

# An Epidemiologic Study of Depressive Symptoms among Cardiometabolic Department Patients in México

—Depression in Cardiometabolic Patients

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Received December 28<sup>th</sup>, 2010; revised April 6<sup>th</sup>, 2011; accepted April 19<sup>th</sup>, 2011.

## ABSTRACT

**Background:** This study estimated the prevalence of depressive symptoms among cardiometabolic department patients in México. **Methods:** To identify patients with depressive symptoms, we used the Beck's Depression Inventory (BDI). We analyzed data from consecutive adult patients who attended during a year to a Cardiometabolic Department in México and described the demographic, metabolic and vascular status differences between depressive and non-depressive patients. The estimates are based on a total of 180 patients aged 22 to 83 years. **Results:** There was a depressive symptoms prevalence rate of 60.5%. Compared with non-depressive patients, depressive patients were more likely to be obese, and to have dysglucemia, hypercholesterolemia, hypoalphalipoproteinemia, microalbuminuria, high uric acid levels, carotid atherosclerosis, insulin resistance and metabolic syndrome. **Conclusions:** Our data suggest that prevalence of depression is elevated among cardiometabolic patients in México. Depression probably plays a role in cardiometabolic physiopathogenic, and must be intentionally assessed in cardiometabolic patients in order to treat it and to improve the cardiometabolic treatment response and adherence.

**Keywords:** Cardiometabolic, Depression, Prevalence

## 1. Introduction

Depression is a common condition in México [1] with a prevalence of 5.8% in a national survey; it is more frequent in women compared with men, 7.8% vs 2.5% [2]. However its prevalence is higher in diabetic patients which more than 30% shows this condition [3]. Higher prevalence of depression has been reported in patients with metabolic syndrome which 43.6% shows depressive symptoms [4]. In spite of the above mentioned, in Mexico depression is not looked for in a deliberate way at the cardiometabolic departments and we ignore which is the frequency of depression like co morbidity in patients with cardiometabolic diseases. This study addresses prevalence rates and associated correlates of depressive symptoms in a cardiometabolic department.

We hypothesized that depression would be common in the cardiometabolic settings in México, and that it would associate significantly with metabolic and vascular co

morbidities.

## 2. Methods

### 2.1. Study Design

This was a cross-sectional study conducted in a Cardiometabolic Department in Mexico in a university-affiliated clinic. The institutional review boards approved the study, and written informed consent was obtained from all patients.

### 2.2. Selection of Participants

Consecutive 180 patients who were treated in a Cardiometabolic Department during a year were enrolled. Inclusion criteria were being aged 18 years or older, compliant with the standard procedures of the cardiometabolic department and having the ability to response the Beck Depression Inventory (BDI).

### 2.3. Data Collection

Professional trained physicians assessed patients' demographic and physical characteristics (age, sex, weight, height, body mass index, abdominal perimeter, and blood pressure). To examine the frequency of depressive symptoms in the past week, the 21 items BDI in a validated Spanish version was included [5]. The BDI distinguishes depressed from non-depressed patients [6]. The standard cut-off point is 10, with scores at or above that value suggesting current depression. This cut-off score was shown to have a sensitivity of 94% and specificity of 92% for detecting depression in primary care settings [7]. Depression was classified in three grades: 10 - 18 points = mild depression, 19 - 29 points = moderate depression,  $\geq 30$  points = severe depression. The BDI was carried out by a trained physician who helps patients to respond the inventory. Fast glycaemia, two-hour postprandial glycaemia, fasting insulin, total cholesterol (TC), triglycerides (TG), high density lipoprotein-cholesterol (HDL-C), urea, creatinina, uric acid, urine analysis, microalbuminuria and haematological tests were carried out by an external certified laboratory. To assess insulin resistance (IR) the homeostasis method assessment (HOMA) was carried out [8]. Low-density lipoprotein cholesterol (LDL-C) was estimated by Friedewald's formula [9]. To assess

atherosclerosis and vasomotor endothelial function, carotid intima-media thickness and brachial flow dependent vasodilation were measured by means of high resolution Doppler ultrasound in agreement with standard international guidelines [10,11]. Ultrasound evaluations were made in an external image diagnostic centre by one certified radiologist.

### 2.4. Primary Data Analysis

All analyses were performed using SAS, Inc (Cary, NC). Data are presented as proportions and means (with standard deviation). Student's *t* test, Kruskal-Wallis,  $\chi^2$ , and Fisher's exact tests were used, as appropriate. A *p*-value of  $< 0.05$  was considered statistically significant. Variables associated with depression were evaluated by means of prevalence odds ratios (OR) and are presented with 95% confidence intervals (CI).

### 3. Results

One hundred and eighty patients were evaluated. Among these patients 109 showed depressive symptoms (60.5%), 62 (56.8%) had mild, 38 (34.8%) moderate and 9 (8.2%) severe depression. **Table 1** shows that there were a higher proportion of women among depressed patients, and also they had higher values of carotid intima-media and non-

**Table 1. Features of depressive versus non-depressive patients from a cardiometabolic department.**

Feature	Depressed ( <i>n</i> = 109)	Non-depressed ( <i>n</i> = 71)
Age (years)	53.9 $\pm$ 12.2	54.09 $\pm$ 12.2
Female <i>n</i> and (%)	78 (71.6)*	48 (67.7)
Weight (kg)	71.7 $\pm$ 12.9	72.8 $\pm$ 17.3
Height (m)	1.57 $\pm$ 0.08	1.57 $\pm$ 0.09
Body mass index	29.1 $\pm$ 4.8	29.3 $\pm$ 5.8
Waist perimeter (cm)	95.5 $\pm$ 11.1	94.1 $\pm$ 13.4
Fasting glycaemia (mg/dl)	117.2 $\pm$ 51.5	112.5 $\pm$ 48.3
Fasting insulin ( $\mu$ U/ml)	12.1 $\pm$ 6.8	12.4 $\pm$ 9.1
HOMA	3.4 $\pm$ 2.6	3.5 $\pm$ 3.1
Total cholesterol (mg/dl)	194.5 $\pm$ 47.07	195.8 $\pm$ 41.04
Triglycerides (mg/dl)	172.03 $\pm$ 103.08	170.8 $\pm$ 114.6
LDL-cholesterol (mg/dl)	111.9 $\pm$ 39.6	112.09 $\pm$ 35.1
HDL-cholesterol (mg/dl)	48.1 $\pm$ 11.8	49.5 $\pm$ 12.6
Urinary albumin excretion (mcg/creatinina mg)	16.10 $\pm$ 13.7	18.06 $\pm$ 27.5
Uric acid (mg/dl)	4.5 $\pm$ 1.3	5.3 $\pm$ 4.6
Leucocytes (thousands/ml)	6420.19 $\pm$ 1485	6827.87 $\pm$ 1716
Lymphocytes (%)	33.5 $\pm$ 7.9	33.004 $\pm$ 7.9
Lymphocytes (total)	2136 $\pm$ 654.6	2217.11 $\pm$ 647.7
SBP (mm Hg)	130.7 $\pm$ 19.3	134.5 $\pm$ 16.7
DBP (mm Hg)	76.5 $\pm$ 10.1	77.15 $\pm$ 10.7
Metabolic Syndrome components	2.8 $\pm$ 1.3	2.5 $\pm$ 1.2
Flow-dependent vasodilation (%)	15.8 $\pm$ 10.3	16.7 $\pm$ 21.5
Carotid intima-media thickness (mm)	0.60 $\pm$ 0.22*	0.53 $\pm$ 0.18
BDI	18.2 $\pm$ 7.4**	5.5 $\pm$ 3.01

\* = *p* < 0.05, \*\* = *p* < 0.0001

**Table 2. BDI score among depressed and non-depressed patients.**

Item	Depressed ( <i>n</i> = 109)	Non-depressed ( <i>n</i> = 71)
1. Sadness	1.165 ± 0.79*	0.324 ± 0.47
2. Pessimism	0.697 ± 0.87*	0.042 ± 0.20
3. Past failure	0.817 ± 1.029*	0.07 ± 0.425
4. Loss of pleasure	0.807 ± 0.79*	0.155 ± 0.43
5. Guilty feelings	0.596 ± 0.79*	0.056 ± 0.28
6. Punishment feelings	0.817 ± 0.76*	0.042 ± 0.20
7. Self-dislike	0.615 ± 0.65*	0.07 ± 0.25
8. Self-criticalness	0.853 ± 0.92*	0.197 ± 0.49
9. Suicidal thoughts or wishes	0.349 ± 0.67*	0.0 ± 0.0
10. Crying	0.881 ± 1.08*	0.141 ± 0.48
11. Irritability	1.21 ± 0.98*	0.57 ± 1.09
12. Social isolation	0.541 ± 0.86*	0.085 ± 0.28
13. Indecisiveness	0.661 ± 0.88*	0.014 ± 0.11
14. Self-image	1.064 ± 0.94*	0.225 ± 0.614
15. Loss energy	0.853 ± 0.54*	0.324 ± 0.501
16. Changes in sleeping pattern	1.679 ± 1.05*	0.859 ± 1.09
17. Tiredness or fatigue	0.89 ± 0.56*	0.451 ± 0.50
18. Changes in appetite	0.468 ± 0.7*	0.113 ± 0.31
19. Loss of weight	0.615 ± 0.92	0.423 ± 0.78
20. Hypochondria	1.725 ± 1.26*	0.958 ± 1.33
21. Loss of interest in sex	1.22 ± 1.18*	0.53 ± 0.82

\*  $p < 0.01$  vs non-depressed.

depressed patients, items 20 (hypochondria), 16 (sleep disturbances), 21 (decreased libido), 11 (irritability), 1 (sadness) and 14 (deterioration of self-image) had the higher scores among depressed patients; while items 9 (suicidal tendency), 18 (changes in appetite) and 5 (guilty feelings) showed the lowest scores. Only item 19 (loss of weight) did not discriminate between depressive and non-depressive subjects. **Table 3** shows that depressed patients had higher frequency of obesity, disglucemia, hypercholesterolemia, hypoalipoproteinemia, microalbuminuria, high uric acid levels, carotid atherosclerosis, insulin resistance and metabolic syndrome; but less proportion of hypertriglyceridemia and endothelial dysfunction than non-depressed patients. There were no differences in prevalence of neither diabetes nor hypertension among depressed and non-depressed subjects. **Table 4** illustrates the OR and CI from the clinical, metabolic and vascular variables related to depression prevalence in our patients; it did not show any significant association between them.

#### 4. Discussion

These data show that a substantial proportion of patients

in a Cardiometabolic Department in México had positive symptoms for depression. The prevalence of depressive symptoms as reported by cardiometabolic patients was 60.5%, higher than reported by Mexican national survey (3.3%) and by Dumbar (10%) [12], Hildrum (6.9%) [13] and by our group (46.3%) in patients with metabolic syndrome. Our results not only demonstrate that the prevalence of depressive symptoms is elevated among cardiometabolic patients, but it also identifies several factors with higher prevalence among patients with depression.

Our trial, in agreement with prior epidemiological studies, indicates that depressive symptoms are more frequent in the female sex even in general population as in Mexican patients with metabolic syndrome [4]. As previously reported [14] carotid atherosclerosis measured by means of intima-media thickness, a marker of subclinical vascular damage was positively associated with depression, we founded a light but significantly higher frequency of carotid atherosclerosis among depressed patients. Although we did not found a statistically significant association between depression and cardiometabolic features, there were a higher frequency of markers and compounds of

**Table 3. Cardiometabolic alterations of depressive versus non-depressive patients from a cardiometabolic department, n (%).**

Disturbance	Depressed	Non depressed
Obesity and overweight	90 (82.5)*	54 (77.1)
Abdominal obesity	97(88.9)*	53 (75.7)
Fasting hyperglycaemia	57 (52.2)*	26 (37.1)
Insulin resistance	57 (55.8)*	31 (46.9)
Hyper TC ( $\geq 200$ )	46 (42.2)*	28 (40)
Hyper TG ( $\geq 150$ )	48 (44.03)	37 (52.8)**
Hyper C-LDL <sup>+</sup>	64 (58.7)*	32 (45.7)
Hypoalphalipoproteinemia	31 (28.4)*	13 (18.5)
Microalbuminuria (30 - 300)	7 (7.7)*	3 (5.3)
Hyperuricemia ( $\geq 7$ )	6 (6.1)*	2 (3.1)
Diabetes mellitus	29 (26.6)	19 (26.7)
Hypertension	55 (50.4)	37 (52.1)
Metabolic syndrome	63 (57.7)*	34 (47.8)
Endothelial Dysfunction	28 (27.7)	19 (32.2)**
Carotid Atherosclerosis	20 (19.8)*	7 (11.8)

\*  $p < 0.05$  vs non-depressed; \*\* $p < 0.05$  vs depressed; + in agreement with NCEP goals.

**Table 4. Prevalence odds ratios of measured variables related to depression symptoms among cardiometabolic department patients.**

Variable	Odds ratio	95% confidence interval
Sex	0.82	-2.147 - 1.773
Overweight	1.40	-1.62 1 - 2.299
Abdominal obesity	2.59	-1.007 - 2.912
Fasting disglucemia	1.85	-1.342 - 2.577
Insulin resistance	0.35	-1.60 - 2.317
Hyper TC	1.09	-1.86 - 2.051
Hyper TG	0.701	-2.31 - 1.60
Hyper LDL-C	1.68	-1.43 - 2.48
Hypoalphalipoproteinemia	1.74	-1.40 - 2.51
Microalbuminuria	1.48	-1.56 - 2.35
Hyperuricemia	2.02	-1.25 - 2.66
Elevated SBP	0.66	-2.32 - 1.54
Elevated DBP	0.998	-1.96 - 1.95
Diabetes	0.992	-1.96 - 1.95
Hypertension	0.935	-2.02 - 1.89
Endothelial dysfunction	0.80	-2.1 - 1.74
Carotid atherosclerosis	1.83	-1.35 - 2.56
Metabolic syndrome	1.49	-1.56 - 2.39

insulin resistance metabolic syndrome in patients with depression symptoms compared with non-depressed subjects. Most of them are associated with obesity as a pathogenic common basis. Obesity has been proved as a risk factor in the depressive patients, in a longitudinal study [15] at age 70 years, 35% of participants (6820 men and 3346 women) who had common mental health disorders (anxiety and depression), were obese compared with 27% of those without mental disorders (odds ratio, 1.46). There are several explanations for addressing the relationship between depression and cardiometabolic disturbances, one is the activation of hypothalamic-pituitary-adreno-cortical (HPA) axis which has been consistently documented to be hyperactive in depressed patients, with elevated corticotrophin-releasing factor (CRF) in cerebrospinal fluid, decreased adrenocorticotrophic hormone (ACTH) response to CRF challenge, nonsuppression of cortisol secretion in response to dexamethasone, hypercortisolemia, and pituitary and adrenal gland enlargement [16-18]. Sympatoadrenal hyperactivity has also been demonstrated in depressed patients [19]. Cortisol and catecholamines may account for central obesity, blood pressure elevation, hyperglucemia and dislipidaemia in depressed patients and they may play a significant role in the effect of depression on the development and prognosis of cardiovascular disease [20].

Central obesity is a systemic inflammatory state in which there is release of cytokines as interleukin 6 (IL-6) and tissue necrosis factor alpha (TNF- $\alpha$ ). Inflammation might actually cause depression by means of local (Thomas 2000) or systemic effects (Leonard 2001). Although it seems likely that inflammation impacts the progression of cardiovascular disease, it remains unclear whether the inflammation seen in depressed patients is a result of the stress response or whether inflammation contributes to the pathogenesis of depression.

## 5. Conclusions

Depression symptoms have high prevalence in cardiometabolic patients in México. Depressed patients have higher prevalence of several metabolic and vascular disturbances related to metabolic syndrome. The elevated frequency of metabolic and subclinical vascular changes in adults with symptoms of depression, stressed the need for considering depression in risk factor profiling.

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