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ORIGINAL ARTICLE

Differences in the cluster of depressive symptoms between subjects with type 2 diabetes and individuals with a major depressive disorder and without diabetes

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Abstract

Background Depressive disorder encompasses a wide spectrum of somatic and psychological symptoms. It is not known whether there are differences regarding the cluster of depressive symptomatology between subjects with depression with and without T2DM.

Purpose To explore whether the cluster of depression that prevails among depressive subjects with T2DM differs from individuals with depression, but without T2DM.

Methods 87 T2DM patients with a pathological Beck Depression Inventory test (BDI) were compared with 50 age- and gender-matched individuals with a major depressive disorder. All 21 items expressed in the BDI were compared between the two groups.

Results The score obtained after administering the BDI was comparable between patients with T2DM and significant depressive symptoms and the control group (18.8 ± 2.7 vs 18.9 ± 3.4; $p = 0.9$). Subjects with T2DM had higher scores compared with the control group in the following items: sadness (1.4 ± 0.9 vs 0.9 ± 0.9; $p = 0.011$), difficulty in concentration (1.3 ± 0.8 vs 0.8 ± 0.8; $p = 0.01$), indecisiveness (1.1 ± 0.8 vs 0.5 ± 0.9; $p = 0.012$), worries about their health (1.3 ± 0.9 vs 0.6 ± 0.9; $p < 0.0001$), fatigue (1.2 ± 0.6 vs 0.8 ± 0.7; $p = 0.003$) and loss of sexual appetite (2.7 ± 0.6 vs 1.2 ± 1.3; $p = 0.0001$). Suicidal ideation was significantly lower among subjects with T2DM compared with the control group (0.1 ± 0.3 vs 0.6 ± 0.8; $p = 0.0001$).

Conclusions Subjects with T2DM and a positive screening for depression presented a different cluster of depression compared with depressed subjects without T2DM, with a predominance of somatic–biological depressive symptoms rather than psychological–cognitive cluster and negative emotions, such as suicidal ideation.

Keywords Type 2 diabetes · Depression · Depressive symptoms · Somatic–biological depressive symptoms

Introduction

Type 2 diabetes (T2DM) and depressive disorder are highly prevalent chronic diseases in developed countries [1, 2]. Interestingly, both disorders are related to each other and recent research demonstrates that the prevalence of depression among adults with T2DM is approximately double that observed in the general population [3]. Prevalence rates of depressive disorder among people with T2DM range between 11 and 30% [4–9]. The detrimental effects of, not only depressive disorder, but also significant depressive symptoms in people with T2DM can be observed in an increased risk of developing chronic complications, a reduced quality of life, poorer self-care behavior, greater

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health-care expenditure and even an increased mortality [9–14].

In contrast, the influence of depressive disorder on glycemic control is still controversial. While some authors have found an association between depressive symptoms and glycated hemoglobin (HbA1c), others could not prove it [15–17].

Moreover, intervention studies with antidepressant therapy aiming to diminish depressive symptoms in people with T2DM have failed to demonstrate a significant improvement in glycemic control, although there was an amelioration of depressive symptomatology and heterogeneity of depression [18–23].

According to DSM-5, the diagnostic criteria for a major depressive disorder consist of a core symptom, either in a diminished/irritable mood or a decreased interest/pleasure (anhedonia) or both, and at least four of the following symptoms: feelings of guilt or worthlessness, fatigue or loss of energy, concentration problems, suicidal thoughts or thoughts about death, changes in weight (5% weight loss or weight gain), psychomotor retardation or activation (change in activity) and/or hypersomnia or insomnia (changes in sleep), lasting for at least 2 weeks [24].

Therefore, there is a wide spectrum of somatic and affective symptoms. This fact implies that a huge variety of symptom profiles are summarized under the same depression diagnosis. These diverse clusters of depressive symptoms could explain differences in antidepressant treatment response [25, 26].

Furthermore, to our knowledge, no studies regarding differences in the depressive symptoms profile between patients with T2DM and subjects with a depressive disorder, but without a T2DM have been reported so far.

In this setting, our study was aimed at exploring whether there were differences between the profile of depressive symptoms in patients with T2DM compared with subjects with a diagnosis of depression and without T2DM. We also wanted to know whether there was a predominance of somatic or affective symptoms among patients with T2DM.

Subjects

In a prior study, 320 subjects with a diagnosis of T2DM were recruited consecutively, using serial selection from 14 primary care settings and the outpatient's clinic of an Endocrine Department from a tertiary center in Palma de Mallorca (Balearic Islands, Spain). Both sources constitute a sanitary area that attends both rural and urban areas with a total population of 250,000 inhabitants. Patients were excluded from the study if they had type 1 or gestational diabetes, were individuals with a diagnosis of T2DM in the preceding 12 months, were patients with T2DM in pregnancy or lactation, had alcohol or substance dependence, were individuals who had an uncontrolled medical condition other than diabetes, had a previous diagnosis of depressive disorder or were currently treated with antidepressant therapy. To rule out depression, a Beck Depression Inventory (BDI) was administered to these 320 subjects, and 87 individuals (27.2%) had a positive screening for depressive disorder [9].

The T2DM patients with a pathological BDI were compared with 50 individuals with a diagnosis of major depressive disorder, recruited consecutively from the outpatient's clinic of the psychiatry department. Exclusion criteria for this group of patients were having another psychiatric condition other than depression (schizophrenia, bipolar disorder or suicide attempt) or any other serious medical illness.

Written informed consent was obtained for both groups prior to study participation. The study was approved by the Ethics Committee of the Hospital Son Llàtzer.

Materials and methods

Assessment of depressive disorder

To investigate the presence and the degree of depressive syndrome, all participating patients rated the presence and severity of depressive symptoms using the Spanish version of the Beck Depression Inventory (BDI), a 21-item questionnaire that assesses mood over the previous month. Total scores range from 0 to 63, with higher scores indicating greater symptoms of depression. The Beck Depression Inventory has been widely used as a screening tool for major depression in the general population. In this setting, a cutoff score equal or greater than 13 is suspicious for significant depression. The BDI has been reported to be highly reliable regardless of the population. It has a high coefficient alpha (0.80), its construct validity has been established, and it is able to differentiate between depressed from non-depressed patients. In patients with a known depressive disorder, BDI has been used to assess the severity of symptoms of depression as well as the response to the antidepressant therapy. In people with a depressive disorder, a total score of 0–13 is considered a minimal range, 14–19 is mild, 20–28 is moderate, and 29–63 is severe [27]. However, among people with diabetes, a cutoff score equal to or greater than 16 for the entire 21-item measure exhibited the best balance between sensitivity and positive predictive value. This cutoff would be able to capture more than 70% of the subjects diagnosed with major depression, yet provide more than 70% of certainty that a person screening positive actually has this condition [28]. Moreover, as scores in every item of the BDI were between 0 and 3, we considered as a relevant result a score equal TO or greater than 2.

Socio-demographic and clinical parameters

Demographic information was gathered during the eligibility determination which included age, gender, race, marital status, education level, employment situation and diabetes duration. Height and weight were measured while each participant was wearing indoor clothing, without shoes. BMI was calculated as weight divided by height squared. Waist circumference was measured at the midpoint between the lowest rib and the iliac crest.

Statistical analyses

Initial statistical plan for the control group was established using a control-to-case ratio of 1:2 and estimating that 30% of our T2DM sample would have a pathological BDI. But, as BDI was only positive for depression in 87 subjects with T2DM (27.2%), we could not perform an age- and gender-matched analysis. However, the two groups were age and gender comparable.

Initial analyses were descriptive and included calculation of mean, median, and standard deviation (SD) for continuous variables and frequencies for categorical variables. The distribution of the sample was analyzed by the Kolmogorov–Smirnoff test. Comparison between the TWO groups was analyzed by a Mann–Whitney *U* test. A p value < 0.05 on the TWO-tail test was considered to indicate statistical significance. Also, crude and age-, sex- and BMI-adjusted odds ratio (OR) of having T2DM using relevant psychological and cognitive depressive symptoms and negative or somatic/biological symptoms as dependent variables was performed. Data were analyzed using SPSS v.16 statistical software (SPSS Inc., Chicago, IL, USA).

Results

The score obtained after administering the BDI was comparable between patients with T2DM and significant depressive symptoms and the control group (18.8 ± 2.7 vs 18.9 ± 3.4; $p = 0.5$).

When taking into account the 87 T2DM subjects with a pathological BDI, 58% of these patients were female with a mean age of 45.9 ± 17.6 years. Mean T2DM duration among these patients was 12.8 ± 10.2 years and mean HbA1c was 7.9 ± 1.8%.

T2DM group was age and gender comparable with the control group. Also, employment situation, educational level and marital status were also comparable between the two groups. However, BMI at the time of the evaluation was significantly higher among subjects with T2DM compared with the control group (31.9 ± 5.9 vs 27.3 ± 4.1 kg/m^2; $p = 0.01$). Waist circumference was also greater among T2DM individuals with a pathological BDI compared with subjects included as controls (105 ± 10 *vs* 97 ± 8 cm; $p = 0.01$). Demographic and clinical data of both groups are summarized in Table 1.

However, there were significant differences between the two groups regarding the scores obtained in the different compounds of the BDI. Subjects with T2DM had higher scores compared with the control group in the following items: Sadness (1.4 ± 0.9 vs 0.9 ± 0.9; $p = 0.011$), indecisiveness (1.1 ± 0.8 vs 0.5 ± 0.9; $p = 0.012$), difficulty in concentration (1.3 ± 0.8 vs 0.8 ± 0.8; $p = 0.01$), worries about their health (1.3 ± 0.9 vs 0.6 ± 0.9; $p < 0.0001$), fatigue (1.2 ± 0.6 vs 0.8 ± 0.7; $p = 0.003$) and loss of sexual appetite (2.7 ± 0.6 vs 1.2 ± 1.3; $p = 0.0001$).

Table 1 Demographic and clinical parameters of both groups

	T2DM subjects with a BDI ≥ 16 ($n = 87$)	Subjects with a MDD ($n = 50$)	p
Gender (male/female)	42/58	42/58	NS
Age (years)	45.9 ± 17.6	44.7 ± 16.8	NS
Educational level (illiterate/primary/superior)	4/71/25	3/73/24	NS
Employment situation (active/non active)	32/68	34/66	NS
Marital status (single/couple)	38/62	35/65	NS
BDI	18.8 ± 2.7	18.9 ± 3.4	NS
T2DM duration (years)	12.8 ± 10.2	–	–
BMI (kg/m^2)	31.9 ± 5.9	27.3 ± 4.1	0.01
Waist circumference (cm)	105 ± 10	97 ± 8	0.01
HbA1c (%)	7.9 ± 1.8	5.7 ± 0.4	0.001
Antidepressant therapy (SSRIs/MAOIs/None)	4/0/96	92/8/0	0.0001
Diabetes treatment (OAs/insulin)	69/31	–	–

Data are mean ± SD or %

T2DM type 2 diabetes, *BDI* Beck Depression Inventory, *BMI* body mass index, *GFR* glomerular filtration rate, *SSRIs* serotonin reuptake inhibitors, *MAOIs* monoamine oxidase inhibitors, *OAs* oral agents

On the other hand, patients with T2DM and significant depressive symptoms reported lower scores compared with individuals with a major depressive disorder in the items: past failure (0.4 ± 0.8 vs 0.9 ± 0.9; $p = 0.001$), guilty feelings (0.5 ± 0.8 vs 1.2 ± 1.1; $p = 0.002$), self-criticalness (0.4 ± 0.6 vs 1.1 ± 1; $p = 0.001$), changes in appetite (0.4 ± 0.7 vs 0.9 ± 1.1; $p = 0.008$) and suicidal thoughts (0.1 ± 0.3 vs 0.6 ± 0.8; $p = 0.0001$).

No significant differences were seen in any other compounds of the BDI between the two groups (pessimism, punishment feelings, crying, loss of interest, worthlessness, weight loss, changes in sleeping pattern or irritability).

These data are summarized in Table 2.

We also performed a crude age-, sex- and BMI-adjusted odds ratio (OR) of having a T2DM using relevant (a score equal or above 2) psychological or cognitive depressive symptoms and negative emotions or somatic/biological symptomatology. The highest odds was associated with complaints about health (OR 4.151; 95% CI 1.111–15.506) and loss of interest in sex (OR 40.784; 95% CI 7.922–209.960). A lower odds among patients with T2DM was associated with loss of pleasure (OR 0.078; 95% CI 0.010–0.585) and changes in appetite (OR 0.040; 95% CI 0.004–0.404). These data are shown in Table 3 and represented in Figs. 1 and 2.

Discussion

Our study aimed to know whether the type of symptoms of depression were different among T2DM subjects with significant depressive symptoms and individuals with a depressive disorder without T2DM.

Our results showed that the score of depressive symptoms, rated with the BDI, was comparable between subjects

Table 2 Comparison of the global score and different items of the BDI between subjects with T2DM and significant depressive symptoms and the control group

	Control group ($n = 50$)		Subjects with BDI ≥ 16 and T2DM ($n = 87$)		p value
	Mean ± SD	Median (Q1–Q3)	Mean ± SD	Median (Q1–Q3)	
BDI global score	18.9 ± 13.4	17 (6–31)	18.8 ± 4.7	18 (16–20)	0.504
Psychological cognitive subscore	10.2 ± 8.3	7 (3–18)	8.6 ± 3.2	8 (7–10)	0.706
Negative emotions subscore	1.4 ± 1.6	1 (0–2.3)	0.8 ± 0.8	1 (0–1)	0.342
Somatic and biological subscore	7.2 ± 4.5	7 (3.8–10.3)	9.1 ± 2.4	9 (8–10)	0.018
Psychological–cognitive depressive symptomatology					
Sadness	0.9 ± 0.9	1 (0–2)	1.4 ± 0.9	1 (1–2)	0.011
Past failure	0.9 ± 0.9	1 (0–1)	0.4 ± 0.8	0 (0-0.5)	0.001
Loss of pleasure	1.1 ± 1.2	1 (0–2)	0.7 ± 0.7	1 (0–1)	0.244
Guilty feelings	1.2 ± 1.1	1 (0–2)	0.5 ± 0.8	0 (0–1)	0.002
Punishment feelings	0.6 ± 1	0 (0–1)	0.3 ± 0.5	0 (0–1)	0.372
Self-dislike	1 ± 1	1 (0–2)	0.7 ± 0.6	1 (0–1)	0.097
Self-criticalness	1.1 ± 1	1 (0–2)	0.4 ± 0.6	0 (0–1)	0.001
Loss of interest	0.8 ± 0.9	1 (0–1.3)	0.6 ± 0.7	1 (0–1)	0.512
Indecisiveness	0.5 ± 0.9	0 (0–1)	1.1 ± 0.8	1 (0–2)	0.001
Worthlessness	1.3 ± 1.2	1 (0–3)	1.5 ± 0.8	1 (1–2)	0.323
Irritability	0.8 ± 0.9	0.5 (0–2)	1.1 ± 0.6	1 (1–1.5)	0.057
Negative emotions					
Suicidal thoughts	0.6 ± 0.8	0 (0–1)	0.1 ± 0.3	0 (0–0)	< 0.001
Pessimism	0.8 ± 0.9	0.5 (0–1.3)	0.7 ± 0.7	1 (0–1)	0.94
Somatic and biological depressive symptoms					
Crying	0.9 ± 1.2	0 (0–2)	0.7 ± 0.5	1 (0–1)	0.584
Concentration difficulty	0.8 ± 0.8	1 (0–1)	1.3 ± 0.8	1 (1–2)	0.012
Weight loss	0.6 ± 1	0 (0–1)	0.4 ± 0.8	0 (0–1)	0.595
Changes in sleeping pattern	1.2 ± 0.9	1 (0.8–2)	1 ± 1	1 (0–2)	0.19
Changes in appetite	0.9 ± 1.1	0.5 (0–2)	0.4 ± 0.7	0 (0–1)	0.02
Health	0.6 ± 0.9	0 (0–1)	1.3 ± 0.9	1 (1–2)	< 0.001
Fatigue	0.8 ± 0.7	1 (0–1)	1.2 ± 0.6	1 (1–2)	0.003
Loss of interest in sex	1.2 ± 1.3	1 (0–3)	2.7 ± 0.6	3 (2–3)	< 0.001

Comparisons between groups were performed using Mann–Whitney U test

Table 3 Crude and age-, sex- and BMI-adjusted odds ratio (OR) of having relevant (score equal o above 2) psychological–cognitive depressive symptoms, negative emotions and somatic/biological symptoms

	Crude OR	95% CI for (OR)	*p* value	Adjusted OR*	95% CI for (OR)	*p* value
Psychological–cognitive depressive symptomatology						
Past failure ≥ 2	0.343	(0.092–1.281)	0.111	0.133	(0.013–1.38)	0.091
Loss of pleasure ≥ 2	0.163	(0.047–0.564)	0.004	0.078	(0.01–0.585)	0.013
Guilty feelings ≥ 2	0.144	(0.042–0.494)	0.002	0.203	(0.029–1.443)	0.111
Punishment feelings ≥ 2	0.097	(0.011–0.85)	0.035	0.29	(0.013–6.623)	0.438
Self-dislike ≥ 2	0.078	(0.016–0.379)	0.002	0.199	(0.02–1.947)	0.165
Self-criticalness ≥ 2	0.118	(0.024–0.59)	0.009	0.27	(0.043–1.716)	0.165
Negative emotions and somatic/biological symptomatology						
Pessimism ≥ 2	0.289	(0.079–1.053)	0.06	0.225	(0.026–1.926)	0.174
Crying ≥ 2	0.118	(0.024–0.59)	0.009	0.101	(0.011–0.94)	0.044
Concentration difficulty ≥ 2	2.66	(0.971–7.287)	0.057	3.198	(0.52–19.667)	0.21
Changes in sleeping pattern ≥ 2	0.457	(0.181–1.157)	0.098	1.3	(0.25–6.772)	0.755
Fatigue ≥ 2	3	(0.891–10.096)	0.076	3.431	(0.42–28.028)	0.25
Changes in appetite ≥ 2	0.089	(0.018–0.435)	0.003	0.04	(0.004–0.404)	0.006
Health ≥ 2	4.355	(1.319–14.373)	0.016	4.151	(1.111–15.506)	0.034
Loss of interest in sex ≥ 2	37.962	(7.858–183.4)	< 0.001	40.784	(7.922–209.96)	< 0.001

T2DM was used as independent variable

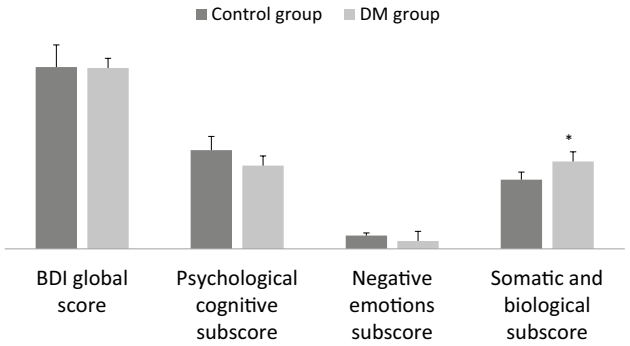

Fig. 1 Values of BDI global score, psychological cognitive subscore, negative emotions subscore and somatic and biological subscore for the group with T2DM and the control group. Values are expressed as mean ± SE. Comparison between groups was performed by Mann–Whitney *U* test. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

with T2DM with a positive screening for depression and individuals with a confirmed major depression. In other words, at the time of the evaluation, the severity of depression was similar between the two groups. The degree of depression in both groups could have been classified as mild to moderate. In patients with T2DM, there had not been a previous diagnosis of depression, probably because the severity of symptoms was not great enough to ask for medical assistance or it could be misdiagnosed as distress related to a chronic disease. On the other hand, patients included in the control group were controlled by psychiatrists and, therefore, undergoing antidepressant therapy, which could have ameliorated the severity of THE initial depressive symptoms.

Furthermore, BMI and waist circumference were greater among patients with T2DM compared with the control group. It is well known that obesity is a major risk factor for the development of T2DM. In fact, overweight and obesity account for more than 90% of the incident cases of T2DM [29, 30]. Although people with a depressive syndrome also have risk factors for overweight/obesity (poorer lifestyle habits, antidepressant drugs, etc.), they are likely to be milder than people with T2DM [31, 32]. Also, T2DM duration might be longer than depression duration, exerting, consecutively, a greater influence on weight.

As mentioned before, depression is a complex disorder that includes a wide variety of symptoms. Therefore, we divided them into three different clusters: somatic and biological depressive symptoms (fatigue, changes in weight, loss of sexual appetite, changes in sleep pattern, changes in appetite, worries about their health and difficulty in concentration), psychological–cognitive depressive symptomatology (past failure, guilty feelings, self-dislike, self-criticalness, sense of punishment, sadness, indecisiveness, loss of pleasure, loss of interest, worthlessness and irritability) and negative emotions (pessimism and suicidal thoughts). When we compared the patients with T2DM and a positive screening for depression with the subjects with a major depressive disorder, we found that the cluster symptomatology predominance was different between the two groups.

Patients with T2DM indicated that more symptoms were included in the somatic–biological cluster of depression,

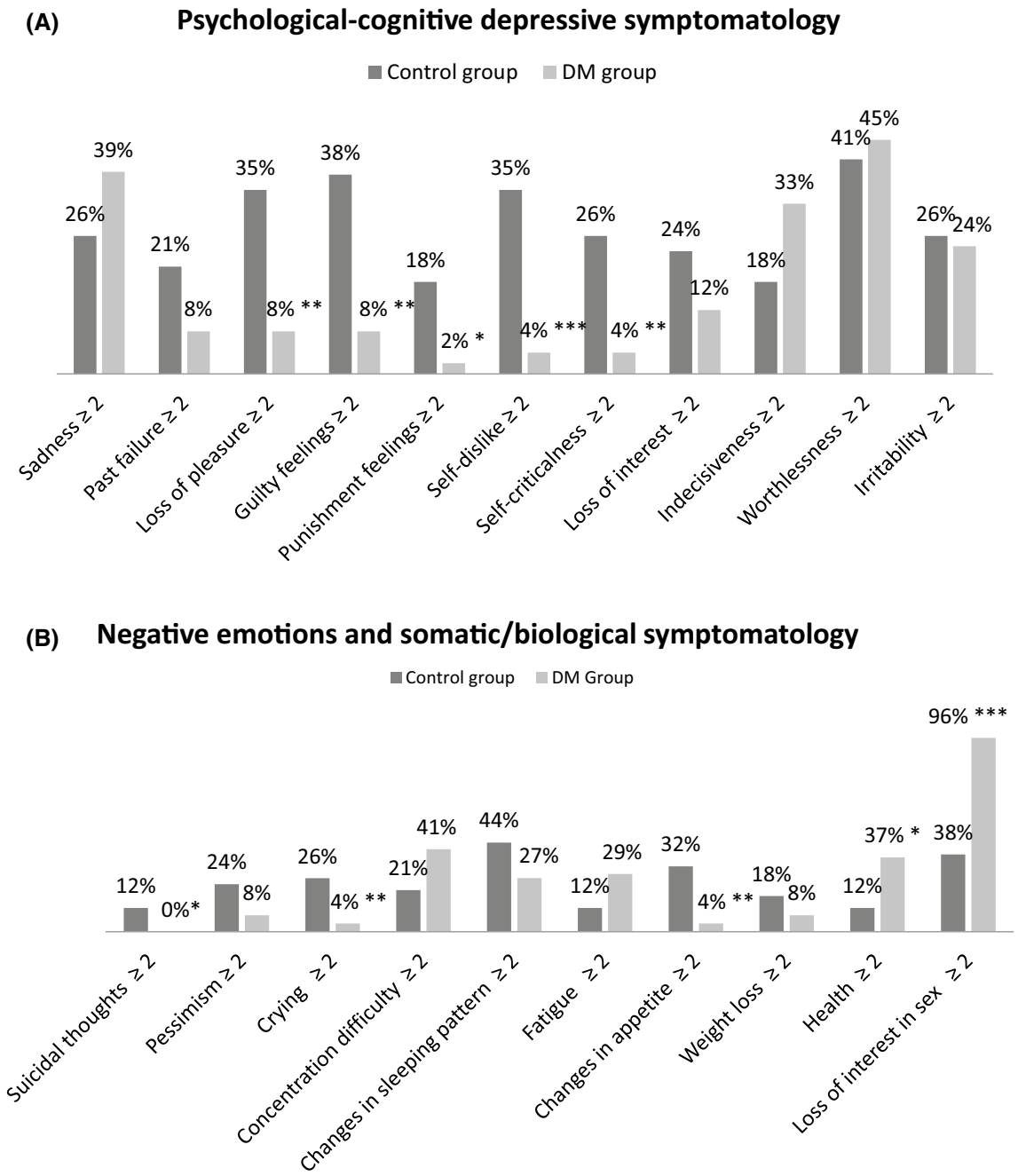

Fig. 2 Percentage of patients with a relevant score (equal o above 2) of psychological and cognitive depressive symptoms (**a**) and negative emotions and somatic/biological symptoms (**b**) for the group with T2DM and the control group. Comparison between groups was performed by Fisher's exact test. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

such as fatigue, loss of sexual appetite and worries about their health. Negative emotions, such as suicidal thoughts, were scarce among type 2 diabetic patients.

The predominance of the somatic–biologic cluster among subjects with T2DM could reflect symptoms related to a poor metabolic control (fatigue and difficulty in concentration) as well as a difficulty to adapt to a chronic condition such as T2DM (worries about their health, sadness and indecisiveness). Furthermore, loss of interest in sex could be due to the frequent erectile dysfunction present among subjects with T2DM and worsened by other comorbidities (hypertension, obesity and dyslipidemia) as well as side effects to drugs prescribed.

The presence of depressive symptoms may be accompanied by suicidal ideation, with thoughts of suicide ranging from passive death wishes to serious consideration of taking

one's own life [33]. Suicidal ideation is probably necessary, but not sufficient, for a suicide attempt [34]. Suicidal ideation is included in the diagnostic criteria for major depression and also in the BDI [24].

There are few studies that have investigated the relationship among diabetes, depression and suicidal ideation. Han et al. found that in a Korean sample, the existence of either diabetes or depression alone increased the presence of suicidal ideation significantly compared with the general population (1.6- and 5.7-fold, respectively). What is more, when both diseases were present, the odds of suicidal ideation increased by more than sevenfold. It is important to highlight that Korea has one of the world's greatest suicide rates, so these findings may not be generalized to adults with diabetes in other countries [33]. In another study, Handley et al. examined the prevalence and correlates of suicidal ideation in a community-based sample of adults with T1DM and T2DM in Australia. Subjects with T2DM treated with insulin reported depressive symptoms more frequently, were more likely to meet criteria for at least moderate depression symptom severity, and were more likely to report suicidal ideation than subjects with either T1DM or non-insulin-dependent T2DM [35]. Ceretta et al. found that suicidal ideation was observed in 13.1% of adults with T2DM compared with only 2.1% in the general community control group [36]. In a recent meta-analysis, diabetes was significantly associated with an increased risk of suicide ($RR = 1.56$; 95% CI 1.29–1.80: $p < 0.01$), with this association being more profound with type 1 diabetes (T1DM). The pooled proportion of deaths attributable to suicide in T1DM patients and T2DM subjects were 7.7% and 1.3%, respectively. They conclude that suicide has an important contribution to mortality in diabetic patients, especially among T1DM patients, with this association being less clear among subjects with T2DM [37].

We compared T2DM patients with subjects with a major depressive disorder instead of the general population, thus explaining the different results when comparing suicidal ideation with all these previously published studies.

It is well known that several adaptation tasks need to be accomplished when a chronic disease like T2DM is present, such as negative and positive self-regulation, daily functioning contingent to treatment needs and the reformulation or beliefs and expectancies about T2DM. It is also important to highlight that these patients do not have significant suicidal thoughts, so the treatment of these depressive symptoms could be done progressively, to diminish maladjustment and improve quality of life [38]. Moreover, it might be difficult to distinguish between "real" somatic/biologic depressive symptoms and poor glycemic control. This fact could be the reason why the response to antidepressants is sometimes scarce until there is an improvement in the metabolic profile. The use of antidiabetic therapies such as GLP-1 (glucagon like peptide-1) analogs, by improving glycemic control as well as other comorbidities, could ameliorate physic symptoms and, therefore, depression and QoL.

On the other hand, subjects with a depressive syndrome included in the control group presented more psychological–cognitive symptoms such as past failure, guilty feelings, self-dislike, self-criticalness and changes in appetite. Moreover, suicidal thoughts were significantly greater among these subjects. These symptoms could be classified as a "traditional" depression.

Another point to consider is that depressive disorder has commonly been related to poor glycemic control. However, so far, results of studies previously published have been inconsistent. While some studies report poorer metabolic control among patients with T2DM and depression, others found no association between depression and glycemic control. One explanation could be the different clusters of depression included in the global term depressive syndrome. In a study recently published, in people with type 1 diabetes, affective symptoms were negatively associated with glycemic control, while somatic symptoms showed a positive association with glycemic control. In this study, in people with T2DM, none of the depressive subtypes were associated with glycemic control, probably due to the small sample size of people with T2DM [39].

As far as we know, this is the first study that compares the different clusters of depression between people with T2DM with significant depressive symptoms and subjects with a depression and without T2DM.

One of the limitations of our study is its small sample size. Also, depressive disorder was not assessed using a structured clinical interview, and it is well known that only about two-thirds of the subjects who reach the major depression score in the self-rated questionnaire were diagnosed as being major depressed cases in the structured interview.

Future research should continue to explore the relationship between depression and T2DM.

In conclusion, subjects with T2DM and a positive screening for depression presented a different cluster of depression compared to depressed subjects without T2DM, with a predominance of somatic–biological depressive symptoms rather than psychological–cognitive cluster and negative emotions, such as suicidal ideation.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval The study was approved by the Ethics Committee of the Hospital Son Llàtzer.

Informed consent Written informed consent was obtained for both groups prior to study participation.

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