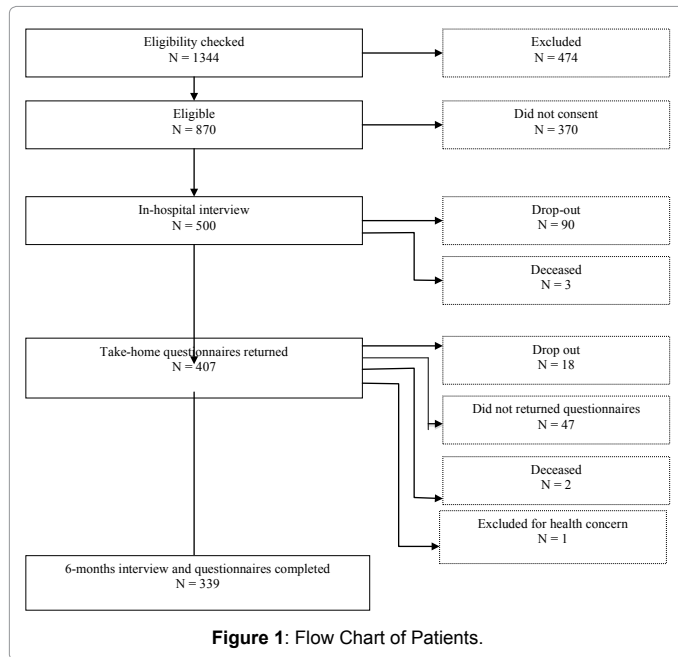


Figure 1: Flow Chart of Patients.

participants who completed the six months follow-up from those who did not. Characteristics of the participants who completed the follow-up are presented in Table 1.

Prevalence of PTSD-depression symptomatology comorbidity

Out of the 339 patients who completed the study, 39 met or exceeded the cut-off score for the presence of symptomatology on both the BDI-II and MPSS-SR at 1 month, thus a prevalence of PTSD-depression comorbidity of 11.5 %. Their average score on the MPSS-SR is 4.4 ($SD=18.7$) and 20.7 ($SD=8.6$) on the BDI-II.

Impact of the 1 month post MI PTSD symptomatology and the comorbid symptomatology on the 6 months post MI depression intensity

The results of the linear regression indicate that the variance of the intensity of depression 6 months post MI is significantly explained by the level of education, the intensity of depression symptom, the intensity of PTSD symptoms and the intensity of the comorbid symptomatology ($R^2=.469$, $F(6,332)=50.79$, $p<.001$). Complete results of the regression analysis are shown in Table 2.

Secondary analyses

Regarding the intensity of depression 6 months following the MI, although the one-way ANOVA ($F(3,336)=7.95$; $p=.005$) was significant, no statistical difference was found between the three different clinical groups. Specifically, although the comorbidity group reported the highest level of depression, it was not statistically different from the mean scores of the patients in the PTSD ($t(52)=1.320$, $p=.063$, $\eta^2=.06$) and depression ($t(79)=1.321$, $p=.190$, $\eta^2=.02$) groups.

Finally, a 2×4 chi-square was performed to compare the proportion

	Not clinical		Depression only		PTSD only		Comorbidity	
	(n = 243)		(n = 42)		(n = 15)		(n = 39)	
	N (%)	M (SD)	N (%)	M (SD)	N (%)	M (SD)	N (%)	M (SD)
Men	203 (83.5)		30 (71.4)		8 (53.3)		25 (64.1)	
Age		58.5 (10.7)		58.4 (10.3)		60 (10.8)		55.5 (11.3)
BMI		27.9 (4.8)		27.3 (4.6)		28.7 (8.5)		28.1 (7.3)
Education \geq college or equivalent	106 (43.6)		15 (35.7)		3 (20)		15 (38.5)	
Annual income < \$30,000	171 (73.4)		28 (66.7)		9 (60)		25 (64.1)	
Working	131 (53.9)		24 (57.2)		7 (46.6)		22 (56.4)	
Weekly drinking	124 (51)		16 (38.1)		4 (26.7)		14 (35.9)	
Smoking (before MI)	53 (21.8)		13 (31)		4 (26.7)		12 (30.8)	
Weekly engaging in physical activities	145 (59.7)		26 (61.9)		6 (40)		20 (51.3)	
History of psychological consultation	33 (13.6)		10 (23.8)		1 (6.7)		18 (46.2)	
Other important health issue	41 (16.9)		10 (23.8)		4 (26.7)		12 (30.8)	
History of MI	33 (13.6)		3 (7.1)		0 (0)		7 (17.9)	
History of CAD (other than MI)	175 (72)		36 (85.7)		10 (66.7)		31 (79.5)	
Past PTSD	2 (.8)		2 (4.8)		1 (6.7)		2 (5.1)	
Family history of CAD	173 (71.2)		31 (73.8)		10 (66.7)		27 (69.2)	
Number of stressful life events in the past 6 months		1.4 (1.3)		1.9 (1.4)		1.5 (1.2)		2.8 (.9)
Ejection fraction (%)		49 (11)		46 (14)		52 (11)		48 (13)

Table 1: Participants' characteristics.

(n = 339)	β	p	95% CI		Partial R	Global R	Adjusted R ²
			Lower Bound	Upper Bound			
Variables						.69	.47
Age	.035	.385	-0.030	0.079	.048		
Sex	.034	.400	-0.816	2.040	.046		
Level of education	-.103	.011	-2.722	-0.362	-.140		
Intensity of depression symptoms at 1 month	.472	.000	4.189	6.765	.417		
Intensity of PTSD symptoms at 1 month	.177	.014	0.018	0.158	.134		
Intensity of comorbidity (Depression X PTSD)	.140	.013	0.093	0.772	.136		

Table 2: Summary of linear regression analysis for the identification of the variables associated with the intensity of 6 months post MI depression symptoms on the BDI-II.

	<i>Not clinical</i>	<i>Depression only</i>	<i>PTSD only</i>	<i>Comorbidity</i>
	(n=243)	(n=42)	(n=15)	(n=39)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
1 month				
Social support	21.9 (6.5)	19.6 (7.8)	24.5 (3.6)	18.9 (6.8)
PTSD intensity	6 (5.9)	14.8 (5.7)	30.8 (10.8)	42.4 (18.7)
Depression intensity	4.1 (2.8)	14.6 (3.4)	7.4 (2.1)	20.7 (8.6)
6 months				
Depression intensity	4.2 (4.4)	12.7 (7.1)	9.7 (5.8)	15.4 (11)
Presence of depression symptomatology (n - %)	23 (9.5%)	23 (54.8%)	5 (33.3%)	23 (59%)

Table 3: Participant's scores on psychological measures.

of subclinical depression in each group. Consequent to the result ($\chi^2(3, N=339)=81.16, p<.001$), a series of 2 X 2 chi-square were calculated to assess the difference between the 4 groups. The results indicate that the comorbidity ($\chi^2(1, N=282)=60.34, p<.001$), depression ($\chi^2(1, N=285)=54.29, p<.001$) and PTSD ($\chi^2(1, N=258)=8.32, p=.004$) groups had a significantly greater proportion of patients with depression symptomatology at 6 months than the not clinical condition. No other comparison was statistically significant. Proportions are presented in Table 3.

Discussion

Based on the presence of symptomatology, data from the actual study indicates 11.5% prevalence of symptomatology comorbidity between PTSD and depression 1 month after myocardial infarction. It is similar to the prevalence reported by Ginzburg, also obtained in a post MI context [12]. However, these numbers are lower than those reported in following non-medical trauma such as war refugees (53%), terrorist attacks (38%) or natural disasters (28%) [32-34]. We believe this discrepancy can be primarily explained by two reasons. First, only 42% of the recruited participants perceived their MI as a traumatic event, either because they didn't think they were having an MI at the time or because they did not consider MI as a life threatening event. Second, although the choice of 1 month post MI was theoretically sound for the measure of PTSD, it may have been too early for the disorder to develop. In the actual study, 45% of the PTSD cases were developed after the first month following the MI, thus diminishing the reported prevalence. This percentage of delayed onset PTSD is similar to the one reported in a recent study on injury survivors, where 47% of the PTSD cases were of delayed onset [35].

Impact of PTSD

The results of the regression analysis clearly demonstrate that the intensity of depression symptomatology at 6 months is best predicted

by the level of depression symptomatology at 1 month. Nonetheless, even with this predictor already in the equation, the level of PTSD intensity at 1 month is a significant predictor of the dependant variable, suggesting that the late development of depression symptomatology can be the result of the specific symptoms related to the PTSD. Our other results support this idea, since 1 in 3 patients with non comorbid PTSD symptomatology at 1 month went on to develop depression symptomatology 6 months post-MI. These patients also reported a level of depression equivalent at 6 months similar to patients who already met the depression symptomatology cut-off at 1 month.

These results support the hypothesis that PTSD is a predisposing condition for the development of depression. Since PTSD and depression share alterations in common biological pathways [14], the impact of PTSD on the brain's normal functioning is one plausible explanation for this facilitating effect of PTSD on depression. Research led by Armony et al. suggests that functional abnormalities in brain responses to emotional stimuli are observed in acute phases of PTSD [36]. Specifically, they observed that the presence of PTSD was associated with a reduced capacity of the amygdala to respond during emotion recognition tasks. As impaired functioning in emotional tasks involving the amygdala has also been observed in depression [37], it is possible that PTSD can facilitate the onset of depression through its effect on the amygdala. However, further researches are needed to clarify the relationship between the functioning of the amygdala and the two disorders.

Another possible explanation concerns the avoidance behaviours exhibited by patients with PTSD. Researchers have demonstrated that patients with PTSD who displayed a high level of avoidance behaviours also reported more social isolation and high level of depression after their MI [38,39]. Although participants in the current study reported adequate level of available social support, we believe that the avoidance behaviours resulting from the traumatic MI may have led patients to

isolate themselves and not use the available social support. People who had suffered a cardiovascular accident often experience changes in their sense of self as well as changes in their professional, home, and community roles that may lead to social isolation [40]. This resulting social isolation, in an effort to evade any discussion or recall of their recent MI, may have facilitated or provoked the development of depressive feeling for the patients, as reported by Hinojosa and his collaborators [41]. This idea finds support in our results, since the correlation between the avoidance behaviours and depression in the actual study is significant (Pearson's $r=0.57$; $p<0.001$). This matter is particularly important, since socially isolated people are at increased risk for myocardial infarction and mortality compared with people who are more socially integrated [42].

Impact of comorbidity

The regression analysis result indicates that the comorbidity of both PTSD and depression symptoms adds a statistically significant, though modest, contribution to the prediction of the development of depression symptomatology 6 months post MI. Since the inclusion of the comorbidity in the regression model adds some explained variance, this leads us to two considerations: 1 - the detection of this particular comorbidity should follow a thorough process, since it is frequently underestimate and complex to treat [43,44] and 2 - this condition should be regarded as a problematic on its own and be treated consequently. On the other hand, although they reported a higher level of depression at 6 months compared to subjects with either significant depressive or PTSD symptoms at the 1 month evaluation, patients with comorbid conditions did not present statistically superior scores compared to the other clinical groups. This absence of expected difference may result from the lack of power consequent to a small number of patients with both PTSD and depression. Nonetheless, the comorbid patients presented high levels of depression, comparable with those found in the literature [12,39]. Comorbid PTSD-depression symptomatology may have led to high levels of depression 6 months post-MI as a result of negative feedback between both disorders (e.g.: lack of sleep brings irritability which leads to isolation). This interaction can prevent or slow down the rehabilitation process and facilitate either the maintenance or the onset of depression. On the other hand, the analogous level of depression at 6 months by depressed and comorbid patients at 1 month imply that the presence of PTSD does not significantly add to the burden of already depressed patients. The mixed findings about the comorbidity suggest that it has some clinical significance, though limited.

Methodological limitations

The large number of participants recruited and the clinically-oriented relationship explored between PTSD and depression were strengths of this prospective multicenter study. However, several considerations limit the potential generalisation of the results. Although five hundred patients consented to participate, we have incomplete data for about a third of them (161 out 500; 32.2%). The direct consequence of this is the small number of patients in the different clinical conditions explored, particularly the PTSD only group ($n = 15$). This raises legitimate questions about the representativeness of our sample. Although self-reported measures and their respective cut-off scores enable researchers to detect subclinical level of depression and PTSD, they don't tell the story from a clinical standpoint. The use of a clinical interview based on the DSM-IV-TR criteria could have led to different conclusion and insight regarding the effect of early depression and PTSD on the 6 month post MI depression. In addition, our final sample did not have an equivalent number of men and women, although it is

proportional to the population it was drawn from. Since epidemiological studies report higher prevalence of PTSD for women than men, future studies should address this recruitment bias for statistical consideration and generalization purpose.

Recommendations

The results of the present study suggest that the assessment of PTSD and its comorbidity with depression following a MI should be reviewed. First, it should be part of a routine post MI psychological investigation. Cardiologists are not, in general, trained to screen patients for psychiatric conditions and the impact of anxiety and depression on cardiovascular health is not a routine discussion in many physician-patient encounters [2]. Second, as PTSD can be mistaken for depression and both disorders call for specific psychological and pharmacological treatments, a more comprehensive approach should be used when screening for PTSD. Although the use of short and accurate screening instrument for PTSD by first line nurses would be a beneficial addition to PTSD detection, a sound clinical approach to detect PTSD symptoms should also implicate in-hospital mental health specialists or adequately trained physician in the process. Involving such specialized resources could also ensure knowledge transfer to the rest of the medical staff. Consequently, an improvement in PTSD detection may likely result in a diminution of the harmful outcomes for post MI patients with post traumatic stress symptomatology.

Conclusion

This study investigated the relation between PTSD and depression in a post MI context. The 11.5 % prevalence of comorbidity observed at 1 month indicates that the PTSD-depression symptomatology comorbidity is a frequent occurrence and further investigations are needed to fully address its development. Our results suggest that in order to facilitate the rehabilitation of MI patients, PTSD and depression should be assessed and promptly treated when needed. Future studies should evaluate more specifically the avoidance behaviours associated with PTSD and their relation to social support, since high levels of support may protect patients from the negative prognostic consequences of depression [45]. Also, to what extent the protective factors identified for PTSD is valid in a post MI context should be addressed by future research. This would help better manage the health care system's resources by targeting patients requiring immediate attention.

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